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Abstract

This paper presents a stylised social-interaction game where the implementation of a lockdown policy is delegated to the decentralised, uncoordinated decision-making of a large population of atomistic agents – assumed risk-neutral and demographically heterogeneous. Compliance with policy prescriptions is socially beneficial but individually costly. In the static, it determines the individual risk of contagion in social interactions (at the micro-level) and the number of new infections (at the macro-). Over time, it affects the peak prevalence of the disease and the duration of the epidemic. Albeit atomistic, agents act *strategically*, for they rationally anticipate others' behaviour when deciding (not) to comply. Three are the key results of our analysis. First, the strategic incentives faced by the agents co-evolve with the epidemic. When prevalence is low, compliance is a dominated strategy. When prevalence is high, individual decisions to comply are *strategic substitutes*: older/weaker agents self-protect by implementing social distancing and younger/healthier ones free-ride. Second, the strategic incentives faced by the agents co-evolve, too, with their beliefs about susceptibility. When they disregard any information about their past behaviour and use the aggregates to estimate susceptibility, strategic substitutability prevails. When beliefs are path-dependent, both complementarity and substitutability may arise. Third, we show that SIR-based models that fail to account for the endogenous response to policy prescriptions may substantially overestimate the effectiveness of lockdowns. Incidentally, we highlight that myopic behaviour may cease to be rational in a dynamic setting where agents' beliefs about susceptibility are path-dependent.

JEL classification: C72, D71, H41, I13

Keywords: COVID-19, Contagion, Social distancing, Collective action, Strategic complements and substitutes

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"As for how to enter the city, Renzo had heard in a general way that there were very strict orders to admit no one without a certificate of health, but that on the other hand anyone who used his wits and seized the right moment could enter quite easily. Such was, in fact, the case..."

— Alessandro Manzoni, *The Betrothed: XXXIV.*

"<You can't make more stringent ones than those we have now.>
<No. But every person in the town must apply them to himself.>
Cottard stared at him in a puzzled manner, and Tarrou went on to say that there were far too many slackers, that this plague was everybody's business, and everyone should do his duty."

— Albert Camus, *The Plague: Part II.*

1. Introduction

Among the many lessons to be learnt after two years of SARS-CoV-2 pandemic, two seem to be – in our opinion – particularly relevant for policy-making. First: albeit necessary, mandatory social distancing in general, and strict lockdown policies in particular, embed a clear trade-off between 'health and wealth': while containing the spread of the disease, they impose significant economic losses onto firms and individuals at once – in the form e.g. of unearned profits, reduced income, and/or higher unemployment rates. Caught between a rock and a hard place, and relentlessly pressed by the state of emergency, the policy-maker strives to mediate between the many competing – often *opposite* – interests that necessarily inform policy-design when public health is at stake – protection of the elderly and of the weak, regular provision of the essential healthcare services, social discontent, economic performance and political consent among the others. Not surprisingly, to strike the right balance is not an easy task. Second: if it is true that to devise social-distancing norms properly is an hard job, even harder it is to have them properly implemented by the citizenry. This is the issue at the core of our paper. Fear and superstition allegedly prevented an effective containment of the great plague of Milan in 1629-31. Apathy and denial took the blame in the fictional plague of Orano, depicted by Camus roughly 300 years later. There is no need to appeal to

irrationality, however, to make sense of inefficient implementation. Things may be (relatively) simpler: "*collective interests do not necessarily produce collective action*" [Heckathorn, 1996][1]. When the population is sufficiently large, compliance with norms is individually costly but irrelevant in the aggregate. Monitoring by the public authority is necessarily partial and typically imperfect, and when punishment for misbehaviour is an unlikely event, free-riding may become a tempting option. The two quotations that open this paper epitomise the clash between the (moral) obligation felt by the individual to *do his/her own duty*, and the selfish but rational incentive to step back and *let others do their duty*. Thus, even if an optimal lockdown policy was indeed available to the public authority, Pareto-efficiency at the social level would by no means guarantee efficient implementation at the individual one.

In a recent paper, [Bisin and Moro, 2022a] highlight that naïvely-designed social-distancing norms entail significant hidden costs, and stress the necessity to analyse the design and implementation of lockdown policies jointly by accounting for the endogenous response of agents to – changes in – policy prescriptions. While the health-wealth trade-off, however, and the related policy dilemmas, have (rightfully) been – and still remain – the object of study of an extensive literature in epidemiological economics[2], the problem of the enforcement of containment policies in general, and that of their decentralized implementation in particular, seem to have received relatively mild(er) attention. There are notable exceptions. [Engle et al., 2021], [Farboodi et al., 2021] and [Toxvaerd, 2020], for instance, embed the workhorse SIR framework[3] with fully microfounded behavioural rules, thus imposing the formal rigour of equilibrium reasoning to the dynamics of the epidemiological aggregates they derive "bottom-up" from the social interactions of rational optimisers. [Battiston et al., 2022] build on the seminal work of [Ehrlich and Becker, 1972] to study self-protection in epidemic through the lens of a two-player insurance game. [Bisin and Moro, 2022a, Bisin and Moro, 2022b] consider adaptive behavioural responses within an ABM framework, where rule-of-thumb agents interact randomly in a limited space and adapt their social behaviour in response to changes in environmental conditions and/or policy prescriptions. One interesting result seems to hold across all modelling approaches briefly outlined above: when endogenous social-distancing is included into the picture,

[1] See page 250 for the quoted passage.

[2] See e.g. [Alvarez et al., 2021], [Jones et al., 2021] and [Eichenbaum et al., 2021].

[3] [Kermack and McKendrick, 1927] – se also [Avery et al., 2020] for an excellent survey of the related literature.

the disease-diffusion process and the dynamics of the main epidemiological aggregates significantly differ from those observed in a standard SIR framework.

The contribution of this paper to the literature that studies the endogenous implementation of social-distancing norms is three-fold. First, it identifies a set of key incentives that drive individual decisions (not) to comply with policy prescriptions when agents are strategically sophisticated. Second, it studies how such incentives (co-)evolve with the main epidemiological aggregates. Third, it shows how they respond to different levels of enforcement. To this end, we present a stylised but sufficiently rich social-interaction game where the implementation of a lockdown policy is delegated to the decentralised, uncoordinated decision-making of a large population of atomistic agents, assumed risk-neutral and demographically heterogeneous. Compliance with policy prescriptions is socially beneficial but individually costly, and affects the individual risk of infection in interactions with others by determining both (i) the total number (mass) of social interactions (extensive margin) and (ii) the composition of the interacting subpopulation in terms of disease-prevalence (intensive margin). At the macro-level, it determines total number (mass) of new infections, the peak prevalence of the disease and the overall duration of the epidemic. Agents act *strategically*, and rationally anticipate others' behaviour when deciding whether or not to comply with norms – in doing so internalizing *partially* the spillover effect on individual risk of the aggregate compliance rate. Closest in spirit to our model is [Engle et al., 2021], that will serve as a natural benchmark for our analysis throughout the entire paper. Three are the key results we outline. First, the strategic incentives that drive agents' decision-making co-evolve with the epidemic. When disease prevalence is low, compliance is not rational(isable) at the individual level. When prevalence is high(er), agents' decisions to comply are *strategic substitutes* and opportunistic behaviour may arise. This result is consistent with the findings of [Engle et al., 2021]. Second, strategic incentives co-evolve, too, with agents' beliefs about their susceptibility to the infection. As an agent grows confident about his/her immunity, his/her propensity to comply increases; the opposite holds true as he/she suspects to be susceptible. In such an environment, the more susceptibles comply, the riskier social interactions are for non-compliant ones. Individual decisions to comply become *strategic complements*. To the best of our knowledge, this result is entirely novel, and therefore complements and clarifies the substitutability result of [Engle et al., 2021]. Third, SIR-based dynamics that do not account for

the endogenous response of agents to policy prescriptions tend to (substantially) overestimate the effectiveness of social-distancing norms.

The paper is structured as follows. In Section 2 we outline the static social-interaction game, and in Section 3 we characterise its unique Nash equilibrium. In Section 4 we use the static equilibrium characterisation to simulate a behavioural SIR augmented with endogenous compliance rates. In Section 5 we extend the static game of Section 2 by allowing agents to know their epidemiological statuses, and show that the path-dependency of beliefs is crucial in the determination of the strategic incentives that drive endogenous compliance. In Section 6 we conclude with a brief summary of the paper and of its main results.

2. The Model

2.1 Setup

Consider a stylised environment populated by a benevolent policy-maker (the *government*) and a continuum of atomistic, risk-neutral agents of measure one (the *citizens*), uniformly distributed over the interval $[0, 1]$ and indexed by i. Citizens are heterogeneous with respect to age, sex, general health conditions, presence of pre-existing and/or chronic pathologies, etc... and their heterogeneity is parameterised by a single summary statistic $x(i) \in \mathbb{R}$, defined as

$$x(i) \;=\; \bar{x} + \varepsilon(i) \;, \tag{1}$$

with $\bar{x} \in \mathbb{R}$ a common component – identical for all citizens –, and where $\varepsilon(i)$ is purely idiosyncratic white noise, in the form

$$\varepsilon(i) \;\sim\; \mathcal{N}\left(0, \sigma^2\right) \tag{2}$$

for all $i \in [0, 1]$. We refer to $x(i)$ as the *demographic profile* of citizen i. All idiosyncratic components $\varepsilon(i)$ are assumed to be i.i.d. across individuals, so that $\bar{x}$ can easily be interpreted as the median/average profile population-wide, while $\sigma > 0$ becomes a natural proxy of the cross-sectional demographic heterogeneity of the citizenry[4]. We impose an arbitrary ordering over the demographic

[4] Indicate with $\hat{x}$ the median/average demographic profile. Citizens are atomistic and uniformly distributed over $[0, 1]$, hence $\hat{x}$ is obtained via integration over the unit interval with a unitary density. Building on [Judd, 1985], it is

profiles, whereby *lower* values of $x(i)$ correspond to younger and/or healthier citizens – conversely, *higher* value of $x(i)$ correspond to older and/or weaker ones. (The ordering is arbitrary but without loss of generality.) All structural parameters and definition (1) are assumed to be common knowledge.

2.2 Disease

At an arbitrary date – that we conventionally indicate with $t = 0$ – the government becomes aware of the fact that a mass $\underline{I} \in (0, 1)$ of citizens had been infected by an unknown pathogen (the *virus*). The virus causes a potentially mortal disease, identified by two characteristics: i) *contagiousness* and ii) *mortality*. The former indicates the probability $\beta \in (0, 1]$ to become infected after being exposed to the virus – it is identical across citizens. The latter refers to the probability of death upon infection. It is heterogeneous across individuals and determined by their demographic profiles: older and/or weaker citizens have an higher probability to die after infection than younger and/or healthier ones. Formally, we define the probability of death upon infection *per-period*, q_i^D, as a function $q : \mathbb{R}^2 \longmapsto [0, 1]$ in the form

$$q_i^D \;=\; \Phi\Big(\theta + \alpha\, x(i) \Big) \;, \tag{3}$$

where $\Phi(\,\cdot\,)$ indicates the Normal Standard CDF, $\alpha > 0$ scales the sensitivity to individual-specific demographic traits $x(i)$, and $\theta \in \mathbb{R}$ parameterises the disease-specific component of mortality – larger values of θ indicate higher mortality, *ceteris paribus*. In words: the risk of death faced by the i-th individual upon infection is determined by both disease-specific characteristics *and* by his/her individual-specific resilience to it. Once infected, an individual faces a probability to die q_i^D in each subsequent period. Following [Ellison, 1964], the median/average mortality population-wide

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immediate to check that

$$\hat{x} \;=\; \int_0^1 x(i)\,\mathrm{d}i \;=\; \int_0^1 \bar{x}\,\mathrm{d}i + \int_0^1 \varepsilon(i)\mathrm{d}i \;=\; \bar{x}\;, \qquad \text{almost surely}\,,$$

since LLN reasoning extended to continua of i.i.d. random variables yields that $\int_0^1 \varepsilon(i)\,\mathrm{d}i \;=\; \mathbb{E}\,[\varepsilon(i)] \;=\; 0$ almost surely, with $\mathbb{E}\,[\,\cdot\,]$ the expectation operator – see also [Vives, 1988], page 854, footnote 4, and [Vives, 2014], page 1207, footnotes 15 and 16. Notice further that the distinction between median and average is here immaterial, for the cross-sectional (joint) distribution of the $x(i)$ statistics is symmetric.

(per-period) $\bar{Q}^D$ can be written as[5]

$$\bar{Q}^D \;=\; \Phi\left(\frac{\theta + \alpha\,\bar{x}}{\sqrt{1+\alpha^2\sigma^2}}\right) \tag{4}$$

with $\bar{x}$ the median/average resilience (that coincides with the median/average demographic profile) and σ^2 the cross-sectional demographic variance – see equation (1). Note that the disease partitions the citizenry into three epidemiological aggregates: *S*usceptibles, *I*nfected, *R*ecovered – SIR[6]. We call $e\,(i)$ the *epidemiological status* of the i-th citizen, i.e.

$$e(i) \;:\; [0,1] \;\longmapsto\; \Big\{SY, ASY, S, R\Big\} \tag{5}$$

for every $i \in [0,1]$. Infected citizens are *ASY*mptomatic with probability $p \in (0,1]$, and *SY*mptomatic with probability $(1-p)$ – and remain so until they recover or die. Symptoms are assumed to be apparent and disease-specific, so that each symptomatic infected is aware of his/her status, that remains unknown, conversely, to the asymptomatic. Irrespective of the presence or lack of apparent symptoms, infected individuals recovers with (per-period) probability p_i^R defined as

$$q_i^R \;=\; \gamma\left(1 - q_i^D\right)\;, \tag{6}$$

with $\gamma \in (0,1)$ and where q_i^D is the (individual-specific) probability of death *post*-infection defined by (3). In words: in each period subsequent to the infection an infected individual faces a probability of recovery q_i^D that is proportional to his/her resilience to the disease. Consistently with (3) and (6), the (per-period) probability of remaining infected, q_i^I, can be written as

$$\begin{aligned} q_i^I \;&=\; 1 - q_i^D - q_i^R \\ &=\; \left(1-\gamma\right)\left(1-q_i^D\right) \end{aligned}\;. \tag{7}$$

The corresponding (population-wide) averages $\bar{Q}^R$ and $\bar{Q}^I$ are defined consistently with (4). Finally, a recovered individual is immune forever, so that a susceptible citizen is, by construction, an

[5] See also [Owen, 1980] for a list of integrals of Gaussian functions where the same result is stated with a different proof.

[6] More precisely, the population is partitioned into *S*usceptibles, *I*nfected, *R*ecoverd *and* *D*ead – SIRD. The distinction is immaterial for the results.

individual who had *never* been previously infected. As in Subsection 2.1, all structural formulae and parameters are assumed to be common knowledge.

2.3 Contagion

Infected citizens are *infective*, and spread the contagion by interacting with susceptible ones. All symptomatic infected are quarantined immediately as they begin to show disease-specific symptoms. It is impossible, however, to identify asymptomatics, that remain free to interact with others and spread the disease. The passing mechanism is such that, when the virus infects a susceptible individual, it remains latent for one period, and suddenly 'wakes' at the beginning of the subsequent one. During the latency period the individual is infected but not infective. Only once the virus turns active can the individual pass the disease to others. The probability of infection of a susceptible citizen is determined by three factors: (i) the extent of his/her social interactions; (ii) the composition of the interacting population – in terms of group-specific disease prevalence; (iii) the environment in which interactions do occur. The first and the second factor(s) relate to *individual* and *collective* behaviour, respectively, and are therefore *endogenous*. The third refers to contingent environmental conditions – city vs. countryside, closed vs. open space, high vs. low demographic density, and the like –, and it is therefore completely exogenous. Besides that based on endogeneity/exogeneity, a second distinction is here relevant: from the point of view of the generic agent, individual social behaviour is a *control variable*, while others' behaviour and its interaction with the environment amount to a pure – global – *externality*[7]. While the effect of the former on the individual probability of infection is straightforward – the larger the number of social interactions, the higher the risk of contagion he/she faces, *ceteris paribus* –, less clear-cut *a priori* is the effect of the latter, that results from the superimposition of an *extensive-* and *intensive-margin* component.

2.3.1 Extensive-Margin Component

The extensive-margin component of the global externality relates to the fact that the marginal effect on the risk of contagion faced by a non-compliant individual of an increase in the number (mass)

[7] Since we avoid imposing any structure on the spatial dimension of the model (e.g. by assuming that agents interact within well-specified network or on a lattice), there is no meaningful way in this model to keep track of local, pairwise interactions. Social interactions are here necessarily of a mean-field type, and the spillover effect of the aggregate social behaviour on the individual risk of infection is *global* in the sense that it is symmetric across agents, irrespective of their (unmodeled) location into the system.

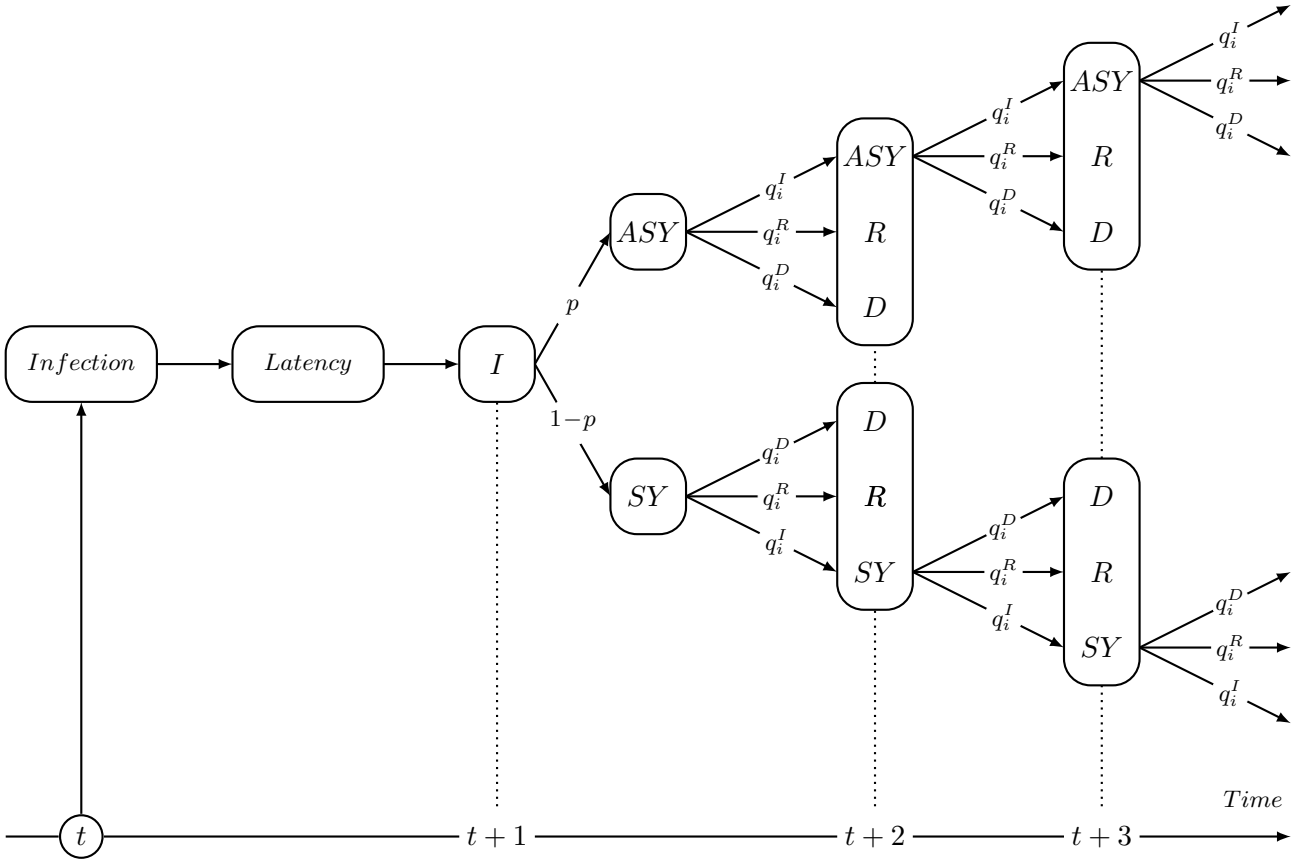

Figure 1. Chain of possible epidemiological patterns post-infection for the generic i-th individual that becomes infected at the arbitrary date t.

of social interactions *per se* depends on the congestion of the environment in which interactions actually occur. Congestion, in turn, is determined by the (exogenous) carrying capacity of the environment: a small room gets overcrowded if few people enter it, while cohorts of individuals are necessary to (over)crowd a stadium. [Bisin and Moro, 2022a] stress the first-order importance of the spatial/geographical dimension of social contacts in determining the dynamics of a contagion diffusion process. Consistently, we embed our model with a (very) reduced-form representation of spatial interactions, whereby the larger is the mass of interacting citizens, the closer are their contacts, the higher is the probability of contagion in social interactions, *ceteris paribus*. Indicating with $M \in [0,1]$ the mass of the interacting (sub)population, we formalize the extensive-margin effect via a congestion externality $\varphi(M)$ in the form

$$\varphi(M) = (M)^{\phi} \ , \tag{8}$$

where the coefficient $\phi \geq 0$ scales the speed of congestion as follows: (i) with $\phi \in (0,1)$ congestion obtains very soon as M becomes larger; (ii) with $\phi = 1$ (over)crowding increases linearly in M ; (iii) with $\phi > 1$ congestion occurs very slowly, and the mass of interacting individuals M must be (very) large to induce overcrowding[8].

2.3.2 Intensive-Margin Component

The intensive-margin component relates to the fact that the risk of contagion in social interactions depends on the composition of the interacting (sub)population in terms of group-specific disease prevalence, and that the latter may *not* remain constant as the mass of interacting individuals varies. This is the case, for instance, if some types of agents systematically ignore social-distancing norms more frequently than others. Consider again a mass $M \in [0,1]$ of citizens that interact, with $ASY(M)$, $S(M)$ and $R(M)$ the number (mass) of ASYmptomaitc infected, Susceptible and Recovered individuals in M, respectively. The ratio

$$\pi(M) = \frac{ASY(M)}{ASY(M) + S(M) + R(M)} \ , \tag{9}$$

indicates the *group-specific* prevalence of the disease in subpopulation M – and can be interpreted as the probability to interact with an asymptomatic infected under the assumption that each individual interacts with all other individuals in M[9]. A simple thought experiment may help to further clarify the issue. Compare the following three situations: in the first, the compliance rate is identical for all citizens, irrespective of epidemiological states $e(i)$; in the second, recovered citizens ignore social-distancing norms while the asymptomatics and susceptibles comply; in the third, recovered and susceptible citizens comply while the asymptomatics ignore social distancing. As the mass of interaction M increases, the group-specific disease prevalence converges (i) to the prevalence population-wide in the first situation, (ii) to zero in the second, and (iii) to one in the third. The take-home message is: when group-specific prevalence is considered, the marginal effect on the risk of contagion faced by a non-compliant individual of an increase in the mass of social interactions

[8] If $\phi = 0$ there is no congestion externality, and the risk of contagion in social interactions is entirely determined by the prevalence of the disease into the interacting (sub)population.

[9] Due to the continuum-player specification, $\pi(M)$ constitutes *de facto* a measure of the *frequency* of encounters with asymptomatic infected – assuming that interactions occur with the entire mass of individuals M.

depends *crucially* on the underlying behavioural rule that generated the observed increase – and its sign is therefore ambiguous *a priori*.

2.3.3 The Global Externality

Indicating with $G(M)$ the global externality, that parameterises the risk of contagion faced by a non-compliant individual when M counterparties opted for the same course of action, we can write

$$\begin{aligned} G(M) &= \beta\,\pi(M)\,\varphi(M) \\ &= \beta\left(\frac{ASY(M)}{ASY(M)+S(M)+R(M)}\right)(M)^{\phi} \end{aligned} \tag{10}$$

with $\varphi(M)$ and $\pi(M)$ the congestion externality (extensive-margin) and the group-specific prevalence effect (intensive-margin), defined by (8) and (9) respectively. In words, as the number of interacting individuals M increases, the risk of contagion faced by the generic non-compliant citizen (i) unambiguously increases along the extensive margin, $\varphi(\cdot)$ due to (over)crowding of the environment, but (ii) may increase or decrease along the intensive margin $\pi(\cdot)$ depending behavioural rule(s) that govern social interactions. In Subsection 3 and in Section 5 we fully endogenise te intensive-margin component $\pi(\cdot)$ by pinning down the compliance rules of citizens as rigorously-defined equilibrium strategies.

2.3.4 Risk of Contagion in Social Interactions: Wrap-Up

To sum up, the modelling strategy we adopted to link the aggregate social behaviour of the citizenry to the risk of contagion faced by the single non-compliant individual can be effectively explained via a simple similitude. Imagine a queue of individuals that enters sequentially a room of given width. As more and more individuals arrive (the interacting subpopulation M), the room gets more and more crowded, until overcrowding finally obtains (the congestion externality $\varphi(M)$, that increases in M). Keeping constant the rate of new arrivals, the speed at which overcrowding occurs is determined by the width of the room (parameter ϕ). As the room gets more and more crowded, interactions become more and more 'concentrated'. What is the effect of overcrowding on the probability of contagion faced by a susceptible individual that enters the room? Concentration increases the likelihood of contagion when the individual interacts with an infected counterparty

(due to increased physical proximity), but is completely *irrelevant* when he/she interacts with a non-infected (the intensive-margin effect $\pi(M)$, whose relation with M is ambiguous). Overall, the net effect of increased concentration on the likelihood of contagion is entirely determined by the composition of new arrivals. If disease-prevalence among the individuals in the queue remains constant, the effect is unambiguously positive: the probability of meeting an infected in the room is stable, but the physical proximity of contacts increases. If disease-prevalence decreases with new arrivals (e.g. as time passes susceptible individuals only remain in the queue), physical proximity increases but the probability of interacting with an infected counterparty in the room decreases. The net effect depends on the relative magnitude of the two margins: if the (positive) marginal effect of increased physical proximity is larger than (negative) the dilution effect of new arrivals with low(er) group-specific prevalence, than risk increases overall. If the opposite holds true, then the overall effect is negative. In Appendix D we test the robustness of the qualitative argument outlined in this Subsection simulating a formal (toy) model of social interaction where physical proximity increases the probability of contagion. Our simulations suggest that the argument may be quite robust.

2.4 Containment: The (Static) Social-Interaction Game

In order to – try to – contain the epidemic, in $t = 0$ the government imposes mandatory social distancing (the *lockdown*) to a share $L \in (0, 1]$ of citizens, that are formally required by the public authority to abstain from social interactions *for one period*. While the lives of citizens extend indefinitely in time, the social-interaction game is played only once – and it is therefore purely *static*. The share $1 - L$ of exempted citizens can be thought of as identifying the providers of goods and services deemed 'essential' by the policy-maker – healthcare, public security and transportation, and the like – se e.g. [Alvarez et al., 2021]. In the presence of partial and imperfect monitoring by the public authority, the implementation of the lockdown boils down to the willingness of individuals to comply (or not) with policy prescriptions. Citizens are here assumed to be (instrumentally) rational, strategically sophisticated utility-maximisers, whose decision-making is driven essentially by self-interest[10]: any appeal to morality and/or sense of duty is therefore bound to remain a dead

[10] Needless to say, we do *not* intended this as a moral theory of the human nature. Rather, we do consider it as an extreme, often unrealistic, but methodologically convenient assumption, useful at imposing some structure – and a

letter. As a consequence, the policy-maker has no suitable alternative to get its policy implemented but to resort to command-and-control[11]: all citizens caught cheating on social distancing norms are imposed a fine $k > 0$. Individuals that are alive and without symptoms are assumed to enjoy a (reservation) utility normalized to zero. Every infected citizens faces, in each period subsequent to the infection, an idiosyncratic probability of death and recovery q_i^D and q_i^R – see equations (3) and (6), respectively. Death entails an arbitrarily large utility loss $D \gg 0$ assumed identical across citizens[12]. Agents' common time preferences are summarized by the (common) discount factor $\lambda \in (0,1)$ so that, at $t = 0$, the expected utility $\tilde{D}_i$ of an infected citizen with demographic profile $x(i)$ can be written as

$$\tilde{D}_i \;=\; \lambda^2 D \left(\frac{\Phi\big(\,\theta + \alpha x(i)\,\big)}{1 - \lambda\,(1-\gamma)\,\Big(\,1 - \Phi\big(\,\theta + \alpha x(i)\,\big)\,\Big)} \right) \quad , \tag{11}$$

see Appendix B for its derivation. At the moment social distancing is mandated by the authority, each citizen is faced with an all-or-nothing (binary) choice: comply ($a(i) = 1$) or not ($a(i) = 0$) with policy prescriptions. The median/aggregate compliance rate population-wide $A \in [0,1]$ can therefore be defined as

$$A \;=\; \int_0^1 a(i)\,\mathrm{d}i \quad . \tag{12}$$

Compliance bears no direct utility, but yields full protection from contagion[13]. Non-compliance yields a fixed (warm-glow) benefit $b > k$, but entails a positive risk of infection, parameterised by the global externality (10) and directly determined by the aggregate social behaviour of the citizenry – the lower the compliance rate A, the larger the number/frequency of interactions, overall, the higher the risk of contagion faced by a non-compliant individual. Note that by complying with social-distancing norms a citizens is forced to give up the warm-glow benefits from social interactions,

great deal of formal rigour – onto the problem(s) under analysis in this paper.

[11] Consider this as a 'worst-case scenario'.

[12] This entails *de facto* that life has the same value to all the individuals, irrespective of their demographic profile.

[13] Differently from [Engle et al., 2021], therefore, the number/frequency of social interactions is explicitly action-contingent in our model and, consequently, so it is the individual risk of contagion – see footnote 12, (page 7) in [Engle et al., 2021].

so that the element

$$c = \begin{cases} c_E = b - k & \text{with lockdown} \\ c_{NE} = b & \text{without lockdown} \end{cases} \tag{13}$$

can be interpreted without loss of generality as the *implicit* cost of social distancing[14] – and where the subscripts E and NE indicate "enforcement" and "no-enforcement", respectively. When the government is enforcing a lockdown, c coincides *de facto* with the cost of compliance with policy prescriptions. When there is no mandatory social distancing, it can be interpreted as the implicit cost of self-isolation[15]. The distinction is important in our model: if the risk of contagion is sufficiently high, older/weaker citizens may use social isolation as a device for self-protection even in the absence of a lockdown[16]. Self-protection is not binding, however, for younger/healthier citizens, that can only be nudged into social distancing via active enforcement by the public authority. By reducing the warm-glow benefit from social interactions, active enforcement entails here a reduction in the implicit cost of compliance, *ceteris paribus* – see equation (13).

3. Equilibrium Characterization

Due to the presence of a global spillover effect $G(\cdot)$ of the aggregate social behaviour on individual risk of contagion, any (candidate) equilibrium profile must entail some form of mutual consistency between (i) the compliance rules individually used by citizens – pinned down by rational cost-benefit reasoning –, and (ii) the actual risk of contagion they induce once aggregated. The subjective beliefs every citizen holds about his/her epidemiological status $e(i)$ play a crucial role in this model – the issue is extensively discussed in Section 5. To ensure sufficient analytical tractability, we assume that (i) a citizens not showing disease-specific symptoms ignores his/her status $e(i)$, but (ii) all epidemiological aggregates be common knowledge. (Both assumptions are relaxed in Section 5.) For the sake of consistency, an important exception must be made for all those citizens that are or have been previously symptomatic: since symptoms are disease-specific, they cannot but be aware

[14] It is an opportunity cost.

[15] See [Toxvaerd, 2020], footnote 1, page 1.

[16] The logic is close in spirit to that outlined by [Ehrlich and Becker, 1972] and, more recently, by [Battiston et al., 2022].

of being infected when symptomatic, and of being recovered once they cease to be so. This is just a detail, but an important one: while symptomatic citizens have no bearing on the outcome of interaction game – they are quarantined as they begin to show symptoms –, this is not the case for the mass $(1-p)\,R$ of citizens that recovered after having been symptomatic in the past. Since the risk of (re)infection is null by assumption, non-compliance is a dominant strategy for them, i.e.

$$\Pr\Big(\, e(i) = S \,\Big|\, symptoms \,\Big) \;=\; 0 \qquad \Longrightarrow \qquad a^{*}\,(i) = 0 \tag{14}$$

holds. All the other citizens, that never showed disease-specific symptoms in the past, have no available alternative to form beliefs about their statuses $e(i)$ but to resort to the observable epidemiological aggregates. Similarly to [Engle et al., 2021], we therefore assume that

$$\Pr\Big(\, e(i) = S \,\Big|\, \text{no symptoms} \,\Big) \;=\; \frac{ASY}{ASY + S + pR} \tag{15}$$

holds. In words: the probability of being susceptible estimated by the i-th citizen that never showed symptoms coincides with the (known) disease-prevalence within the mass of citizens that never showed symptoms. Differently from the citizens that recovered from a symptomatic infection, those which never showed symptoms cannot condition their decisions (not) to comply with social-distancing on on their states $e(i)$: they are forced to use the *same* compliance rule. As a consequence, irrespective of the specific compliance rule adopted by the non-symptomatics, the aggregate mass $M = M(A)$ of non-compliant (interacting) citizens must be

$$M(A) \;=\; (1 - LA)\,\big(ASY + S + pR\,\big) + (1-p)\,R \tag{16}$$

so that, consistently, the intensive-margin component of the risk of contagion – the congestion externality (8) – can be (re)written as

$$\varphi\,(A) \;=\; \Big[\,(1 - LA)\,\big(ASY + S + pR\,\big) + (1-p)\,R\,\Big]^{\phi} \tag{17}$$

and the intensive-margin component – the group-specific disease prevalence (9) within the interacting subpopulation – becomes

$$\pi\left(A\right) \;=\; \frac{ASY\left(1-LA\right)}{\left(1-LA\right)\left(ASY+S+pR\right)+\left(1-p\right)R} \quad . \tag{18}$$

for any arbitrary compliance rate $A \in [0,1]$ as defined by (12). Substituting (16) and (18) into (10) we can finally re-write the individual risk of infection faced by the generic non-compliant citizen – that never showed symptoms – as

$$G(A) \;=\; \frac{\left(ASY\right)\left(1-LA\right)}{\Big[\left(1-LA\right)\big(ASY+S+pR\big)+\left(1-p\right)R\Big]^{1-\phi}} \quad . \tag{19}$$

Note that, when all non-symptomatic citizens are forced to adopt the same compliance rule, it holds that

$$\frac{\partial}{\partial A}\,\varphi\left(A\right) \;=\; -\frac{\phi\,L\left(ASY+S+pR\right)}{\Big[\left(1-LA\right)\left(ASY+S+pR\right)+\left(1-p\right)R\Big]^{1-\phi}} \;<\; 0 \tag{20a}$$

$$\frac{\partial}{\partial A}\,\pi\left(A\right) \;=\; -\frac{\left(1-p\right)L\left(R\right)\left(ASY\right)}{\Big[\left(1-LA\right)\left(ASY+S+pR\right)\;+\;\left(1-p\right)R\Big]^{2}} \;<\; 0 \tag{20b}$$

for every compliance rate A. The interpretation is straightforward. As A increases, the mass of interacting people *decreases*, overall. This drives down the risk of contagion *ceteris paribus* via the marginal effect of the congestion externality – for any given carrying capacity of the environment, the smaller the mass of interacting individuals, the lower the level of congestion. Since the recovered from symptomatic infections $\left(1-p\right)R$ do not comply for sure, an increase in A amounts to a reduction in the share of asymptomatic infected in the interacting subpopulation, hence to a decrease in the group-specific prevalence $\pi\left(A\right)$. The overall effect on the risk of contagion $G(A)$ is therefore negative, for

$$\frac{\partial}{\partial A}\,G(A) \;=\; \varphi\left(A\right)\left(\frac{\partial}{\partial A}\,\pi\left(A\right)\right)+\pi\left(A\right)\left(\frac{\partial}{\partial A}\,\varphi\left(A\right)\right) \;<\; 0 \tag{21}$$

must hold via (20a) and (20b). For the sake of brevity, we adopt henceforth the following compact notation: (i) the vector $\boldsymbol{\Omega}$ summarises all the environmental parameters (including the calibra-

tion)[17]; the vector $\mathbf{E} = \langle ASY, S, R \rangle$ summarises the epidemiological aggregates. Consistently, the action-contingent expected utility of the generic non-symptomatic citizen can be written as

$$u\Big(a(i), A; \mathbf{\Omega}, \mathbf{E}\Big) \;=\; \begin{cases} -c & \text{if } a(i) = 1 \\ \\ -\tilde{D}\,\beta\,G(A) & \text{if } a(i) = 0 \end{cases}, \tag{22}$$

where $\beta\,G(A)$ is the individual risk of contagion in social interactions, and $\tilde{D}_i$ is the discounted cost of death post-infection defined by (11). A rational citizen abstains from social interactions if and only if $u\,(a(i) = 1, A; \cdot\,) \geq (a(i) = 0, A; \cdot\,)$ for any arbitrary compliance rate A[18]. Indicate with $\Delta_i\,(A)$ the differential payoff

$$\begin{aligned} \Delta_i\,(A) \;&=\; u\big(a(i) = 1, A; \mathbf{\Omega}, \mathbf{E}\big) - \big(a(i) = 0, A; \mathbf{\Omega}, \mathbf{E}\big) \\ &=\; -c + \tilde{D}\,\beta\,G(A) \end{aligned} \tag{23}$$

that parameterises the relative appeal of compliance w.r.t. non-compliance to a citizen with demographic profile $x(i)$ when the aggregate compliance is A. It is immediate to check that, for any $x(i)$, the incentive to comply strictly decreases as the aggregate/median compliance rate A increases. Hence, when citizens ignore their epidemiological states $e(i)$, their individual decisions to comply are strategic substitutes. Proposition 1 summarizes the result.

PROPOSITION 1. *If all citizens that never showed disease-specific symptoms ignore their epidemiological states $e(i)$, then their individual decisions to comply with social-distancing norms are strategic substitutes.*

Proof. The proof is trivial, for the result stems directly from (20)-(21), whereby

$$\frac{\partial}{\partial A}\,\Delta_i\,(A) \;=\; \tilde{D}_i\,\beta\left(\frac{\partial}{\partial A}\,G(A)\right) \;<\; 0$$

holds. □

[17] For the sake of formal rigour, it holds that $\mathbf{\Omega} \in \mathcal{O} \subset \mathbb{R}^2 \times \mathbb{R}^5_+ \times [0,1]^3$, with $\omega = \langle\, \theta, \bar{x}, \alpha, \sigma, \phi, c, D, \beta, \gamma, p \,\rangle$ its generic element.

[18] Tie-breaking rules have no bearing on the results due to the continuum-player specification of the model.

The result is in line with the findings of [Engle et al., 2021], and the interpretation is similar. If an increase in the aggregate compliance A unambiguously reduces the risk of contagion in social interactions, then the propensity to comply decreases as high compliance rates are expected. Public health becomes a public good, and its efficient provision may be hindered by free-riding. Our analysis, however, complements and clarifies the result by characterising *who* free-rides: not surprisingly, the citizens that risk less upon infection are those with the lowest propensity to comply, for they can free-ride on the (costly) effort of citizens that risk more and opt observe social distancing for self-protection. Note indeed from (23) that the individual incentive to implement social distancing strictly increases in $x(i)$ via the discounted cost of death post-infection $\tilde{D}_i$. This is sensible: the incentive to (self-)protect from contagion is larger the higher is the probability of death upon getting infected. As a consequence, the propensity of older/weaker citizens to abstain from social interaction is higher than that of younger/healthier ones, *ceteris paribus*. Notice further that $\Delta_i(A)$ is continuous in $x(i)$, so that it is sensible to guess that, in equilibrium, individual decisions to comply be governed by *monotone strategies* – contingent to the (observed) demographic profiles $x(i)$. Consistently, we characterise the (unique) equilibrium of the social-interaction game via a guess-and-solve approach, whereby we postulate that

$$x(i) \geq \hat{x}(A; \boldsymbol{\Omega}, \mathbf{E}) \qquad \Longrightarrow \qquad a(i) = 1$$

holds, in equilibrium, for all $i \in [0,1]$ that never showed disease-specific symptoms and for every arbitrary compliance rate $A \in [0,1]$ – with $\hat{x}(A; \boldsymbol{\Omega}, \mathbf{E}) \in \mathbb{R} \cup \{+\infty\}$ an arbitrary threshold value. If this is the case, then the aggregate compliance rate can be written as a function of the arbitrary threshold $\hat{x}(A; \boldsymbol{\Omega}, \mathbf{E})$, for

$$A(\hat{x}(\cdot)) = \Phi\left(\frac{\bar{x} - \hat{x}(A; \boldsymbol{\Omega}, \mathbf{E})}{\sigma}\right) \tag{24}$$

holds due to the continuum-player specification[19]. We prove that, for any parameterisation $\boldsymbol{\Omega}$ and every configuration of the epidemiological aggregates $\mathbf{E}$, there exists a unique value $A = A^*$ of the

[19] For the sake of formal rigour, (24) holds almost surely, for it involves the evaluation of the aggregate/average realization *ex post* of a continuum of i.i.d. random variables – see the *caveat* outlined in Footnote 4 for additional details and references.

aggregate compliance rate such that

$$A\Big(\hat{x}\left(A^*;\boldsymbol{\Omega},\mathbf{E}\right)\Big) \;=\; A^*$$

holds. Moreover, since $A\left(\hat{x}\left(\cdot\right)\right)$ is a monotone function of $\hat{x}\left(\cdot\right)$, there exists a unique threshold value $x^*\left(\boldsymbol{\Omega},\mathbf{E}\right)$ consistent with A^*. The result is summarised by Propositions 2 and 3 for $\phi > 1$ and $\phi \in [0,1]$, respectively.

PROPOSITION 2. *Let $\phi > 1$ hold. Then, for any parameterisation $\boldsymbol{\Omega} \in \mathcal{O}$ and any configuration of the epidemiological aggregates $\mathbf{E} \in \mathcal{E}$, there exists a critical value $\underline{ASY}\left(\boldsymbol{\Omega},\mathbf{E}\right)$ of the total mass of asymptomatic infected citizens such that*

$$A^*\left(\boldsymbol{\Omega},\mathbf{E}\right) \;=\; \begin{cases} 0 & \text{if } ASY \leq \underline{ASY}\left(\boldsymbol{\Omega},\mathbf{E}\right) \\ A\left(\,x^*\left(\boldsymbol{\Omega},\mathbf{E}\right)\right) \in (0,1) & \text{if } ASY > \underline{ASY}\left(\boldsymbol{\Omega},\mathbf{E}\right) \end{cases}, \tag{25}$$

where the i-th citizen complies with social distancing norms if $x(i) \geq x^*\left(\boldsymbol{\Omega},\mathbf{E}\right)$. The marginal type is $x^*\left(\boldsymbol{\Omega},\mathbf{E}\right) = +\infty$ when $A^* = 0$, and it is identified by the indifference condition

$$\lambda^2 D\left[\frac{\Phi\Big(\theta + \alpha\, x^*\left(\boldsymbol{\Omega},\mathbf{E}\right)\Big)}{1-\lambda\left(1-\gamma\right)\Big(1-\Phi\Big(\theta+\alpha\, x^*\left(\boldsymbol{\Omega},\mathbf{E}\right)\Big)\Big)}\right]\beta\, G\big(A^*\left(\boldsymbol{\Omega},\mathbf{E}\right)\big) \;=\; c \tag{26}$$

when $A^* \in (0,1]$.

Proof. See Appendix A. □

PROPOSITION 3. *Let $\phi \in [0,1]$ hold. Then...*

TO BE WRITTEN

Proof. See Appendix A. □

4. Microfounded Behavioural SIR

In this section we simulate a discrete-time SIR model augmented with the fully microfounded behavioural responses characterized in Section 2 as static Nash equilibria of the social-interaction game. Our goal here is to study how the endogenous (equilibrium) level of compliance with mandatory social distancing affects – provided it does – the diffusion process of a stylised epidemic with known structural characteristics. To this end, we enrich an otherwise standard SIR framework by assuming that, in each period t: (i) the mass of new infected NI_t be determined by the equilibrium level of compliance $A_t^* \in [0,1]$ arsing from the social interaction game; (ii) the epidemiological aggregates $\mathbf{E}_t \equiv \langle ASY_t, S_t, R_t \rangle$ be consistent with the entire history of past equilibria. What we obtain are *naïve dynamics*, where the evolution in time of the aggregates is driven by the collective, heterogeneous decisions of *myopic* optimisers – that best-respond to contingent environmental and strategic conditions, but fail to internalize the future effects of their actions[20]. Recall that, by assumption, each player of our social-interaction game (except when he/she recovered from a symptomatic infection) ignores his/her epidemiological state, and must therefore resort to observable aggregates to form meaningful (common) subjective beliefs about that. (For instance, as in [Engle et al., 2021] the subjective probability of being infected and asymptomatic in t coincides with the instantaneous prevalence population-wide π_t.) As a consequence, individuals are assumed to be both myopic and *forgetful* – each being able to keep of track of the aggregates but not of his/her individual past behaviour. Both assumptions are extreme and admittedly unrealistic, but essential to ensure the analytical tractability of the model. They are further discussed in Section 5, where we partially relax forgetfulness and highlight the – somehow surprising – relevance of individual beliefs in the determination of strategic incentives.

4.1 Model (Highlights) and Calibration

Consider an environment identical to that outlined in Section 2 but for the fact that interactions are indefinitely repeated over time – that is discrete and indexed by $t = 0, 1, 2 \ldots$ Within each period, the sequence of events that trigger the instantaneous variations in the epidemiological aggregates

[20] [Engle et al., 2021] argue that myopia is consistent with (equilibrium) forward-looking behaviour in a continuum-player differential game – see footnote 12, page 7. We are not sure whether their argument readily extends or not to our model specification, for it significantly differs from that outlined in their paper.

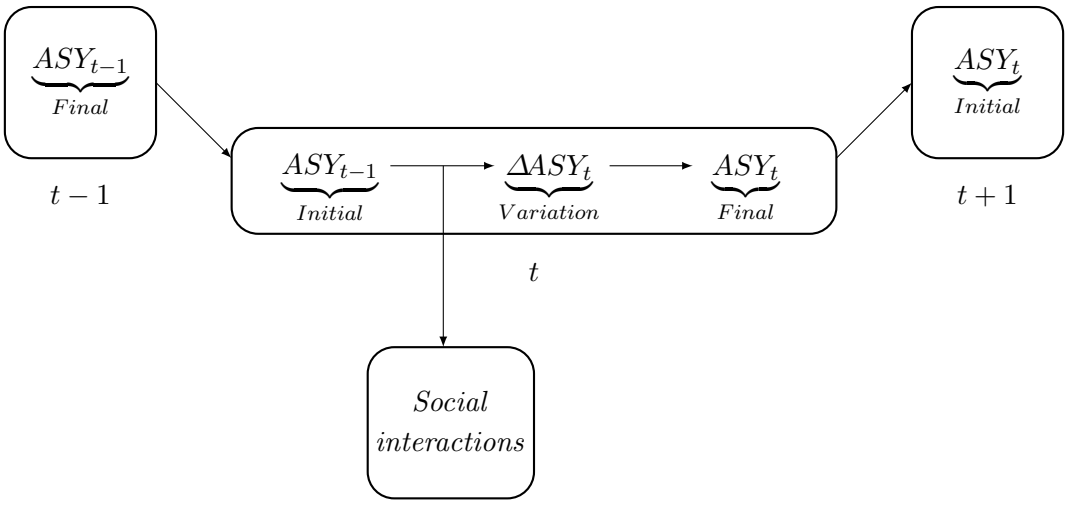

Figure 2. The within-period sequential structure that determines the (action-contingent) instantaneous variation ΔASY of the asymptomatic – with $\Delta ASY = p\,(NI_t - ND_t - NR_t)$ and ND_t and NR_t the masses of new dead and recovered, respectively.

unfolds in three sub-periods: initial conditions are set in the first, social interactions – if any – take place in the second, and consistently determine within-period variations in the third. Each period t inherits initial conditions from $t-1$, and provides $t+1$ with new ones. Figure 2 provides a graphical representation of the within-period sequential structure of events that determine the law of motion of the *ASY*mptomatic infected. Our augmented model shares many of its equations with a standard SIR: to avoid redundancy, we omit here a detailed description of the model specification – that is postponed to Appendix C. Rather, in the following subsection we present in detail its most 'exotic' characteristics: (i) the endogenous infection-passing mechanisms that determines the *incidence rate* of the disease[21] – hence, the within-period mass of *N*ew *I*nfected NI_t; (ii) the cumulation process for *D*ead and *R*ecovered. The calibration we use for the simulations is presented in Table 1 below.

4.2 Infection-Passing & Cohort-Specific Mortality

Within-period *N*ew *I*nfected are determined by the social interactions between *ASY*mptomatic infected and *S*usceptible citizens that opted for non-compliance with social-distancing norms. Recall, that, in case of interaction, an infected citizen passes the disease to a susceptible one with probability β. Moreover, since all citizens ignore their epidemiological state by assumption, (non-)compliance

[21] In a continuum-player framework where the initial mass of agents is normalized to one, the distinction between masses and rates is shaded and generally immaterial for the analysis.

(i)

Parameter	Value	Definition
$\overline{x}$	-1.5	Median/average demographic profile
σ^2	4	Demographic heterogeneity
θ	-2	Disease-specific mortality
α	1	Sensitivity of mortality to demographic traits
β	1	Disease contagiousness
γ	0.4	Scaling coefficient of the probability of recovery
p	0.7	Fixed share of asymptomatic infected
λ	0.99	Time discount factor
D	150	Individual cost of death

(ii)

Aggregate	IC	Definition
Pop_0	1	Population
I_0	0.001	Infected
S_0	0.999	Susceptibles
D_0	0	Dead
R_0	0	Recovered

Table 1 *(i) Calibration of the main parameters (ii) Initial conditions (ICs) for the aggregates*

is *not* state-contingent. With a total mass of interacting susceptibles equal to $S_t\left(1 - LA_t^*\right)$, the mass of new infected NI_t can be easily defined as

$$NI_t \;=\; \underbrace{S_t\Big(1 - LA_t^*\Big)}_{\text{Interacting } \mathcal{S}\text{usceptibles}} \times\, \beta\, G\left(A_t^*\right) \tag{27}$$

with $\beta > 0$ the disease-specific passing chance upon interaction, $L \in [0, 1]$ the share of strategic citizens, and where coefficient $\phi \geq 0$ scales the congestion externality. The law of motion of the $\mathcal{ASY}$mptomatics can be written as

$$ASY_{t+1} \;=\; ASY_t + p\Big(NI_t - ND_t - NR_t\Big)\;, \tag{28}$$

with $p \in (0, 1]$ the probability of being asymptomatic after infection, and where ND_t and NR_t are, respectively, the within-period new $\mathcal{D}$ead and $\mathcal{R}$ecovered calculated according to their cohort-

specific rates – see below. Note from (C.7) that the congestion externality $\varphi(A)$ that scales the aggregate probability of infection is the same that appears in the *individual* probability of infection that citizens use in (22) to asses the riskiness of non-compliance. Thus, every citizen internalizes the spillover by accounting for it in his/her optimal choice, for he/she '(fore)sees' the other citizens through the (anticipated) average/aggregate compliance rate A_t^*. Recall further that, in equilibrium, when the risk of infection in social interaction increases, the older/weaker citizens start complying with social-distancing norms. When this is the case, the demographic composition of the cohort of new infected NI_t ceases to be aligned with that of the entire population, for only younger/healthier citizens actually become (new) infected. The rates of mortality and recovery must be adjusted accordingly. In particular, assuming that the demographic composition of the share $(1-L)$ of exempted citizens is aligned with that of the population, the endogenous mortality rate population-wide $\bar{Q}^D(x_t^*)$ can be written as

$$\bar{Q}^D(x_t^*) = \rho \underbrace{\mathbb{E}\Big[\Phi(\theta+\alpha x) \,\Big|\, x \geq x_t^*\Big]}_{\text{Cohort-specific mortality}} + (1-\rho)\,\bar{Q}^D \tag{29}$$

where x_t^* is the equilibrium demographic threshold for compliance with social distancing, $\bar{Q}^D$ is the median/average mortality population-wide – see equation (4) –, and $\rho \in (0,1]$ is the relative weight of the cohort of new infected in the whole interacting population $(1-LA_t^*)$, i.e.

$$\rho = \frac{L(1-A_t^*)}{1-LA_t^*} \ .$$

Note that a model-consistent treatment of the endogenous evolution of cohort-specific mortality rates is *essential* for a proper analysis of model dynamics. If the cohorts of new infected are, on average, younger and healthier than the average citizen, their mortality will be below-average, too. Accordingly, above-average will be both the probability of recovery *and* the probability of remaining infected in the future. Younger/healthier infected therefore die less, but remain infected and infective for a longer time span, thus prolonging in time the propagation of the disease.

4.3 The Standard SIR Benchmark: A Note

It is commonplace in the analysis of behavioural SIR models to compare and contrast the results of the augmented specification with a benchmark SIR deemed 'standard'. But how do we define how such 'standard-SIR' benchmark? In [Engle et al., 2021] the SIR benchmark model is conceptually similar to the *laissez faire* scenario considered by [Toxvaerd, 2020], in that it entails no disease-containment by the policy-maker, nor self-protection by the citizens via self-isolation. The authors, therefore, seem to implicitly consider as *the* benchmark case an 'unrestricted epidemic' scenario, where the diffusion process evolves according to its natural dynamics. However, another benchmark is available: that in which social distancing is implemented by automaton-like 'agents' that implement any policy prescription they are faced with. This second benchmark case is similar in spirit to the optimal-control problem studied by [Alvarez et al., 2021]. The good news is: our specification of the augmented (behavioural) version subsumes *both* standard-SIR benchmarks as special cases. The unrestricted-epidemic benchmark corresponds to a calibration with $L = 0$ – i.e. to a scenario in which all citizens are non-strategic and ignore social-distancing norms by construction. The automaton-citizens scenario corresponds to a calibration with $c < 0$[22] – i.e. to a scenario in which compliance with norm is always a dominant strategy, whatever the value of L.

4.4 A Look at Naïve Dynamics

Using the calibration presented in Table 1, we now simulate our augmented behavioural SIR model and study its naïve dynamics. Some general intuitions are (qualitatively) derived via simple comparative-statics exercises. First, we compare the equilibrium-augmented specification with a standard-SIR benchmark where all citizens comply with norms – the simulations are presented in Figures 3, 4 and 5 with $L = 0.1$ and ϕ set to 0.3, 1 and 3, respectively[23]. It is apparent from both figures that the standard SIR significantly overestimates the effectiveness of social distancing. The result corroborates those of [Engle et al., 2021] and [Farboodi et al., 2021] by confirming their robustness to alternative model specifications. As a second exercise of comparative statics, we now

[22] Recall that $c = b - k$, where $b > 0$ is a fixed benefit from social activity, and $k > 0$ is the fine for cheating on norms. The calibration $c < 0$ thus entails that $k > b$. In words: this is an hypothetical scenario in which the enforcement by the public authority is so effective that non-compliance is strongly dominated by compliance for every configuration of the epidemiological aggregates, and $A^* = 1$ for every t.

[23] With an initial condition $I_0 = 0.001$ the epidemic stops almost immediately in the SIR with $L > 0.3$.

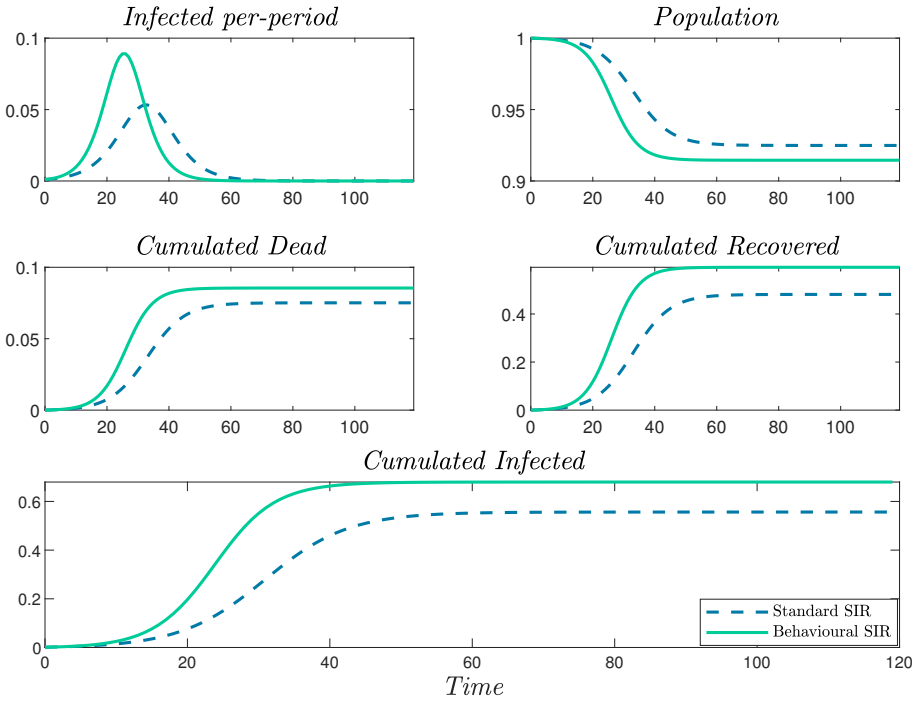

Figure 3. Dynamics of the main aggregates: standard vs. augmented SIR with $L = 0.1$ – $\phi = 0.3$.

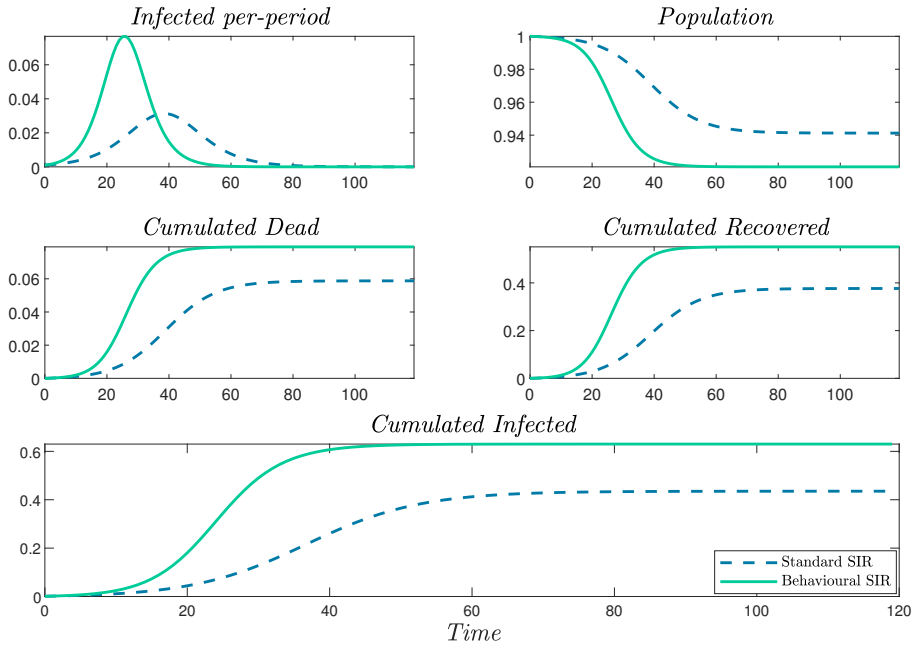

Figure 4. Dynamics of the main aggregates: standard vs. augmented SIR with $L = 0.1$ – $\phi = 1$.

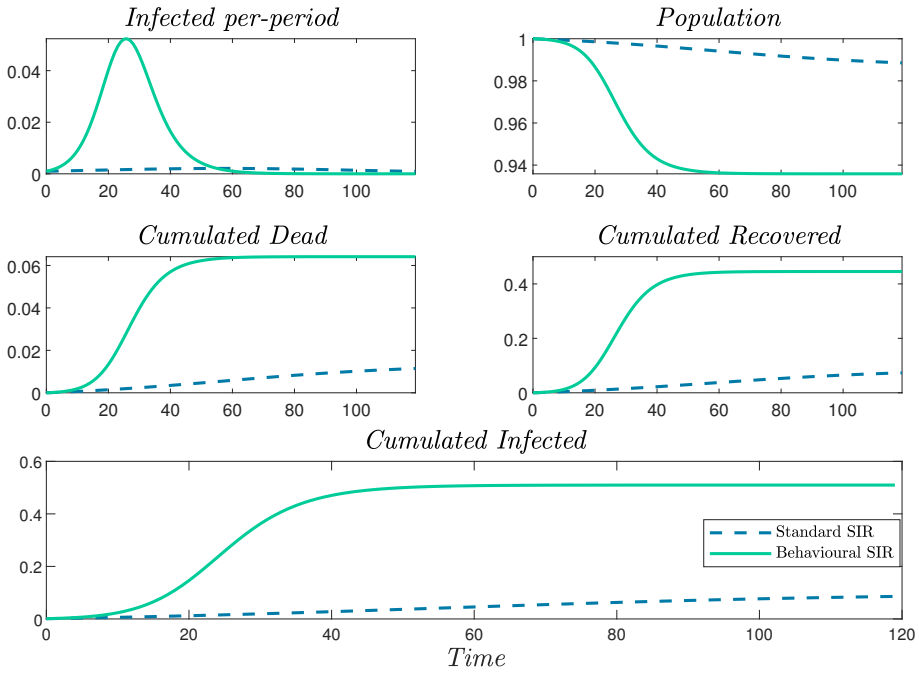

Figure 5. Dynamics of the main aggregates: standard vs. augmented SIR with $L = 0.1$ – $\phi = 3$.

simulate the augmented model with different masses of exempted citizens $(1 - L)$. Albeit in the narrative proposed in Section 2.4 the parameter L indicates the share of citizens that do not provide essential services, with a slight abuse of interpretation the same parameter can be seen as a proxy for the 'strictness' of the lockdown. That is, we can interpret L as policy design – how many citizens are required to stay at home – and k as the intensity of enforcement – how lenient/aggressive the policy-maker is at monitoring compliance and punishing deviations. The simulations are shown in Figure 6 and 7. Their inspection immediately reveal that the stricter the lockdown is (larger L), the more effective it is at containing the spread of the disease. As L increases, peak-prevalence decreases and the epidemic ends sooner. The other aggregates vary consistently – as L increases the total mass of infected decreases, and so do the cumulated dead. When looking at compliance rates (Figure 7), another phenomenon emerges: as total and new infected decrease with L, so does the equilibrium compliance rate A_t^*. A group-size effect of the kind identified by [Olson, 1965] seems to be present: as L decreases the mass $1 - L$ of citizens that do not observe social distancing for sure increases and, to contain risk – both individual and aggregate – higher level of compliance are needed. In other words, as the group size decreases, the marginal effect of small variations in

collective behaviour on the global payoff externality increases, spurring agents' propensity to 'co-operate for the collective good'. Third, and last, we compare a scenario in which social distancing

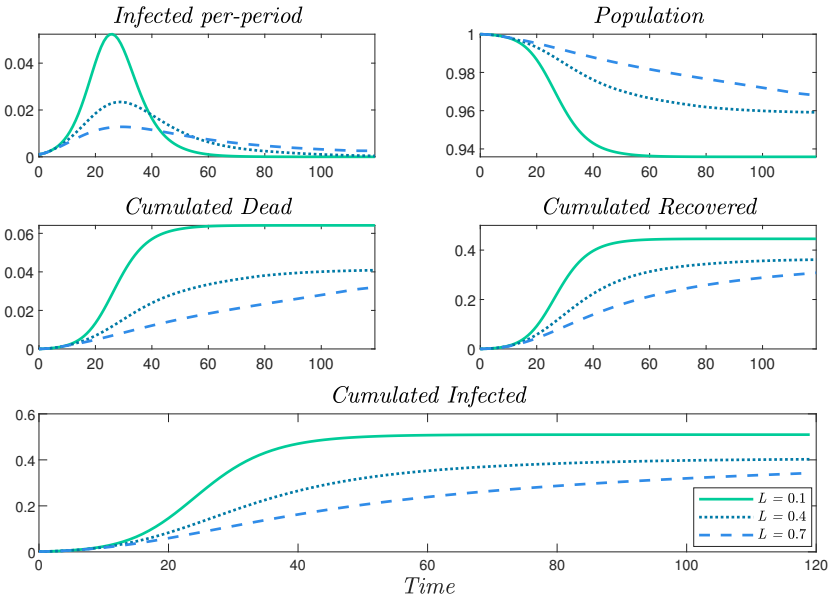

Figure 6. Dynamics of the main aggregates: standard vs. augmented SIR with several values of $L - \phi = 3$.

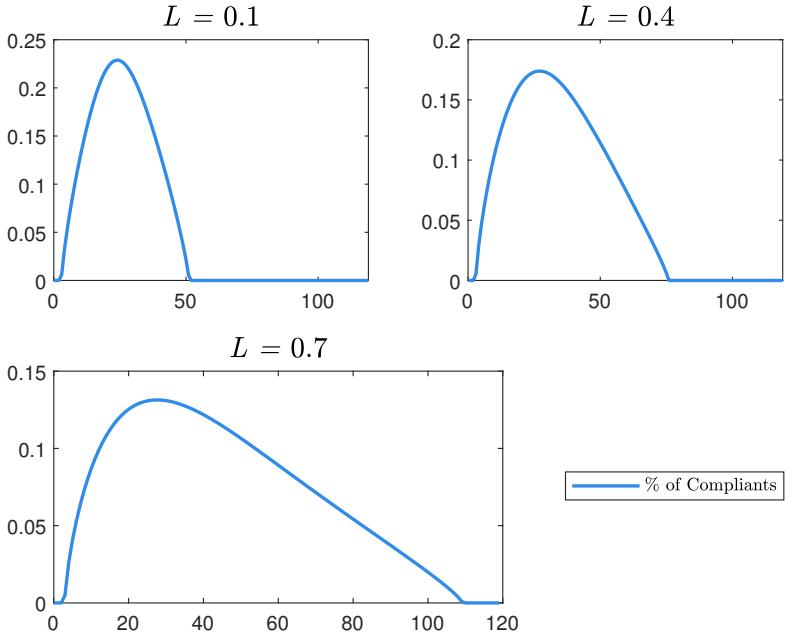

Figure 7. Aggregate/average equilibrium compliance rate A_t^* underlying the dynamics of the aggregates shown in Figure 6 – $\phi = 3$.

is enforced by the policy-maker via command-and-control, with a no-enforcement scenario where citizens may reduce their risk of infection by *willingly* implementing self-isolation. We calibrate the benefit from social activity as $b = 0.8$, and simulate the scenario with centralized enforcement with a fine for cheating $k = 0.6$ – see equation (13). The implicit cost of compliance is therefore $c_E = 0.2$ in the scenario with enforcement, and $c_{NE} = b = 0.8$ in the self-isolation scenario. The dynamics pf the main aggregates, and the corresponding dynamics of the equilibrium level of compliance A_t^*, are shown in Figure 8 and 9 respectively. As expected, by reducing the implicit cost of compliance faced by the citizenry active enforcement of social distancing by the policy-maker spurs compliance.

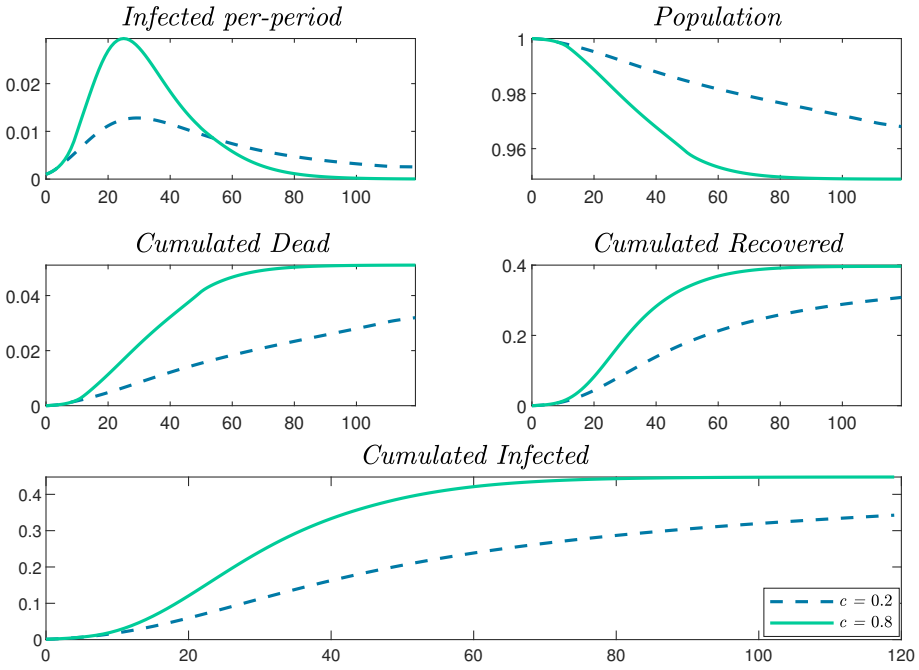

Figure 8. Dynamics of the main aggregates: enforcement vs. no enforcement – $\phi = 3$.

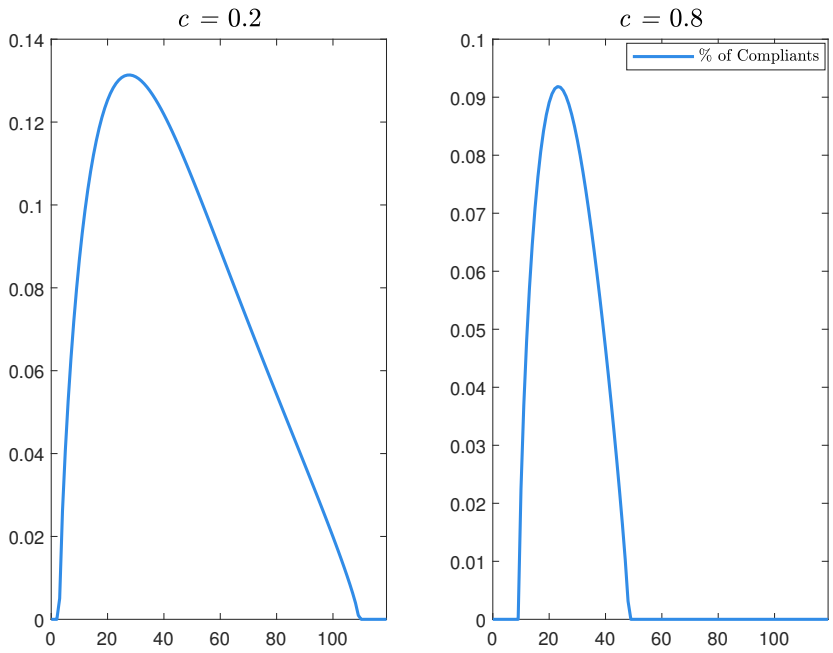

Figure 9. Aggregate/average equilibrium compliance rate A_t^* underlying the dynamics of the aggregates shown in Figure 8 – enforcement vs. no enforcement.

5. The Role of Individual Beliefs

In line with [Engle et al., 2021], in order to be able to characterise analytically the (unique) Nash equilibrium of our static social-interaction game, we resorted in Section 2 to an extreme form of forgetfulness, whereby citizens' *past* social behaviour is assumed to have no bearing at all on the beliefs they hold about their *current* (unobserved) susceptibility to infection. As a consequence, the citizenry is forced to rely on the observation of aggregates to be able to form meaningful beliefs. In this Section we highlight how crucial forgetfulness is in the determination the strategic incentives faced by the citizenry, and study how its (partial) relaxation affects the equilibrium characterisation outlined in Section 3. We show that when citizens are forgetful and hold common beliefs, individual decisions to comply with social-distancing norms are always strategic substitutes. When some form of recall is present, and subjective beliefs begin to diverge because of path-dependence, both complementarity and substitutability may arise in equilibrium.

5.1 Path-Dependence and the Evolution of Subjective Beliefs

At the onset of an epidemic information may be scarce and imprecise, and rationality and attention may be impaired by panic. Doubtlessly far from realism *tout court*, the forgetfulness assumption is somehow fair in such environment: unable to keep track of all past interactions, occurred while the virus was operating undercover, citizens have no suitable alternative for belief-formation but to resort to the public information about the epidemiological aggregates disclosed by the media. As time passes and the epidemics evolves, however, this ceases to be the case, for in the long run is individual behaviour – past and present – that shapes the odds of each epidemiological state $e(i)$, not the instantaneous aggregates. When subjective beliefs become path-dependent, their heterogeneity in the cross-section of the citizenry cannot but increase. Figures 6 and 7 clearly reveal that the median/aggregate compliance rate in equilibrium, A^*, peaks around 25%: this means that a significant share of younger/healthier citizens never complies throughout the entire epidemic. Recall from Subsection 2.3 that, at every date t, the (instantaneous) risk of contagion is null if social distancing is implemented, and strictly positive and equal to $\beta\, G\left(A_t^*\right)$ otherwise – with $G(A)$ defined by (10) and (19). The subjective, path-dependent probability of being susceptible in t, conditional to the entire history of individual behaviour, can be written as

$$\Pr\left(e_t(i) = S \,\middle|\, \left\{ a_\tau^*(i) \right\}_{\tau=0}^{t-1} \right) \;=\; \prod_{\tau=0}^{t-1} \left[1 - \left(1 - a_\tau^*(i) \right) \left(\beta\, G(A_\tau^*) \right) \right] . \tag{30}$$

Using (30) we can easily write the probability of being susceptible in t after a history of full non-compliance as

$$\Pr\left(e_t(i) = S \,\middle|\, a_\tau^*(i) = 0 \text{ for all } \tau < t \right) \;=\; \prod_{\tau=0}^{t-1} \left[1 - \left(\beta\, G(A_\tau^*) \right) \right] .$$

that quickly converges to zero as t increases – four simulations with fixed probabilities of infection are presented in Figure 10. Conditioning on survival, and after a sufficiently large number of periods post-infection, the probability of being recovered from a previous infection is therefore close to one. The situation is less clear-cut for those (older/weaker) citizens that always observe social distancing norms in the central part of the epidemic. Albeit less compelling, a similar reasoning extends to these individuals. In Subsection 5.2 we study how path-dependence in subjective beliefs may affect

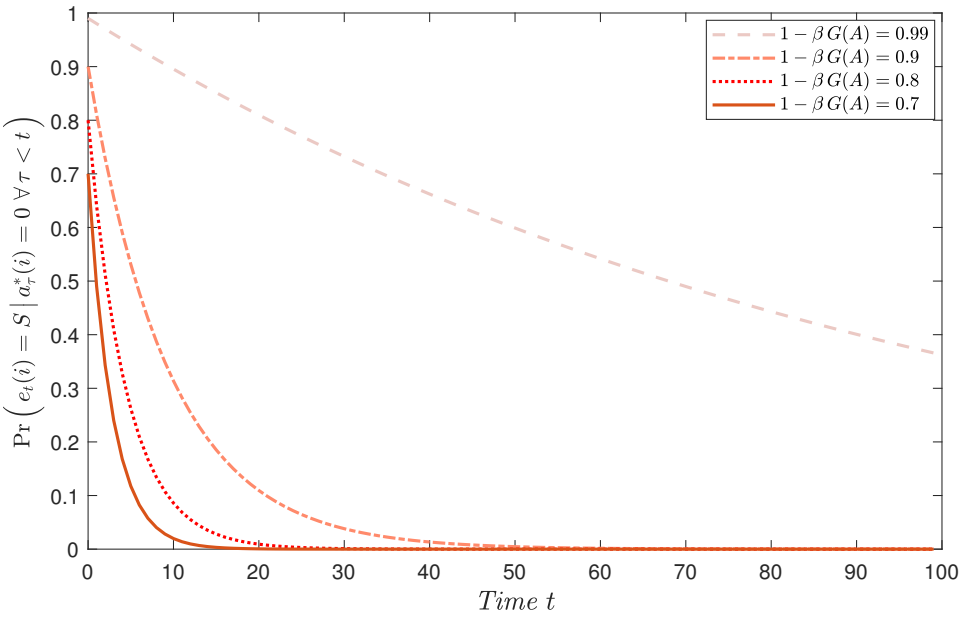

Figure 10. Probability of the being susceptible after t periods of non-compliance with fixed probabilities of infection $\beta\,G(A)$.

the equilibrium behaviour of citizens in the long run via a slightly modified specification of the (static) social-interaction game outline and extensively discussed in Sections 2 and 4.

5.2 The Social-Interaction Game, Once More

Consider again the social-interaction game outlined in Section 2, but with a slightly modified setup in which all epidemiological types $e(i)$ are assumed to be common knowledge. In light of the argument outlined in Subsection 5.1, the common-knowledge assumption can be interpreted *cum grano salis* as a limit case that approximates the informational state to which the citizenry tends to converge in time – as the epidemic evolves and additional information is extracted from past behaviour. It is immediate to notice that, in such an environment, strategic uncertainty applies to susceptible citizens only: absent any risk of reinfection, compliance with social distancing is a (strongly) dominated strategy for all asymptomatics and recovered. As a consequence the total mass $M(A)$ of interacting individuals can be written as

$$M_{CK}(A) \;=\; ASY + R + S\,(1 - LA)\;\;, \tag{31}$$

where the subscript *CK* stands for Common Knowledge. It is apparent from (31) that, differently from the original specification of the interaction game – where the epidemiological states were unknown –, the aggregate compliance A rate here only affects the mass of susceptible citizens that opt for non-compliance with social-distancing norms. When it comes to the global externality $G(A)$ that parameterises the individual risk of infection, this is a crucial element: while the differential effect of A on different types of citizens has no particular bearing on the congestion externality $\varphi_{CK}(A)$ – i.e. the extensive-margin component

$$\varphi_{CK}(A) \;=\; \Big[\, ASY + R + S\big(1 - LA\big) \,\Big]^{\phi} \,, \tag{32}$$

that continues to be strictly decreasing in A, it dramatically changes the behaviour of group-specific prevalence $\pi_{CK}(A)$ – the intensive-margin component

$$\pi_{CK}(A) \;=\; \frac{ASY}{ASY + R + S\,(1 - LA)} \tag{33}$$

that now *strictly increases in* A. Differentiating (33) with respect to A, it is indeed immediate to check

$$\frac{\partial}{\partial A}\,\pi_{CK}(A) \;=\; \frac{L\,(S)\,(ASY)}{\Big[\, ASY + R + S\,(1 - LA) \,\Big]^{2}} \;>\; 0 \;. \tag{34}$$

Again, the interpretation is straightforward, for it hinges on the argument extensively discussed in 2.3.2: if some types of agents systematically ignore social-distancing norms more frequently than others, the group-specific prevalence of the disease (co-)varies with the mass of interacting individuals. When the epidemiological types $e(i)$ are known, asymptomatics and recovered systematically ignore social distancing, while susceptibles choose strategically whether or not to comply with norms. As a consequence, any increase in the aggregate compliance rate A amounts to a decrease in the mass of susceptible individuals within the interacting (sub)population, hence to an *increase* in the group-specific prevalence of the disease, *ceteris paribus*. The overall effect on the risk of contagion is therefore ambiguous *a priori*, for

$$\frac{\partial}{\partial A}\,G_{CK}(A) \;=\; \varphi_{CK}(A)\left(\frac{\partial}{\partial A}\,\pi_{CK}(A)\right) + \pi_{CK}(A)\left(\frac{\partial}{\partial A}\,\varphi_{CK}(A)\right) \;\gtrless\; 0$$

holds. Recall that the propensity to comply of a citizen with demographic profile $x(i)$ is parameterised, in binary-action game, by the payoff differential $\Delta_i(A)$ – see definition (23). Differentiating $\Delta_i(A)$ with respect to A under the common-knowledge assumption, we obtain the following result.

PROPOSITION 4. *Let the epidemiological types $e(i)$ be common knowledge. Then, for any calibration $\mathbf{\Omega} \in \mathcal{O}$ and any configuration $\mathbf{E} = \langle ASY, S, R \rangle$ of the epidemiological aggregates, the individual decisions to comply are:*

(i) strategic complements, if $\phi \in [0, 1)$;

(ii) independent, if $\phi = 1$;

(iii) strategic substitutes, if $\phi > 1$;

where $\phi \geq 0$ is the scaling coefficient of the congestion externality $\varphi_{CK}(A)$ defined by (32), and with $A \in [0, 1]$ the median/aggregate compliance rate with-social distancing norms.

Proof. See Appendix B. □

The interpretation is the following. Recall from Subsection 2.3 that $\varphi(A) \in [0, 1]$ is a congestion externality that summarizes how citizens' aggregate (non-)compliance affects the individual risk of contagion via overcrowding – the extensive margin effect, see expression (8). As the median/aggregate compliance rate A decreases, more and more citizens interact socially. In doing so, they add to the (over)crowding of the environment in which social interactions do occur. Depending on the scaling coefficient $\phi \geq 0$, congestion may arise quickly ($\phi < 1$) or slowly ($\phi > 1$), but the net effect on individual risk is unambiguous: the larger the number of interacting individuals, the higher the risk of infection faced by a non-compliant susceptible, *ceteris paribus*. When the epidemiological states are known, ignoring social distancing is a dominant strategy for all *ASY*mptomatics and *R*ecovered, so that any increase (decrease) in the compliance rate A amounts to a decrease (increase) in the mass of susceptible citizens in the interacting (sub)population, hence in an increase (decrease) in the group-prevalence $\pi_{CK}(A)$ – see the discussion above. With $\phi \in [0, 1)$ congestion is severe, so that the effect along the extensive margin dominates that on the intensive one: as more individuals opt for compliance, group-specific prevalence increase, but the corresponding reduction in congestion is more than offsetting.

5.3 Aggregate Effects of Strategic Complementarity

When strategic complementarity prevails, the equilibrium characterisation is less clear-cut than in the presence of pure strategic substitutability. Complementarity typically invites equilibrium multiplicity, and our interaction game is not an exception. Preliminary analysis shows that the set of equilibria of this more complex game may be structured as follows.

CLAIM 1. *Let the epidemiological types $e(i)$ be common knowledge. Then the set of the (pure-strategy) Nash equilibria of the social-interaction game is structured as follows:*

(i) if $\phi \geq 1$ the game has a unique equilibrium similar to that characterized in Proposition 2;

(ii) if $\phi \in [0, 1)$, the game has multiple equilibria, structured as follows:

a) if $ASY \leq \underline{ASY}_{CK}(\mathbf{\Omega}, \mathbf{E})$, then non-compliance is (strongly) dominant for all citizens, and $A^*(\mathbf{\Omega}, \mathbf{E}) = 0$ holds;

b) if $\underline{ASY}_{CK}(\mathbf{\Omega}, \mathbf{E}) < ASY < \overline{ASY}_{CK}(\mathbf{\Omega}, \mathbf{E})$, then the game has a symmetric Nash equilibrium with $A^*(\mathbf{\Omega}, \mathbf{E}) = 0$ and two monotone equilibrium: one stable ($A^*_S(\mathbf{\Omega}, \mathbf{E})$) and one unstable ($A^*_{NS}(\mathbf{\Omega}, \mathbf{E})$), with $A^*_S(\mathbf{\Omega}, \mathbf{E}) > A^*_{NS}(\mathbf{\Omega}, \mathbf{E}) > 0$;

c) if $ASY > \overline{ASY}_{CK}(\mathbf{\Omega}, \mathbf{E})$, then the game has a symmetric Nash equilibrium with $A^*(\mathbf{\Omega}, \mathbf{E}) = 0$, and a stable monotone equilibrium $A^*_S(\mathbf{\Omega}, \mathbf{E}) > 0$.

Arbitrarily selecting the stable monotone equilibrium $A^*_S(\mathbf{\Omega}, \mathbf{E})$ as *the* equilibrium of the interaction game when the aggregate mass of asymptomatic infected exceeds the critical value $\underline{ASY}(\mathbf{\Omega}, \mathbf{E})$, we simulate an augmented SIR model with endogenous compliance of the type studied in Section 4, but allowing now for both substitutability ($\phi = 3$) and complementarity ($\phi = 0.3$). Figures 11 and 12 compare the corresponding dynamics of the main epidemiological aggregates, as well as the (endogenous) compliance rates that pin them down. Albeit purely qualitative, the results are (very) sensible. As a general stance, strategic substitutes can be thought as self-moderating mechanisms, for they embed a natural incentive to "lean against the wind". Strategic complements, on the contrary, tend to induce cascades and bandwagons via the multiplier effect implicit in their structure of incentives. Our simulations show that such mechanics may indeed be at work in our

model. Strategic complements display a sudden and significant increase in the aggregate compliance rate at the very beginning of the epidemic, accompanied by a sudden contraction: once compliance plummets to zero (relatively early, around $t = 50$), it remains there forever. When compared with complements, strategic substitutes display smoother dynamics: compliance takes over slowly, peaks around $t = 50$ similarly to the complementarity scenario, and decreases steadily but slowly. Recall that out thesis is the following: as time passes and the epidemic evolves, the nature of the strategic incentives faced by citizens when they are called to decide whether or not to implement social distancing undergoes a phase transition, whereby strategic substitutability *may* turn into complementarity. If this is the case, a behavioural SIR where the endogenous compliance mechanism is steadily regulated by strategic substitutes may systematically over-estimate average compliance rates in the final part of the epidemic.

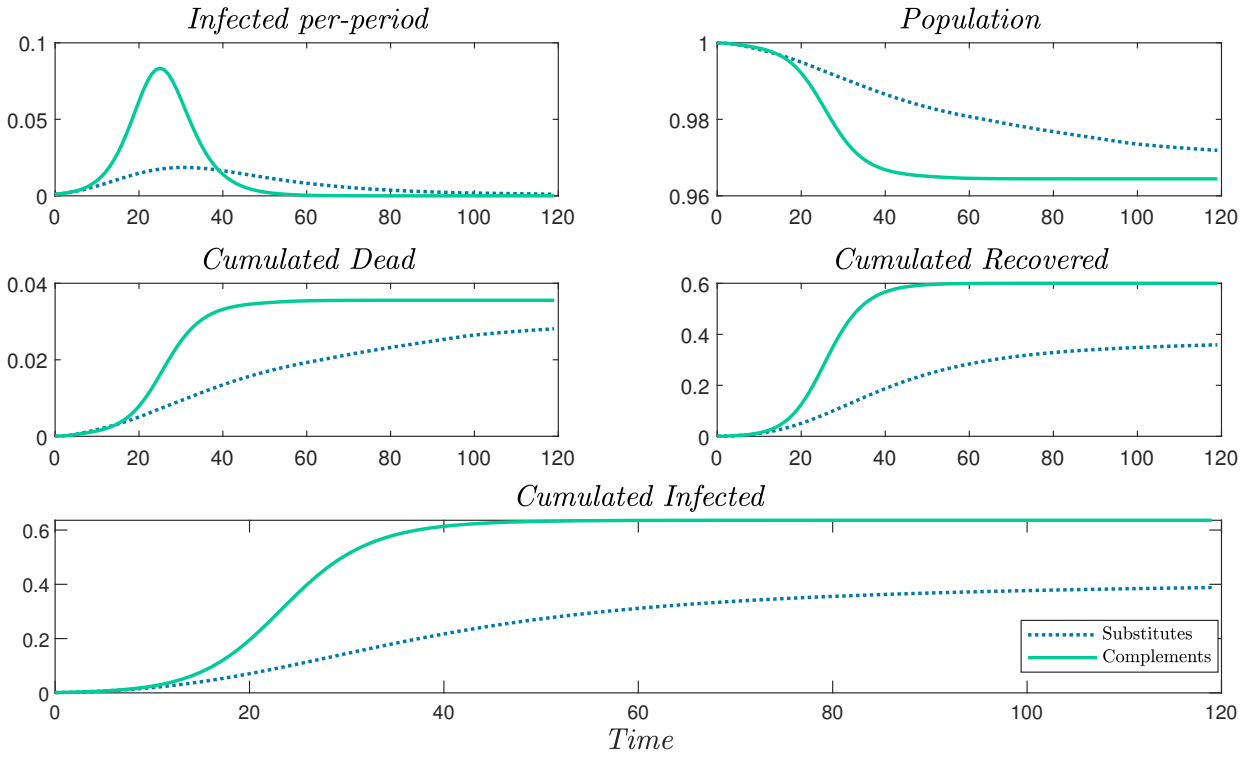

Figure 11. Dynamics of the main aggregates. The calibration is the same used for the simulations presented in Section 4. Strategic complementarity and substitutability in compliance is induced by setting $\phi = 0.3$ and $\phi = 3$, respectively.

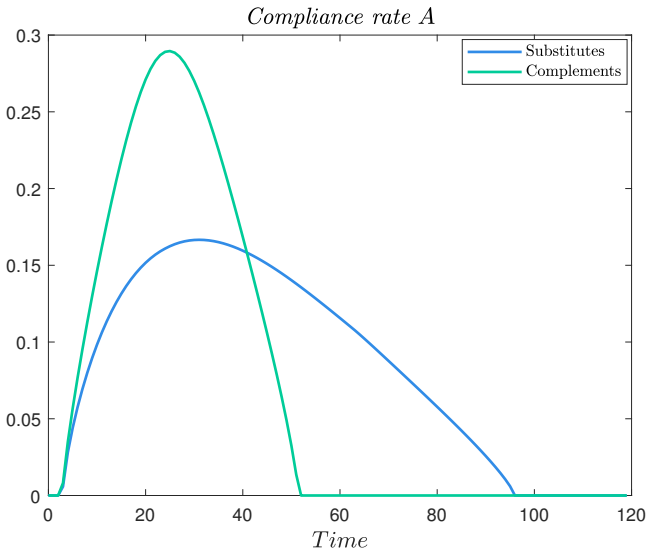

Figure 12. Evolution in time of the aggregate compliance rate A underlying the dynamics of the epidemiological aggregates presented in Figure 11.

6. Summary & Conclusions

In this paper we outlined a parsimonious, microfounded model of social interaction between risk-neutral, demographically heterogeneous agents. In order to contain the spread of an infectious disease, a benevolent policy-maker imposes mandatory social distancing to a large population of rational, atomistic citizens. Policy-design and enforcement are centralised, but implementation is decentralised and uncoordinated: all individuals are allowed to decide autonomously whether or not to comply with policy prescriptions – equivalently, whether or not to abstain from (risky) social interactions. Compliance is riskless, socially beneficial but individually costly. Non-compliance is costless, but entails an *aggregate* risk of infection proportional to the social distancing implemented by others. In choosing which action to take, a citizen trades off the direct cost of compliance with his/her idiosyncratic (expected) cost of death upon infection – the latter determined by his/her demographic profile. Citizens are strategically sophisticated, and rationally anticipate others' choices via equilibrium-consistent second-guessing.

Our model is tractable and relatively flexible, but highly stylized. Yet, it is sufficiently rich to capture some interesting phenomena and to provide the reader with some fresh intuitions. Three are the key results. First result: the incentives that govern both decision-making and strategic

interaction co-evolve with the epidemic in a very specific way. When disease-prevalence is low in the active population, the risk of infection remains negligible even if social-distancing norms are completely ignored by citizens. When disease-prevalence is high(er), the increased risk of infection serves as a discipline device for older/weaker citizens, that observe social-distancing norms while younger/healthier ones slack. In the first case, compliance is a dominated strategy at the individual level, social distancing is impossible to implement, and the epidemic evolves according to standard SIR(D) dynamics. In the second case, individual decisions to comply are *strategic substitutes*: high compliance rates induce a low risk of infection that, rationally anticipated by the citizens, reduces their incentive to comply in the first place. Opportunistic behaviour is observed in equilibrium, where young/healthy citizens free-ride on the (costly) social distancing implemented by older/weaker ones. The general result is not novel: it is in line with [Engle et al., 2021] and [Toxvaerd, 2020], albeit the details differ significantly. Both works, for instance, use continuous-time differential games to study endogenous social distancing, and consistently appeal to modelling assumptions that we are able, at least in part, to relax in our discrete-time setup[24]. The result confirms, in any case, that the threshold form of agents' behavioural responses to aggregates is robust to alternative model specifications, thus warranting once more their use in reduced-form within ABM models such as [Bisin and Moro, 2022a, Bisin and Moro, 2022b]. Second result: agents' beliefs about their susceptibility to the infection appear to be *crucial* in the determination of strategic incentives. If agents ignore their states, and use observable aggregates to form (common) beliefs about those states, individual decisions to comply with social-distancing norms cannot be but strategic substitutes. If, conversely, they come to their states, individual actions can be substitutes *or* complements, depending on the intensity of the global payoff externality. The result is somehow counter-intuitive, but boils down to a sensible and relatively simple intuition: if agents know their states, their actions become state-contingent. If this is the case, asymptomatic and infected agents have no incentive to implement social distancing, and susceptible agents only are left playing the social interaction game. Being rational, they realize that, in such an environment, collective decisions affect *both* the mass of social interactions (via the congestion externality) *and* the group-specific prevalence of the disease: the higher the aggregate level of compliance, the larger the relative mass of asymptomatic infected in the interacting population, the higher the risk faced by susceptible

[24] E.g. the representative-agent framework of [Engle et al., 2021] – see footnote 14, page 7.

agents that do not comply. Conversely, the lower the aggregate level of compliance, the smaller the relative mass of asymptomatic agents in the interacting population, the lower the risk faced by non-compliant susceptibles. When this intensive -margin effect offsets the extensive-margin of the congestion externality, strategic complementarity dominates and a multiplier effects arises. It is worth noting that, in the complementarity scenario, group-specific prevalence is *endogenous* and departs from that measured population-wide: there share of infected is higher in the sub-population of interacting agents than in the population as a whole. To the best of our knowledge, the result is entirely novel. Third result: when endogenous compliance is taken into account, lockdown policies are typically less effective at containing the epidemics that standard SIR models predict.

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Appendix A.

PROOF OF PROPOSITION 2

The proof of Proposition 2 is relatively simple but lengthy. To ease the exposition and highlight the most important intermediate results, the proof is subdivided into several lemmas. All formal arguments are commented and discussed, and complemented by qualitative ones and by several graphs.

A.1 Preliminary Remarks: The (Expected) Utility from Non-Compliance

Recall from Subsection 3 in the main text that, when citizens are assumed to ignore their epidemiological states $e(i)$, the action-contingent (expected) utility $u_i(\cdot)$ of a citizen with demographic profile $x(i)$ is

$$u_i\Big(a(i), A; \mathbf{\Omega}, \mathbf{E}\Big) = \begin{cases} -c & \text{if } a(i) = 1 \\ -\tilde{D}_i\left(\dfrac{S}{ASY + S + pR}\right)\beta\, G(A) & \text{if } a(i) = 0 \end{cases} \tag{A.1}$$

where: (i) the vector $\mathbf{\Omega} \in \mathcal{O}$ summarises the parameterisation of the model – see footnote 3 in the main text; (ii) the vector $\mathbf{E} \in \mathcal{E}$ stands for a generic tuple of the epidemiological aggregates $\langle ASY, S, R \rangle$ in the space $\mathcal{E} \subset [0,1]^3$ of all possible configurations that are *consistent*, i.e.

$$\mathcal{E} = \Big\{ \mathbf{E} \in [0,1]^3 \ : \ \left(p^{-1}ASY + S + R\right) \leq (1 - D)\,, \forall\, D \in [0,1] \Big\}\,;$$

(iii) $c > 0$ is the implicit cost of compliance defined by (13) – and included in $\mathbf{\Omega}$; (iv) $A \in [0,1]$ is the aggregate/average compliance rate with social-distancing norms defined by (12) and (24); (v) $\beta\, G(A)$ is the probability (risk) of contagion faced by a susceptible citizens when he/she ignores social distancing and interacts with other non-compliant individuals – it is defined as

$$G(A) \;=\; \pi\,(A)\,\varphi\,(A) \tag{A.2}$$

with $\varphi\,(A)$ and $\pi\,(A)$ the extensive- and intensive-margin components, respectively, defined as

$$\varphi\,(A) \;=\; \Big[\,(1-LA)\,\big(ASY+S+pR\big)+(1-p)\,R\,\Big]^{\phi} \tag{A.3a}$$

$$\pi\,(A) \;=\; \frac{ASY\,(1-LA)}{(1-LA)\,\big(ASY+S+pR\big)+(1-p)\,R} \tag{A.3b}$$

with $\phi \geq 0$ – see equations (18) and (17) in the main text; (vi) $\tilde{D}\,(x_i)$ is the discounted (expected) cost of death after infection, defined as

$$\tilde{D}_i \;=\; \lambda^2 D\left[\frac{\Phi\,(\theta+\alpha x(i))}{1-\lambda\,(1-\gamma)\,\Big(1-\Phi\,(\theta+\alpha x(i))\Big)}\right] \quad , \tag{A.4}$$

where $\Phi\,(\,\cdot\,)$ indicates the Normal Standard CDF, $D \gg 0$ is the (common) cost of death, $\lambda \in (0,1)$ is the (time) discount factor, and $\gamma \in (0,1)$ parameterises the probability of recovery after infection – see Appendix B.1 for its derivation.

Note that the expected utility of non compliance of the generic i-th citizen depends on his/her demographic profile only via the (expected) discounted cost of death D_i. As a consequence, it is strictly decreasing in $x(i)$. The following Lemma states and proves the result.

LEMMA 1. *The expected utility of non-compliance is strictly decreasing in $x(i)$, i.e.*

$$\frac{\partial}{\partial x(i)}\,u_i\,(a(i)=0,A;\mathbf{\Omega},\mathbf{E}) \;<\; 0 \tag{A.5}$$

for every model parameterisation $\mathbf{\Omega} \in \mathcal{O}$ and every configuration of the aggregate $\mathbf{E} \in \mathbf{E}$.

Proof. The above derivative (A.5) can be written as

$$\frac{\partial}{\partial x(i)}\, u_i\left(a(i)=0, A; \boldsymbol{\Omega}, \mathbf{E}\right) \;=\; -\left(\frac{\partial}{\partial x(i)}\, \tilde{D}_i\right) \beta\, G\left(A\right) \;. \tag{A.6}$$

Differentiating $\tilde{D}_i$ as defined by (A.4) w.r.t. $x(i)$ we obtain that

$$\frac{\partial}{\partial x(i)}\, \tilde{D}_i \;=\; \alpha\, \phi\left(\theta + \alpha\, x(i)\right) \left[\frac{1-\lambda\left(1-\gamma\right)}{\Big[1-\lambda\left(1-\gamma\right)\big(1-\Phi\left(\theta+\alpha\, x(i)\right)\big)\Big]^2}\right] \;>\; 0 \tag{A.7}$$

and, since $\beta G\left(A\right) \geq 0$, the derivative (A.6) must be strictly negative. □

Notice further that, for every demographic profile $x(i) \in \mathbb{R}$, the expected utility of non-compliance defined in (A.1) is strictly in the aggregate/average compliance rate $A \in [0,1]$. Graphically, as A increases from zero to one, the entire mapping of $u_i\left(a(i)=0, A;\, \cdot\,\right)$ shifts *upwards* towards zero and becomes *flatter* – see Figure A.1. The result is formally stated and proved in Lemma 2 here below.

LEMMA 2. *For every pair $A', A'' \in [0,1]$ of arbitrary values of the aggregate/average compliance such that $A'' > A'$, the relation*

$$u_i\left(a(i)=0, A=A''; \boldsymbol{\Omega}, \mathbf{E}\right) \;\geq\; u_i\left(a(i)=0, A=A'; \boldsymbol{\Omega}, \mathbf{E}\right) \tag{A.8}$$

holds for any parameterisation $\boldsymbol{\Omega} \in \mathcal{O}$ and every consistent configuration $\mathbf{E} \in \mathcal{E}$ of the aggregates.

Proof. Consider the expected utility of non-compliance for the generic i-th citizen with demographic profile $x(i) \in \mathbb{R}$ – see the definition in (A.1). Differentiating it w.r.t. the aggregate/average compliance rate A it is immediate to check that

$$\frac{\partial}{\partial A}\, u_i\left(a(i)=0, A; \boldsymbol{\Omega}, \mathbf{E}\right) \;=\; \frac{\beta\, \tilde{D}_i\left(S\right)\left(ASY\right)\Big[\left(1-p\right)R + \phi\left(1-LA\right)\left(ASY+S+pR\right)\Big]}{\left(ASY+S+pR\right)\Big[\left(1-L\right)\left(ASY+S+pR\right)+\left(1-p\right)R\Big]^{2-\phi}} \tag{A.9}$$

with $\tilde{D}_i \geq 0$ the (expected) discounted cost of death post-infection defined by (A.4). It is immediate to check that (A.9) is strictly positive for every $A \in [0,1]$ and every $\phi \geq 0$. □

Notice further that the utility of non-compliance defined in (A.1) is *bounded* in $x(i)$. In particular,

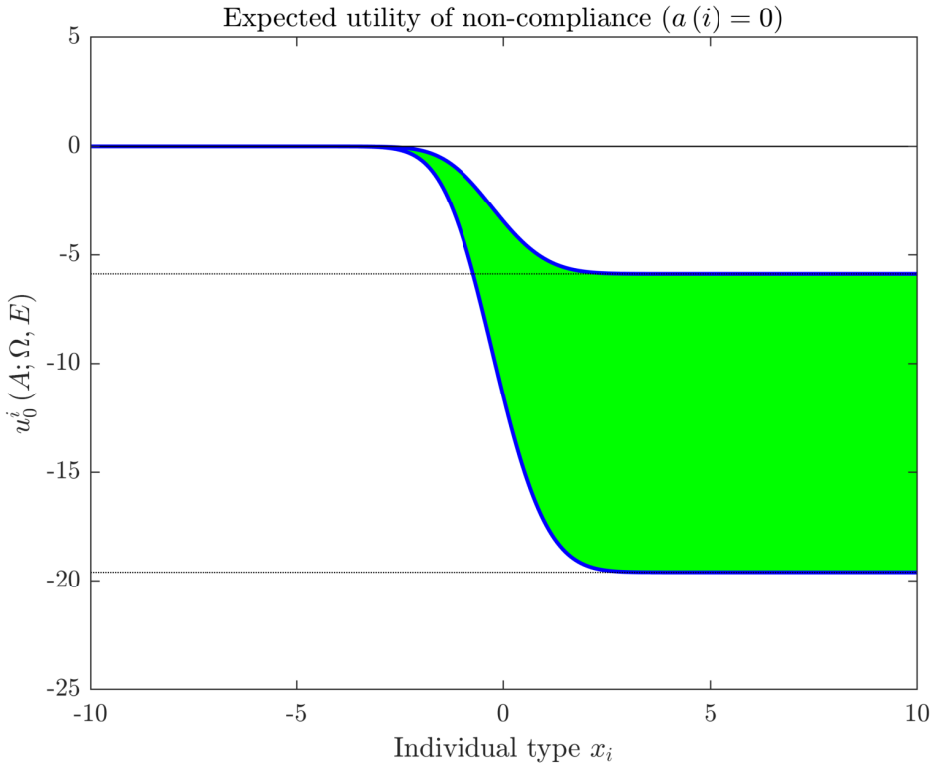

Figure A.1. Expected utility of non-compliance as a function of the individual resilience to the disease x_i. Calibration: $\theta = 1$, $\alpha = 1$, $\bar{x} = 0$, $\gamma = 0.7$, $D = 150$, $\lambda = 0.99$, $L = 0.7$, $\phi = 1$.

it holds that

$$\lim_{x \longrightarrow -\infty} u_i\,(a(i)=0, A;\,\cdot\,) \;=\; u_0^{-\infty}\,(A;\,\cdot\,) \;=\; 0 \tag{A.10a}$$

$$\lim_{x \longrightarrow +\infty} u_i\,(a(i)=0, A;\,\cdot\,) \;=\; u_0^{+\infty}\,(A;\,\cdot\,) \;=\; -\lambda^2 D \left(\frac{S}{ASY + S + pR}\right) \beta\, G(A) \tag{A.10b}$$

for it is immediate to check from (A.4) that

$$\lim_{x \longrightarrow -\infty} \tilde{D}_i \;=\; 0$$

$$\lim_{x \longrightarrow +\infty} \tilde{D}_i \;=\; \lambda^2 D$$

holds. Note that the asymptote $u_0^{+\infty}\,(A;\,\cdot\,)$ defined by (A.10b) is a function of A. Via Lemma 2 it must therefore hold that

$$u_0^{+\infty}\,(A;\,\cdot\,) \;\in\; \left[\,u^{+\infty}\,(A{=}0;\,\cdot\,)\,,\, u_0^{+\infty}\,(A{=}1;\,\cdot\,)\,\right] \tag{A.11}$$

for every $A \in (0,1)$, where the extrema

$$u_0^{+\infty}\left(A{=}0;\,\cdot\,\right) = -\lambda^2 D\,\beta\left[\frac{(S)(ASY)}{ASY+S+pR}\right]\Big(ASY+S+R\Big)^{\phi-1} \tag{A.12a}$$

$$u_0^{+\infty}\left(A{=}1;\,\cdot\,\right) = -\lambda^2 D\,\beta\left[\frac{(S)(ASY)(1-L)}{ASY+S+pR}\right]\Big[(1-L)\left(ASY+S+pR\right)+(1-p)\,R\Big]^{\phi-1} \tag{A.12b}$$

are easily obtained by substituting the definition (A.2)-(A.3) of $G(A)$ into (A.10b). Figure A.1 provides a graphical representation of the (expected) utility of non-compliance plotted against the domain of $\mathcal{X} = \mathbb{R}$ of the demographic profiles x of the citizenry. The shaded area – in green – between the bounds $u_i\left(a(i){=}0; A{=}0;\,\cdot\,\right)$ and $u_i\left(a(i){=}0; A{=}1;\,\cdot\,\right)$ – the solid lines in blue – is the "oscillation band" in which the utility takes on values for every possible $A \in [0,1]$. Via Lemma 2, every value of A unambiguously identifies one specific curve in the band.

A.2 Identification of the Strategic Scenarios

As the epidemic evolves, the environment in which the agents interact may change dramatically, and the same holds true for set of incentives that govern both rational behaviour and strategic interactions. We call a 'scenario' an environment in which the co-evolution between the epidemiological aggregates and the behaviour of the agents is pinned down by a well-defined set of incentives – significantly different and clearly distinguishable from those of other scenarios. In other words, every scenario is a "small world" governed by specific and well-defined strategic incentives, and every change of scenario amounts to a 'phase transition'. We identify three different scenario: in the first, non-compliance is a dominant strategy for the risk of contagion in social interaction is too low; in the second, the symmetric equilibrium where all citizens do not comply is still rationalisable, but compliance is no longer dominated a strategy; in the third, compliance is always dominant for *some* citizens and the symmetric equilibrium at full non-compliance is no longer rationalisable. Every scenario corresponds to a different equilibrium structure – and to different best-response dynamics –, but equilibria are *unique* within scenarios. Every scenario occurs more or less frequently depending the scaling coefficient $\phi \geq 0$. To proceed with the characterisation, it is therefore convenient to distinguish between three subcases: (i) $\phi > 1$, the simplest; (ii) $\phi = 1$; (iii) $\phi \in [0,1)$, the most difficult.

A.2.1 Characterisation for $\phi > 1$

We begin with the simplest case. The following Lemma shows that, when $\phi > 1$, for every parameterisation $\boldsymbol{\Omega} \in \mathcal{O}$ and every arbitrary but configuration $\mathbf{E} \in \mathcal{E}$ of the aggregates[A2] every scenarios is unambiguously identified by the aggregate mass of asymptomatic infected ASY population-wide.

LEMMA 3. *Let $\phi > 1$. Then, for every model parameterisation $\boldsymbol{\Omega} \in \mathcal{O}$ with $\phi > 1$ and every configuration $\mathbf{E} \in \mathcal{E}$ of the epidemiological aggregates, there exists a unique pair of non-negative values $\left\langle \underline{ASY}_\phi(\boldsymbol{\Omega}, \mathbf{E}), \overline{ASY}_\phi(\boldsymbol{\Omega}, \mathbf{E}) \right\rangle$ of the aggregate mass ASY of asymptomatic infected agents, with $\overline{ASY}_\phi(\boldsymbol{\Omega}, \mathbf{E}) > \underline{ASY}_\phi(\boldsymbol{\Omega}, \mathbf{E})$, such that*

$$-c \leq u_0^{+\infty}(A{=}0; \boldsymbol{\Omega}, \mathbf{E}) \qquad \text{if } ASY \leq \underline{ASY}_\phi(\boldsymbol{\Omega}, \mathbf{E}) \quad \text{(A.13a)}$$

$$-c \in \left(u_0^{+\infty}(A{=}0; \boldsymbol{\Omega}, \mathbf{E}), u_0^{+\infty}(A{=}1; \boldsymbol{\Omega}, \mathbf{E})\right] \quad \text{if } ASY \in \left(\underline{ASY}_\phi(\boldsymbol{\Omega}, \mathbf{E}), \overline{ASY}_\phi(\boldsymbol{\Omega}, \mathbf{E})\right] \quad \text{(A.13b)}$$

$$-c > u_0^{+\infty}(A{=}1; \boldsymbol{\Omega}, \mathbf{E}) \qquad \text{if } ASY > \overline{ASY}_\phi(\boldsymbol{\Omega}, \mathbf{E}) \quad \text{(A.13c)}$$

hold, where $c > 0$ is the (implicit) cost of compliance, and with $u_0^{+\infty}(A; \boldsymbol{\Omega}, \mathbf{E})$ the (expected) utility from non-compliance of the least resilient agent – i.e. for $x \to +\infty$ – when the median/aggregate compliance rate is $A \in [0, 1]$. The critical values $\underline{ASY}_\phi(\boldsymbol{\Omega}, \mathbf{E})$ and $\overline{ASY}_\phi(\boldsymbol{\Omega}, \mathbf{E})$ are unambiguously identified by the conditions

$$-\left[\frac{(S)\left(\underline{ASY}_\phi(\boldsymbol{\Omega}, \mathbf{E})\right)}{\underline{ASY}_\phi(\boldsymbol{\Omega}, \mathbf{E}) + S + pR}\right]\left(\underline{ASY}_\phi(\boldsymbol{\Omega}, \mathbf{E}) + S + R\right)^{\phi-1} = -\frac{c}{\lambda^2 D\,\beta} \quad \text{(A.14a)}$$

$$-\left[\frac{(S)(1-L)\left(\overline{ASY}_\phi(\boldsymbol{\Omega}, \mathbf{E})\right)}{\overline{ASY}_\phi(\boldsymbol{\Omega}, \mathbf{E}) + S + pR}\right]\left[(1-L)\left(\overline{ASY}_\phi(\boldsymbol{\Omega}, \mathbf{E}) + S + pR\right) + (1-p)R\right]^{\phi-1} = -\frac{c}{\lambda^2 D\,\beta} \quad \text{(A.14b)}$$

respectively.

Proof. We begin by proving the existence and uniqueness of the lower critical value $\underline{ASY}_\phi(\boldsymbol{\Omega}, \mathbf{E})$ identified by expression (A.14a). Using the definition (A.12a) of the lower asymptote $u_0^{+\infty}(A{=}0; \boldsymbol{\Omega}, \mathbf{E})$

[A2] See Subsection A.1 for a formal definition of what a consistent configuration is.

of the expected utility of non-compliance, we can rewrite condition (A.13a) in explicit form as

$$-c \leq -\lambda^2 D\,\beta \left[\frac{(S)(ASY)}{ASY+S+pR}\right]\Big(ASY+S+R\Big)^{\phi-1} \tag{A.15}$$

that can easily be rewritten as

$$-\frac{c}{\lambda^2 D\,\beta} \leq \underbrace{-\left[\frac{(S)\,(ASY)}{ASY+S+pR}\right]\Big(ASY+S+R\Big)^{\phi-1}}_{\underline{g}\,(ASY)} \quad . \tag{A.16}$$

Differentiating $\underline{g}\,(ASY)$ – i.e the RHS of (A.16) – w.r.t. ASY, simplifying off and rearranging, we obtain

$$\frac{\partial \underline{g}\,(ASY)}{\partial ASY} = -\frac{(S)\left[\phi\,(ASY)\,(S+pR\,)+(ASY)^2\,(\phi-1)+(S+R\,)\,(S+pR\,)\,\right]}{(ASY+S+R\,)^2\,(ASY+S+pR\,)^{2-\phi}} \tag{A.17}$$

that is (strictly) negative if

$$\phi\,(ASY)\,(S+pR\,)+(ASY)^2\,(\phi-1)+(S+R\,)\,(S+pR\,) > 0 \tag{A.18}$$

holds. An inspection of (A.18) immediately reveals that the condition is always met if $\phi \geq 1$ – for the LHS would be unambiguously negative in that case. Hence

$$\phi \geq 1 \qquad \Longrightarrow \qquad \frac{\partial}{\partial ASY}\,\underline{g}\,(ASY) < 0 \tag{A.19}$$

holds. Note that the LHS of (A.16) is strictly negative, while for the RHS $\underline{g}\,(ASY=0)=0$ holds. Since $\underline{g}$ strictly decreases in ASY if $\phi \geq 1$. there must be a unique intersection $ASY = \underline{ASY}_{\phi}\,(\mathbf{\Omega},\mathbf{E})$ such that

$$\frac{c}{\lambda^2 D\,\beta} = \left[\frac{(S)\Big(\underline{ASY}_{\phi}\,(\mathbf{\Omega},\mathbf{E})\Big)}{\underline{ASY}_{\phi}\,(\mathbf{\Omega},\mathbf{E})+S+pR}\right]\Big(\underline{ASY}_{\phi}\,(\mathbf{\Omega},\mathbf{E})+S+R\Big)^{\phi-1}\,,$$

and such that, via (A.19),

$$-c \leq u_0^{+\infty}\,(A=0;\mathbf{\Omega},\mathbf{E}) \qquad \text{if } ASY \leq \underline{ASY}_{\phi}\,(\mathbf{\Omega},\mathbf{E})$$

$$-c > u_0^{+\infty}\,(A=0;\mathbf{\Omega},\mathbf{E}) \qquad \text{otherwise}$$

must hold. We now proceed by proving the existence and uniqueness of the upper critical value $\overline{ASY}_{\phi}(\mathbf{\Omega}, \mathbf{E})$ identified by expression (A.13c). The procedure is the same used for $\underline{ASY}_{\phi}(\mathbf{\Omega}, \mathbf{E})$: using the definition (A.12b) of the upper asymptote $u_0^{+\infty}(A=1; \mathbf{\Omega}, \mathbf{E})$ of the expected utility of non-compliance, we can rewrite condition (A.13c) in explicit form as

$$-\frac{c}{\lambda^2 D\,\beta} > \underbrace{-\left[\frac{(S)(ASY)(1-L)}{ASY+S+pR}\right]\Big[(1-L)\left(ASY+S+pR\right)+(1-p)\,R\Big]^{\phi-1}}_{\bar{g}(ASY)}. \tag{A.20}$$

Differentiating the function $\bar{g}(ASY)$ – i.e. the RHS of (A.20) – w.r.t. ASY we obtain

$$\frac{\partial \bar{g}(ASY)}{\partial ASY} = -\frac{(S)(1-L)\Big[\mathcal{B}(ASY)\Big]}{(ASY+S+pR)^2\Big[(1-L)(ASY+S+pR)+(1-p)\,R\Big]^{2-\phi}} \tag{A.21}$$

where the polynomial $\mathcal{B}(ASY)$ is

$$\begin{aligned}\mathcal{B}(ASY) = {} & (1-p)\,R\,(S+pR) + \\ & + (1-L)\left[(ASY)^2(\phi-1)+(S+pR)\Big(\phi(ASY)+(S+pR)\Big)\right]. \end{aligned} \tag{A.22}$$

A closer inspection of (A.21) immediately reveals that its sign is entirely determined by the sign of $\mathcal{B}(ASY)$, in particular

$$\frac{\partial \bar{g}(ASY)}{\partial ASY} < 0 \qquad \Longrightarrow \qquad \mathcal{B}(ASY) > 0 \tag{A.23}$$

holds, and note from (A.22) that $\mathcal{B}(ASY) > 0$ if $\phi \geq 0$, so that

$$\phi \geq 1 \qquad \Longrightarrow \qquad \frac{\partial}{\partial ASY}\,\bar{g}(ASY) < 0 \tag{A.24}$$

holds. Note again that the LHS of condition (A.20) is strictly negative, while the RHS is strictly decreasing in ASY via (A.24). Thus, there must exist a unique intersection $ASY = \overline{ASY}_{\phi}(\mathbf{\Omega}, \mathbf{E})$ such that

$$\frac{c}{\lambda^2 D\,\beta} = \left[\frac{(S)\Big(\overline{ASY}_{\phi}(\mathbf{\Omega}, \mathbf{E})\Big)(1-L)}{\overline{ASY}_{\phi}(\mathbf{\Omega}, \mathbf{E})+S+pR}\right]\Big[(1-L)\left(\overline{ASY}_{\phi}(\mathbf{\Omega}, \mathbf{E})+S+pR\right)+(1-p)\,R\Big]^{\phi-1},$$

and such that, again via (A.24),

$$-c > u_0^{+\infty}\left(A{=}1; \boldsymbol{\Omega}, \mathbf{E}\right) \qquad \text{if } ASY > \overline{ASY}_{\phi}\left(\boldsymbol{\Omega}, \mathbf{E}\right)$$

$$-c \leq u_0^{+\infty}\left(A{=}1; \boldsymbol{\Omega}, \mathbf{E}\right) \qquad \text{otherwise}$$

must hold. □

A brief remark may be useful here. Recall the graphical interpretation of the behaviour of the expected utility of non-compliance $u_i\left(a(i){=}0, A; \cdot\right)$ outlined in Subsection A.1 above: for every value $A \in [0,1]$ the entire mapping moves upwards (as A increases) or downwards (as A decrease) within an "oscillation band" identified by the bounds $u_i\left(a(i){=}0; A{=}0; \cdot\right)$ – below – and $u_i\left(a(i){=}0; A{=}1; \cdot\right)$ – above. What Lemma 2 proves is that, when $\phi > 1$, the oscillation band varies with ASY in a very regular fashion: as ASY increases, the oscillation band moves downwards. The formal proof outlined above, indeed, essentially proves that the asymptotes of the bounds as $x \to +\infty$, i.e. $u_0^{+\infty}\left(A{=}1; \cdot\right)$ $u_0^{+\infty}\left(A{=}1; \cdot\right)$, are strictly decreasing in ASY when $\phi > 1$.

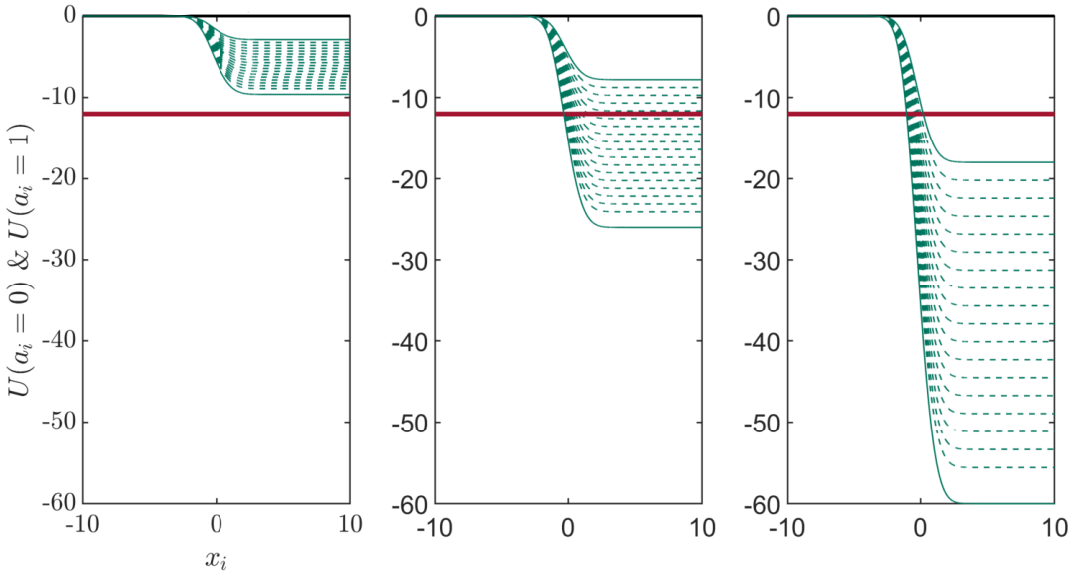

Figure A.2. The three strategic regimes when $\phi > 1$.

A.2.2 Characterisation for $\phi = 1$

TO BE WRITTEN

A.2.3 Characterisation for $\phi \in [0, 1]$

TO BE WRITTEN

A.3 Equilibrium Characterisation Per-Scenario

A.3.1 EC Scenario I

In the first scenario, the aggregate mass ASY of asymptomatic infected – the citizens that spread the contagion via social interactions with non-infected – is too low to induce compliance. As a consequence, $a(i) = 0$ is a (strictly) dominant strategy, the game is dominance-solvable and has a unique (symmetric) Nash equilibrium such that $A^* = 0$.

LEMMA 4. *Let* $ASY < \underline{ASY}_{\phi}(\mathbf{\Omega}, \mathbf{E})$ *hold. Then, the compliance game has a unique equilibrium in which all citizens choose not to comply, i.e.*

$$a^*(i) = 0$$

for all $i \in [0, 1]$. *The equilibrium median/aggregate compliance rate is therefore* $A^* = 0$.

Proof. Recall from Lemma 3 that, in the first scenario, the condition (A.13a)

$$-c \leq u_0^{+\infty}(A{=}0; \mathbf{\Omega}, \mathbf{E})$$

holds if $ASY \leq \underline{ASY}_{\phi}(\mathbf{\Omega}, \mathbf{E})$. Since $-c$ is the net utility of compliance $u(a(i){=}0, A; \mathbf{\Omega}, \mathbf{E})$, we can rewrite the above condition as

$$u(a(i){=}0, A; \mathbf{\Omega}, \mathbf{E}) \leq u_0^{+\infty}(A{=}0; \mathbf{\Omega}, \mathbf{E}) \ . \tag{A.25}$$

In words, expression (A.25) states that, if $ASY < \underline{ASY}_{\phi}(\mathbf{\Omega}, \mathbf{E})$, then the implicit cost of compliance exceeds the expected cost of non-compliance (i) for the least resilient citizen(s) with $x(i)$

arbitrarily large, (ii) under the worst possible circumstance in which the risk of contagion in case of non-compliance is maximal ($A = 0$). As a consequence, no citizen has any incentive to comply, irrespective of his/her demographic profile $x(i)$ and/or of other citizens' (anticipated) aggregate/average compliance rate A. The game is therefore dominance-solvable in this scenario, and the unique Nash equilibrium is such that all citizens ignore social-distancing norms – $a^*(i) = 0$ for all $i \in [0, 1]$, hence $A^* = 0$. □

A.3.2 EC in Scenario II

In the second scenario, the mass ASY of asymptomatic infected citizens is large enough to induce compliance with social-distancing norms *potentially*. However, it is not sufficiently large to ensure that compliance be rationalisable by the citizenry for *every* expected compliance rate $A \in [0, 1]$. The equilibrium characterisation for this scenario is structures as follows: Lemma 5 shows that, if the aggregate compliance rate A exceeds a critical value $\hat{A} \in (0, 1)$, then non-compliance becomes dominant. Lemma 6 proves that, for every $A \leq \hat{A}$ the best-response function is downward-sloping in A – thus confirming that the result of Proposition 2 that characterises the individual decisions to comply with norms as *strategic substitutes*; Lemma 7 completes the characterization by showing that the downward-sloping best-response function has a fixed point in the range $[0, \hat{A}]$ of its domain. Finally, Lemma 8 highlights a a robustness results, by showing that if the average mortality population-wide $\bar{Q}^D$ is sufficiently low, then the monotone equilibrium characterised by Lemmas 5 to 7 is also stable (in the sense of best-response dynamics), and is therefore the unique equilibrium that survives the iterated elimination of (strongly) dominated strategies.

LEMMA 5. *Let* $ASY \in \left(\underline{ASY}_{\phi}(\mathbf{\Omega}, \mathbf{E}), \overline{ASY}_{\phi}(\mathbf{\Omega}, \mathbf{E}) \right]$ *hold. Then, there exists a unique critical value* $\hat{A} \in (0, 1]$ *of the aggregate/average compliance rate* A*, unambiguously identified by the condition*

$$\frac{c\,(ASY + S + pR)}{\lambda^2 D\,\beta\,(S)\,(ASY)} \;=\; (1 - LA)\left[\,(1 - LA)\,(ASY + S + pR) + (1 - p)\,R\,\right]^{\phi - 1}, \tag{A.26}$$

such that (i) compliance with norms is rationalisable if $A \leq \hat{A}$*, while (ii) it is – strongly – dominated otherwise.*

Proof. Lemma 2 ensures that $u_i\,(a(i){=}0, A{=}1;\,\cdot\,) > u_i\,(a(i){=}0, A{=}0;\,\cdot\,)$ holds for any $\phi \geq 0$,

since the expected utility of non-compliance is strictly increasing in A for every demographic profile $x(i)$ and every arbitrary $A \in [0,1]$. The asymptote $u^{+\infty}(A;\cdot)$ defined by (A.10), too, must be therefore be strictly increasing in A. Recall further from Lemma 3 that the condition $ASY \in \left(\underline{ASY}_{\phi}(\mathbf{\Omega},\mathbf{E}), \overline{ASY}_{\phi}(\mathbf{\Omega},\mathbf{E})\right]$ ensures by construction that

$$u_0^{+\infty}(A{=}0;\mathbf{\Omega},\mathbf{E}) \;<\; -c \;\leq\; u_0^{+\infty}(A{=}1;\mathbf{\Omega},\mathbf{E}) \tag{A.27}$$

holds. As a consequence, there must exists a unique value $A = \hat{A} \in (0,1]$ such that

$$-c \;=\; u_0^{+\infty}\left(A{=}\hat{A};\mathbf{\Omega},\mathbf{E}\right) \tag{A.28}$$

holds. Substituting for the definition (A.10b) of the generic $u_0^{+\infty}(A{=}A;\cdot)$ into (A.28), we obtain

$$\frac{c\,(ASY+S+pR)}{\lambda^2 D\,\beta\,(S)\,(ASY)} \;=\; \underbrace{(1-LA)\Big[\,(1-LA)\,(ASY+S+pR)+(1-p)\,R\,\Big]^{\phi-1}}_{f(A)}\,, \tag{A.29}$$

that is condition (A.26) in Lemma 5. Differentiating the RHS of (A.29) – i.e. the function $f(A)$ – w.r.t. A we obtain

$$\frac{\partial}{\partial A}\,f(A) \;=\; -\frac{(1-p)\,R+\phi\,(1-LA)\,(ASY+S+pR)}{L\Big[\,(1-LA)\,(ASY+S+pR)+(1-p)\,R\,\Big]^{2-\phi}} \;<\; 0\;. \tag{A.30}$$

Since the LHS of (A.29) is a negative constant, and the RHS is strictly decreasing in A, there must exist a unique intersection. It is immediate to check that condition (A.27) ensures that such unique intersection must occur in the admissible range $[0,1]$. Hence condition (A.29) unambiguously identifies the critical value $\hat{A} \in (0,1]$. Note that

$$A \;>\; \hat{A} \qquad \Longrightarrow \qquad -c \;<\; u_0^{+\infty}(A;\mathbf{\Omega},\mathbf{E})$$

hold by construction. In words: if $A > \hat{A}$, compliance is (strongly) dominated by non-compliance even for the least resilient citizen with $x(i) \longrightarrow +\infty$. As a consequence, if $A > \hat{A}$ compliance is not rationalisable by any citizen, irrespective of his/her demographic profile. □

Indicate with $A^*(A) : [0,1] \longmapsto [0,1]$ the best-response function that maps any arbitrary value of the aggregate/average compliance rate into A into the compliance rate derived by aggregating the optimal actions of citizens that are consistent with A – see equation (24) in the main text. Lemma 5 essentially states that

$$A^*(A) \,=\, 0\,, \qquad \text{for all } A \in [\hat{A}, 1]\,. \tag{A.31}$$

It remains to be determined what is the best-response $A^*(A)$ when $A \in [\,0, \hat{A}\,)$, i.e. when compliance is rationalisable for *some* citizens. The following Lemma provides such result.

LEMMA 6. *Let* $ASY \in \left(\underline{ASY}_{\phi}(\mathbf{\Omega}, \mathbf{E}), \overline{ASY}_{\phi}(\mathbf{\Omega}, \mathbf{E})\right]$ *hold. Then, the best-response function* $A^*(A)$ *is downward-sloping in* A *for any* $A \leq \hat{A}$ *– with* $\hat{A} \in (0,1)$ *the critical value identified in Lemma 5.*

Proof. We construct the best-response $A^*(A)$ for the case $A \in [\,0, \hat{A}\,)$ by deriving citizens' optimal compliance rule for an *arbitrary* value $A = \tilde{A}$ of the compliance rate. If $A = \tilde{A}$ holds, then it is rational for a citizen to comply if

$$-c \,\geq\, \lambda^2 D \left[\frac{\Phi\left(\theta + \alpha\, x(i)\right)}{1 - \lambda\,(1-\gamma)\left(1 - \Phi\left(\theta + \alpha\, x(i)\right)\right)}\right] \left(\frac{S}{ASY + S + pR}\right) \beta\, G(\tilde{A})\,. \tag{A.32}$$

The marginal type $x^*(\tilde{A})$ is the demographic profile $x \in \mathbb{R}$ that such that the generic i-th citizen with $x(i) = x^*(\tilde{A})$ is indifferent between compliance and non-compliance when the aggregate/average compliance rate is $A = \tilde{A}$. Since the expected utility of non-compliance strictly decreases in $x(i)$ via Lemma 1, and strictly increases in A via Lemma 2, the marginal type must be *unique* for every arbitrary $A \in [0,1]$. Solving (A.32) in $x(i)$ we obtain

$$x^*(\tilde{A}) \,=\, \frac{1}{\alpha}\left[\,\Phi^{-1}\!\left(\frac{c}{\lambda}\,\mathcal{Z}\big(\tilde{A}; \mathbf{\Omega}, \mathbf{E}\big)\right) - \theta\,\right], \tag{A.33}$$

where the polynomial $\mathcal{Z}\big(\tilde{A};\mathbf{\Omega},\mathbf{E}\big)$ is defined as

$$\mathcal{Z}\big(\tilde{A};\mathbf{\Omega},\mathbf{E}\big) \;=\; \big(\,1-\lambda\,(1-\gamma)\,\big)\Big(ASY+S+pR\Big)\;\times$$

$$\times\left\{\lambda\beta D(S)(ASY)\big(1-L\tilde{A}\big)\Big[(1-L\tilde{A})\,(ASY+S+pR)+(1-p)R\Big]^{\phi-1}-c(1-\gamma)\big(ASY+S+pR\big)\right\}^{-1} \tag{A.34}$$

where the condition $A\in[\,0,\hat{A}\,)$ ensures that $\mathcal{Z}\,(c/\lambda)\in[0,1]$ so that $x^*(\tilde{A})$ is well defined. Since the expected utility of non-compliance strictly decreases in $x(i)$ via Lemma 1, then, for every $A=\tilde{A}<\hat{A}$: (i) compliance is strongly preferred to non-compliance for all citizens with $x(i)>x^*(\tilde{A})$; (ii) non-compliance is strongly preferred to compliance for all citizens with $x(i)<x^*(\tilde{A})$. Via the definition (1) of $x(i)$, LLN reasoning applied to continua of i.i.d. random variables yields

$$A^*\left(x^*(\tilde{A})\right) \;=\; \Pr\Big(\,x(i)\geq x^*(\tilde{A})\,\Big) \qquad \text{almost surely}\;, \tag{A.35}$$

i.e. the mass of citizens with $x(i)\geq x^*(\tilde{A})$ is equal (almost surely) to the probability *ex ante* of the event $\varepsilon(i)\geq x^*(\tilde{A})-\bar{x}$. Since the idiosyncratic shocks $\varepsilon(i)$ are normally distributed with zero mean and variance $\sigma^2>0$, the aggregate/average compliance rate $A^*(\tilde{A})$ induced by the arbitrary rate $A=\tilde{A}$ is

$$A^*(\tilde{A}) \;=\; \Phi\left(\frac{\bar{x}-x^*(A)}{\sigma}\right) \tag{A.36}$$

that is equation (24) in the main text. Differentiating (A.36) w.r.t. $\tilde{A}$ yields

$$\frac{\partial}{\partial\tilde{A}}\,A^*(\tilde{A}) \;=-\frac{1}{\sigma}\left[\phi\left(\frac{\bar{x}-x^*(\tilde{A})}{\sigma}\right)\right]\left(\frac{\partial}{\partial\tilde{A}}\,x^*(\tilde{A})\right) \tag{A.37}$$

where $\phi\,(\,\cdot\,)\geq 0$ indicates the Normal Standard PDF. Differentiating the marginal type $x^*(\tilde{A})$ w.r.t. $\tilde{A}$ we obtain

$$\frac{\partial}{\partial\tilde{A}}\,x^*(\tilde{A}) \;=\; \frac{c}{\alpha\lambda}\left[\phi\left(\Phi^{-1}\left(\frac{c}{\lambda}\,\mathcal{Z}(\tilde{A})\right)\right)\right]^{-1}\left(\frac{\partial}{\partial\tilde{A}}\,\mathcal{Z}(\tilde{A})\right) \tag{A.38}$$

where

$$\frac{\partial}{\partial \tilde{A}} \mathcal{Z}(\tilde{A}) = \frac{\left(\mathcal{Z}(\tilde{A})\right)^2}{\lambda D \beta(S)(ASY)\left(1-\lambda\left(1-\gamma\right)\right)\left(ASY+S+pR\right)} \times$$

$$\times \frac{L}{\left(1-L\tilde{A}\right)^2} \times \frac{(1-p)R+\phi(1-L\tilde{A})\left(ASY+S+pR\right)}{\left[\left(1-L\tilde{A}\right)\left(ASY+S+pR\right)+\left(1-p\right)R\right]^{2-\phi}} \quad \text{(A.39)}$$

that is strictly positive. Since (A.39) is strictly positive, then (A.38) is positive, too. Hence (A.37) is strictly negative. Overall, therefore, the following

$$\frac{\partial}{\partial \tilde{A}} A^*(\tilde{A}) = \underbrace{-\frac{1}{\sigma}\left[\phi\left(\frac{\bar{x}-x^*(\tilde{A})}{\sigma}\right)\right]}_{<0} \underbrace{\left(\frac{\partial}{\partial \tilde{A}} x^*(\tilde{A})\right)}_{>0} < 0$$

holds, which completes the proof. □

Lemma 6 completed the characterisation of the best-response function $A^*(A)$, that can now be written as

$$A^*(A) = \begin{cases} 0 & \text{if } A \geq \hat{A} \\ A*(x*(A)) \in (0,1) & \text{if } A < \hat{A} \end{cases} \quad \text{(A.40)}$$

with

$$A^*\left(x^*(A)\right) = \Phi\left(\frac{\bar{x}-x^*(A)}{\sigma}\right)$$

strictly decreasing in A, and where $\hat{A}$ is the critical value identified by Lemma 5. The following Lemma completes the characterisation of the unique Nash equilibrium in the second scenario, by showing that a fixed point of $A^*(A)$ exists and it is unique.

LEMMA 7. *Let* $ASY \in \left(\underline{ASY}_\phi(\mathbf{\Omega},\mathbf{E}), \overline{ASY}_\phi(\mathbf{\Omega},\mathbf{E})\right]$ *hold. Then, the social-interaction game has a unique Nash equilibrium in monotone strategies, where citizens comply according to the threshold rule*

$$a^*(i) = 1 \qquad \Longleftrightarrow \qquad x(i) \geq x^*(\mathbf{\Omega},\mathbf{E}) \ , \quad \text{(A.41)}$$

with the equilibrium cut-off $x^*(\mathbf{\Omega}, \mathbf{E})$ *identified by the indifference condition*

$$\lambda^2 D \left[\frac{\Phi\left(\theta + \alpha x^*(\mathbf{\Omega}, \mathbf{E})\right)}{1 - (1-\gamma)\left(1 - \Phi\left(\theta + \alpha x^*(\mathbf{\Omega}, \mathbf{E})\right)\right)} \right] \beta\, G(A^*) \;=\; c\;, \tag{A.42}$$

and where

$$A^* \;=\; A\left(x^*\right) \;=\; \Phi\left(\frac{\bar{x} - x^*(\mathbf{\Omega}, \mathbf{E})}{\sigma}\right) \tag{A.43}$$

holds – with $\bar{x} \in \mathbb{R}$ *the median/average demographical profile population-wide and* $\sigma > 0$ *the cross-sectional variance.*

Proof. Lemma 6 proved that the best-response function (A.43) strictly decreases in A when $A \in [\,0, \hat{A}\,]$, and it equal to zero (flat) otherwise. Since $A^*(A)$ is also continue in A over its entire domain, to prove both the existence and the uniqueness of a fixed point it is sufficient to prove that

$$A^*(A = \hat{A}) \;<\; \hat{A}\;. \tag{A.44}$$

The logic is straightforward: no fixed point can be found in the range $A \in [\,\hat{A}, 1\,]$, for the best-response is always zero there. If a fixed point indeed exists, it must be in the range $A \in [\,0, \hat{A}\,)$. Moreover, since $A^*(A)$ strictly decreases in A, if $A^*(\hat{A}) < \hat{A}$ then $A^*(A)$ must cross the 45 degree line – from above – at some point in $[\,0, \hat{A}\,)$. Recall from Lemma 5 that the definition of $\hat{A}$ is implicit, so it is impossible to evaluate $A^*(A = \hat{A})$ directly. However, we know from (A.26) that for $A = \hat{A}$ it must hold that

$$\frac{c\,(ASY + S + pR)}{\lambda^2 D\,\beta\,(S)\,(ASY)} \;=\; \left(1 - L\hat{A}\right)\left[\,\left(1 - L\hat{A}\right)(ASY + S + pR) + (1-p)\,R\,\right]^{\phi - 1}\;. \tag{A.45}$$

Note that the RHS of (A.45) appears at the denominator of equation (A.34), that defines an element of the marginal type $x^*(A)$ – see equation (A.33). Substituting properly into (A.34) we obtain that

$$\mathcal{Z}(A = \hat{A}) \;=\; \frac{\lambda}{c}\;, \tag{A.46}$$

hence

$$\begin{aligned} x^*(A=\hat{A}) \;&=\; \frac{1}{\alpha}\left[\,\Phi^{-1}\!\left(\frac{c}{\lambda}\mathcal{Z}(A=\hat{A})\right)-\theta\,\right] \\ &=\; \frac{1}{\alpha}\left[\,\Phi^{-1}\big(1\big)-\theta\,\right] \end{aligned} \tag{A.47}$$

with $\Phi^{-1}(\,\cdot\,)$ the inverse of the Normal Standard CDF. Since it holds that

$$\lim_{y\longrightarrow 1}\,\Phi^{-1}(y) \;=\; +\infty\;,$$

then we have that

$$\lim_{A\longrightarrow \hat{A}}\,x^*(A) \;=\; +\infty \tag{A.48}$$

and, consequently,

$$\begin{aligned} A^*(A=\hat{A}) \;&=\; \lim_{A\longrightarrow \hat{A}}\,A^*(A) \\ &=\; \lim_{x^*\longrightarrow +\infty}\,A^*(x^*) \\ &=\; 0\;. \end{aligned} \tag{A.49}$$

Since $A^*\left(A=\hat{A}\right)=0$ and $\hat{A}\in(0,1]$, then $A^*(\hat{A})<\hat{A}$. And since $A^*(A)$ strictly decreases in A, then there must exists a unique value $A*(\mathbf{\Omega},\mathbf{E})$ such that

$$A^*\left(A^*(\mathbf{\Omega},\mathbf{E})\right) \;=\; A^*(\mathbf{\Omega},\mathbf{E}) \tag{A.50}$$

holds – with $A^*(\mathbf{\Omega},\mathbf{E})\in(\,0,\hat{A}\,)$. And since $A^*(\,\cdot\,)$ is a monotone function of $x^*(\,\cdot\,)$, there exists a unique marginal type $x^*(\mathbf{\Omega},\mathbf{E})$ such that

$$A \;=\; A^*(\mathbf{\Omega},\mathbf{E}) \;\Longrightarrow\; \lambda^2 D\left[\frac{\Phi\left(\theta+\alpha x^*(\mathbf{\Omega},\mathbf{E})\right)}{1-(1-\gamma)\left(1-\Phi\left(\theta+\alpha x^*(\mathbf{\Omega},\mathbf{E})\right)\right)}\right]\,\beta\,G(A^*(\mathbf{\Omega},\mathbf{E})) \;=\; c\;, \tag{A.51}$$

i.e. such that when $A=A^*(\mathbf{\Omega},\mathbf{E})$ every citizen with $x(i)=x^*(\mathbf{\Omega},\mathbf{E})$ is indeed indifferent between compliance and non-compliance with social-distancing norms. □

LEMMA 8. *Let* $ASY\in\left(\underline{ASY}_{\phi}(\mathbf{\Omega},\mathbf{E}),\overline{ASY}_{\phi}(\mathbf{\Omega},\mathbf{E})\right]$ *hold. Then, if the no-cycle condition*

$$\theta+\alpha\,\bar{x} \;<\; \Xi(\mathbf{\Omega},\mathbf{E}) \tag{A.52}$$

hold, with

$$\Xi\left(\boldsymbol{\Omega},\mathbf{E}\right) \;=\; , \tag{A.53}$$

the fixed poin A^* identified in Lemma 7 is stable – in the sense of best-response dynamics.

A.3.3 EC in Scenario III

In the third (and last) scenario, the mass ASY of asymptomatic infected citizens is large enough to induce compliance with social-distancing for *every* expected compliance rate $A \in [0,1]$. The results outlined in Subsection A.3.2 for the equilibrium characterisation in the second scenario extend easily to this scenario as well, for the best-response $A^*(A)$ is here the same considered in Lemma 6.

LEMMA 9. *Let $ASY > \overline{ASY}_{\phi}\left(\boldsymbol{\Omega},\mathbf{E}\right)$ hold. Then, the social-interaction game has a unique Nash equilibrium in monotone strategies, where citizens comply according to the threshold rule*

$$a^*(i) \;=\; 1 \qquad \Longleftrightarrow \qquad x(i) \;\geq\; x^*\left(\boldsymbol{\Omega},\mathbf{E}\right) \;, \tag{A.54}$$

with the equilibrium cut-off $x^*\left(\boldsymbol{\Omega},\mathbf{E}\right)$ identified by the indifference condition

$$\lambda^2 D\left[\frac{\Phi\left(\theta+\alpha x^*\left(\boldsymbol{\Omega},\mathbf{E}\right)\right)}{1-\left(1-\gamma\right)\Big(1-\Phi\left(\theta+\alpha x^*\left(\boldsymbol{\Omega},\mathbf{E}\right)\right)\Big)}\right]\beta\, G(A^*) \;=\; c\;, \tag{A.55}$$

and where

$$A^* \;=\; A\left(x^*\right) \;=\; \Phi\left(\frac{\bar{x}-x^*\left(\boldsymbol{\Omega},\mathbf{E}\right)}{\sigma}\right) \tag{A.56}$$

holds – with $\bar{x}\in\mathbb{R}$ the median/average demographical profile population-wide and $\sigma>0$ the cross-sectional variance.

Proof. Since $ASY > \overline{ASY}_{\phi}\left(\boldsymbol{\Omega},\mathbf{E}\right)$, the condition

$$-c \;>\; u_0^{+\infty}\left(A{=}1;\boldsymbol{\Omega},\mathbf{E}\right) \tag{A.57}$$

holds. It is immediate to check that the condition ensures that an intersection between the mapping of the expected utility of non-compliance $u\left(a(i){=}0,A;\boldsymbol{\Omega},\mathbf{E}\right)$ and the utility of compliance $u\left(a(i){=}1,A;\boldsymbol{\Omega},\mathbf{E}\right) = -c$ always exists *for every* $A\in[0,1]$. As in the proof of Lemma 6, we can

derive the best-response function $A^*(A)$ by: (i) fixing an arbitrary value $A = \tilde{A}$ for the aggregate compliance rate; (ii) identifying the (unique) marginal type $x^*(\tilde{A})$ consistent with $A = \tilde{A}$; (iii) calculating $A^*(\tilde{A}) = A(x^*(\tilde{A}))$. □

Appendix B.

OTHER PROOFS & DERIVATIONS

B.1 Discounted Cost of Death: Derivation

Recall from Subsection 2.2 in the main text that, once infected at an arbitrary date t, an individual faces an idiosyncratic probability of death q_i^D in every subsequent period – with q_i^D defined by (3). In case of death, he/she experiences an arbitrarily large individual loss $D \gg 0$, while his/her net utility in case survival is normalized to zero without significant loss of generality. Recall further that, in every period after the infection, the individual remains infected (and infective) with probability q_i^I, and recovers from the disease with probability q_i^R – with q_i^R and q_i^I defined by (6) and (7), respectively. Figure B.1 summarises the chain of possible epidemiological patterns after infection occurs, together with the corresponding transition probabilities per-period.

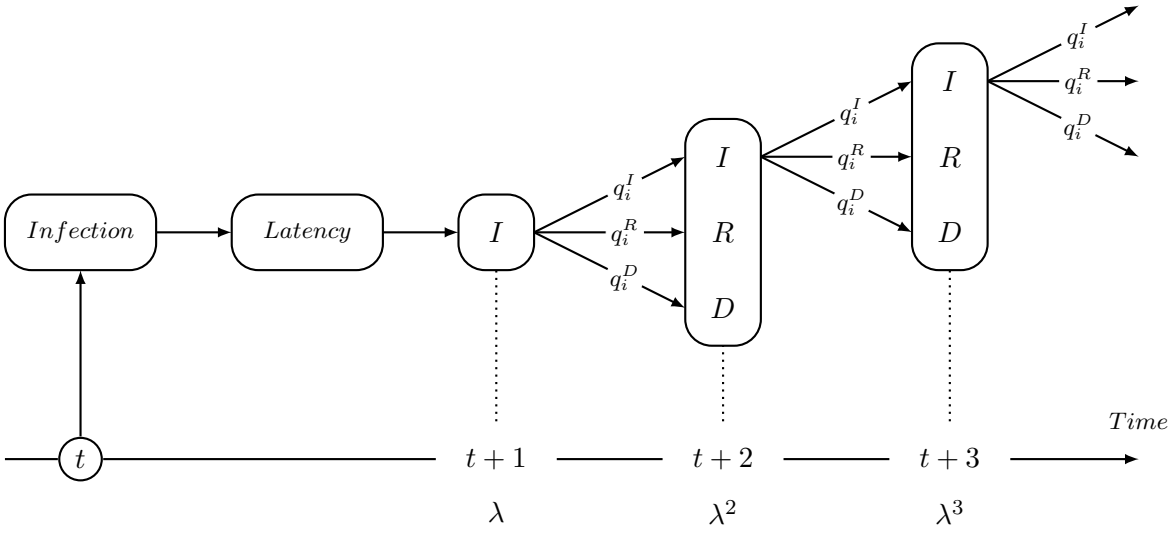

Figure B.1. Chain of possible epidemiological patterns post-infection for the generic i-th individual that becomes infected at the arbitrary date t.

With time preferences summarized by the (common) discount factor $\lambda \in (0,1)$, the discounted sum in t of all future expected payoffs after infection, $\tilde{D}_i$, can be written as

$$\begin{aligned}
\tilde{D}_i &= \lambda^2 D\, q_i^D + \lambda^3 D\, q_i^I\, q_i^D + \lambda^4 D \left(q_i^I\right)^2 q_i^D + \lambda^5 D \left(q_i^I\right)^3 q_i^D + \ \ldots \ = \\
&= \lambda^2 q_i^D D \left[\ 1 + \lambda q_i^I + \left(\lambda q_i^I\right)^2 + \left(\lambda q_i^I\right)^3 + \ \ldots \ \right] = \\
&= \lambda^2 q_i^D D \left[\ \sum_{\tau=0}^{+\infty} \left(\lambda q_i^I\right)^\tau \right]
\end{aligned} \tag{B.1}$$

for every arbitrary date t. Note that both $\lambda \in (0,1)$ and $q_i^I\,(0,1)$, hence $\lambda\, q_i^I \in (0,1)$ holds and the geometric series in (B.1) converges to

$$\tilde{D}_i \ = \ \lambda^2 D \left(\frac{q_i^D}{1 - \lambda q_i^I} \right)$$

that, using (7), can be rewritten as

$$\tilde{D}_i \ = \ \lambda^2 D \left[\frac{q_i^D}{1 - \lambda\,(1-\gamma)\left(1 - q_i^D\right)} \right] \ , \tag{B.2}$$

i.e. as a function of the sole probability of death per-period q_i^D. Substituting into equation (B.2) above for the definition (3) of q_i^D provided in the main text, we finally obtain

$$\tilde{D}_i \ = \ \lambda^2 D \left[\frac{\Phi\big(\,\theta + \alpha x(i)\,\big)}{1 - \lambda\,(1-\gamma)\left(1 - \Phi\big(\,\theta + \alpha x(i)\,\big)\right)} \right]$$

that is exactly expression (11) in the main text.

B.2 Proof of Corollary 4

When the epidemiological states $e(i)$ are common knowledge, the differential payoff $\Delta_i^{CK}\,(A)$ can be written as

$$\begin{aligned}
\Delta_i^{CK}\,(A) &= -c + \beta\, G_{CK} \\
&= -c + \beta\,(ASY) \left[R + S\,(1 - LA) \right]^{\phi - 1} \ ,
\end{aligned} \tag{B.3}$$

with $\phi \geq 0$ the scaling coefficient of the congestion externality $\varphi_{CK}(A)$. Differentiating (B.3) w.r.t. A we obtain

$$\frac{\partial}{\partial A}\Delta_i^{CK}(A) \;=\; -\beta(ASY)(S)(\phi-1)\Big[ASY+R+S(1-LA)\Big]^{\phi-2} \tag{B.4}$$

that clearly reveals that

$$\begin{aligned}
\phi \in (0,1) \quad &\Longrightarrow \quad \frac{\partial}{\partial A}\Delta_i^{CK}(A) \;>\; 0 \\
\phi = 1 \quad &\Longrightarrow \quad \frac{\partial}{\partial A}\Delta_i^{CK}(A) \;=\; 0 \\
\phi > 1 \quad &\Longrightarrow \quad \frac{\partial}{\partial A}\Delta_i^{CK}(A) \;<\; 0
\end{aligned}$$

so that the individual decisions to comply with norms are: (i) strategic complements if $\phi \in (0,1)$; (ii) non-strategic if $\phi = 0$; (iii) strategic substitutes if $\phi > 1$.

Appendix C.

SIR MODEL WITH ENDOGENOUS COMPLIANCE

C.1 Equations

In this Appendix we present the equations that describe the augmented SIR environment simulated and commented in Section 4 in the main text. Except for the law of motion of the new infected (NI_t), that is governed by the endogenous compliance mechanism pinned down by equilibrium of the (static) social-interaction game, and for those of the new dead (ND_t) and the new recovered (NR_t), that are devised to keep track of the cohort-specific mortality rates induced by endogenous compliance, the equations are quite standard.

C.1.1 Laws of Motion

Law of motion of the population

$$Pop_{t+1} = Pop_t - ND_t \ . \tag{C.1}$$

Law of motion of the susceptibles

$$S_{t+1} = S_t - NI_t \ . \tag{C.2}$$

Law of motion of the infected

$$I_{t+1} = I_t + NI_t - ND_t - NR_t \ . \tag{C.3}$$

with the corresponding law of motion of the asymptomatic infected

$$ASY_{t+1} = ASY_t + p\,(NI_t - ND_t - NR_t) \ . \tag{C.4}$$

Law of motion of the recovered

$$R_{t+1} = R_t + NR_t \ . \tag{C.5}$$

Law of motion of the dead

$$D_{t+1} = D_t + ND_t \ . \tag{C.6}$$

C.1.2 Variations Intra-Period

New infected

$$NI_t \;=\; \beta\left(S_t\right)\left(ASY_t\right)\left(1-LA_t^*\right)^2\left[\left(1-LA_t^*\right)\left(\,ASY_t+S_t+pR_t\,\right)+\left(1-p\right)R_t\right]^{\phi-1}\;. \tag{C.7}$$

New recovered

$$\begin{aligned} NR_t \;&=\; \sum_{\tau=0}^{t}\bar{Q}_\tau^R\,NI_\tau\left(1-\bar{Q}_\tau^D-\bar{Q}_\tau^R\right)^{t-\tau} \\ &=\; \gamma\sum_{\tau=0}^{t}NI_\tau\left(1-\gamma\right)^{t-\tau}\left(1-\bar{Q}_\tau^D\right)^{t+1-\tau}\;. \end{aligned} \tag{C.8}$$

New dead

$$ND_t \;=\; \sum_{\tau=0}^{t}\bar{Q}_\tau^D\,NI_\tau\left(1-\bar{Q}_\tau^D-\bar{Q}_\tau^R\right)^{t-\tau}\;. \tag{C.9}$$

C.1.3 Additional Simulations

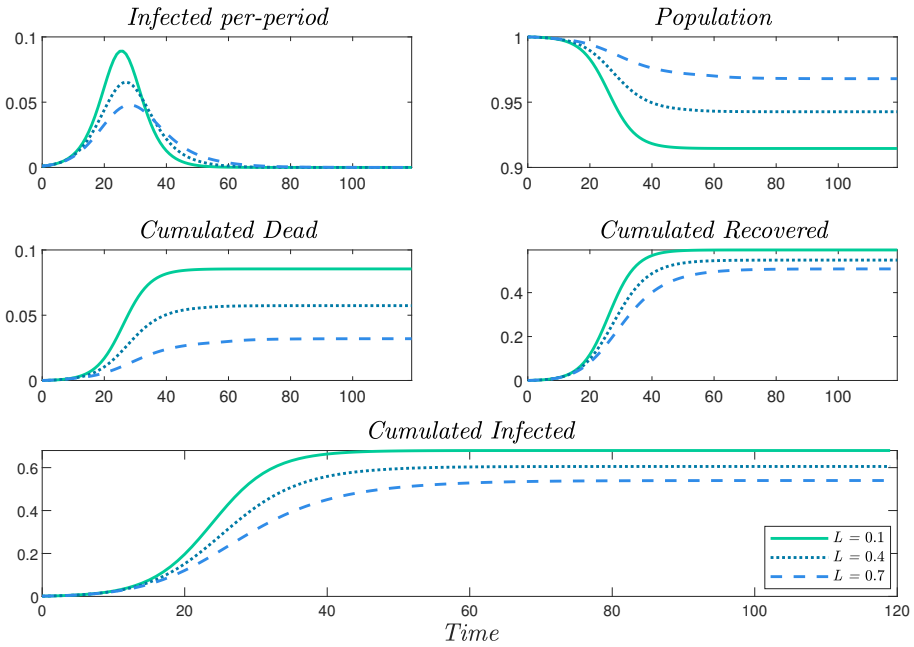

Figure C.1. Dynamics of the main aggregates: several values of L, $\phi = 0.3$.

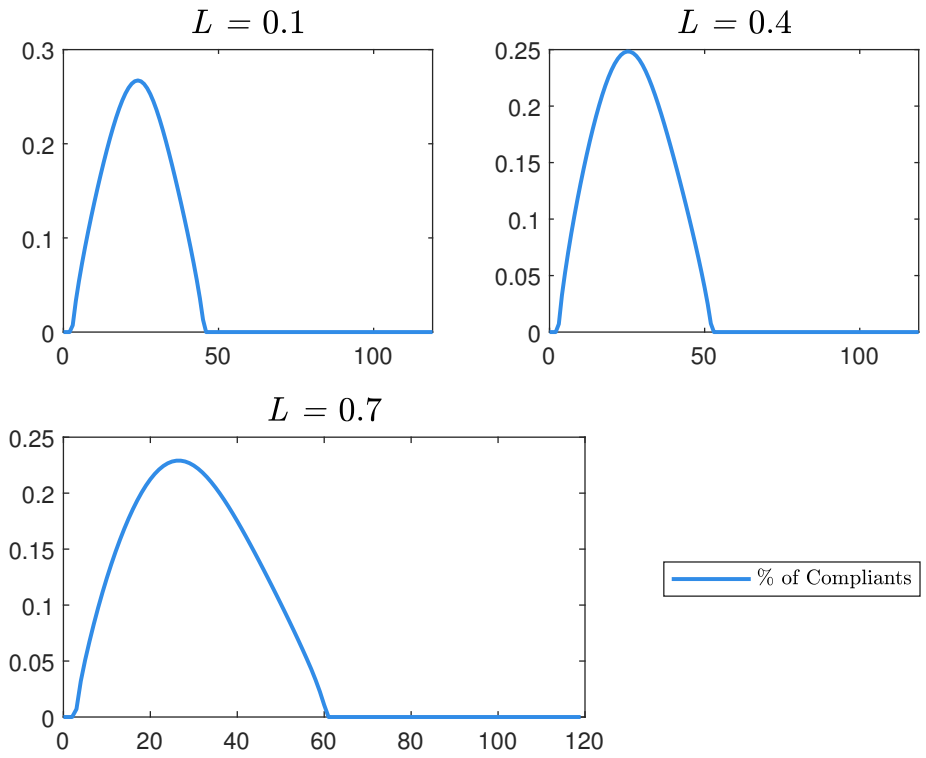

Figure C.2. Endogenous compliance: several values of L, $\phi = 0.3$.

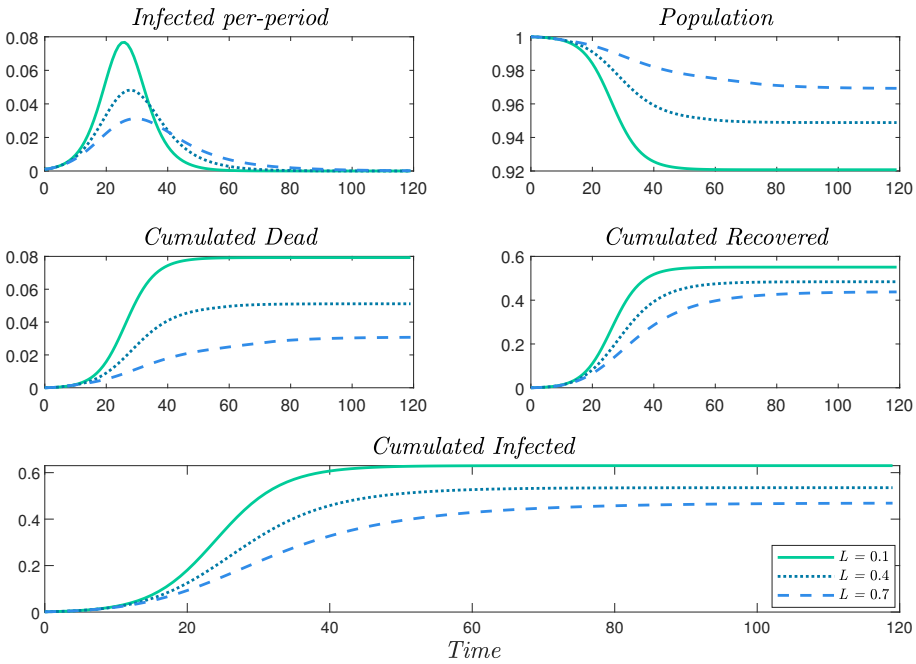

Figure C.3. Dynamics of the aggregates: several values of L, $\phi = 1$.

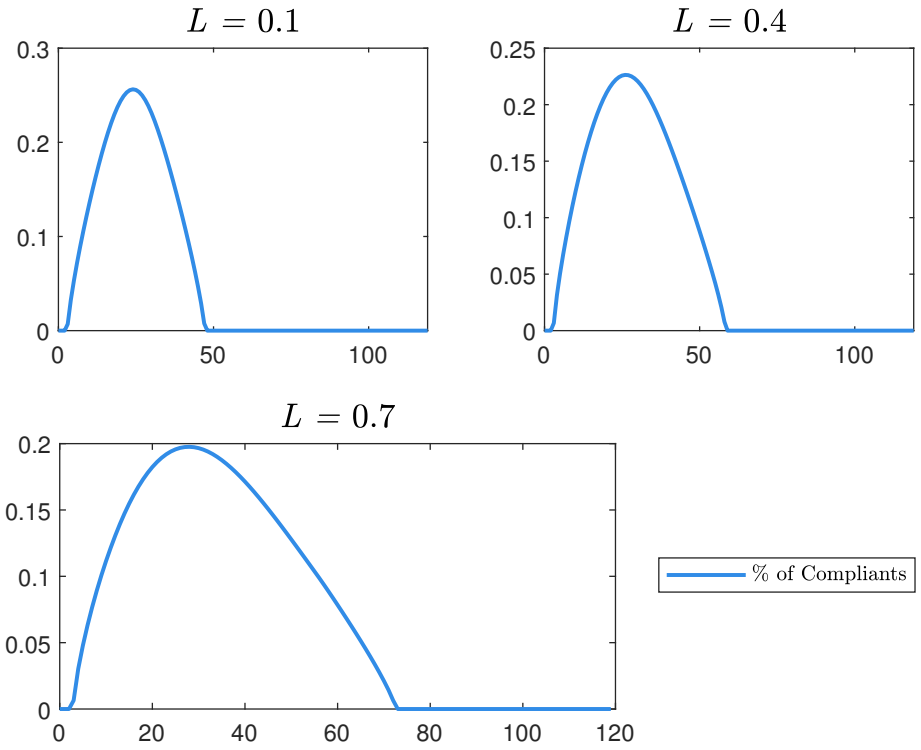

Figure C.4. Endogenous compliance: several values of L, $\phi = 1$.

Appendix D.
CONGESTION VS. PREVALENCE:
A (TOY) MODEL OF LOCAL INTERACTION

D.1 Action-Contingent Probability of Infection: A Recap

The probability of infection faced by a susceptible individual in our model is determined by (i) the extent of his/her social interactions, (ii) the prevalence of the disease within the interacting population, and (iii) the environment in which interactions do occur. To the single decision-maker, individual social behaviour is a *control variable*, while others' amounts to a pure – global – *externality*, that we model as a payoff-relevant spillover $G(A) \in [0,1]$ mapping the *aggregate* social behaviour of agents, as measured by their median/average compliance rate $A \in [0,1]$ with social-distancing norms, into the *individual* risk of contagion they face when non-compliant. Notwithstanding the continuum-player specification, actual strategic reasoning is therefore involved in the decision-making process of the (infinitely many) agents that populate our model, for the rational anticipation of A at the moment they decide whether or not to comply entails a partial internalization of the global spillover $G(A)$ – see Subsection 2.3 in the main text for the details. Recall from equation (10) that, formally, the global externality $G(A)$ can be written as

$$G(A) \;=\; \pi(A)\;\varphi(A)\;\;,$$

where $\varphi(A)$ and $\pi(A)$ are its extensive- and an intensive-margin component, respectively. The extensive-margin $\varphi(A)$ relates to the fact that the marginal effect on the risk of contagion faced by a non-compliant individual of an increase in the number of social interactions *per se* depends on the congestion of the environment in which interactions actually occur. The effect is unambiguously *positive*: increased congestion always entails an higher risk of contagion, *ceteris paribus*. The

intensive-margin $\pi(A)$ relates to the fact that the risk of contagion in social interactions depends on the composition of the interacting (sub)population in terms of group-specific disease prevalence, and that the latter may *not* remain constant as the mass of interacting individuals varies. This is the case, for instance, if some types of agents systematically ignore social-distancing norms more frequently than others.

In 2.3.4 we explained the logic behind this structure by means of a practical example. Some individuals enter sequentially a room of given width: some of them are infected, the others are not. As more and more individuals arrive, the room gets crowded, until overcrowding finally obtains – the congestion externality $\varphi(A)$, that increases in in the mass of interacting individuals. Keeping constant the rate of new arrivals, the speed at which overcrowding occurs is determined by the width of the room – parameter ϕ –. As the room gets more and more crowded, interactions become more and more concentrated. Concentration increases the likelihood of contagion when an individual interacts with an infected counterparty (due to increased physical proximity), but it is completely *irrelevant* when he/she interacts with a non-infected – the intensive-margin effect $\pi(A)$, whose effect is ambiguous. Overall, the net effect of increased concentration on the likelihood of contagion is entirely determined by the composition of new arrivals. If disease-prevalence among the individuals in the queue remains constant, the effect is unambiguously positive: the probability of meeting an infected in the room is stable, but the physical proximity of contacts increases. If disease-prevalence decreases with new arrivals (e.g. as time passes susceptible individuals only remain in the queue), physical proximity increases but the probability of interacting with an infected counterparty in the room decreases. The net effect depends on the relative magnitude of the two margins. In this Appendix we formalise the example of the room with sequential entering by means of a simple model of local interaction, and show that, within the limits of the model, the qualitative argument seems indeed to hold.

D.2 A Reduced-Form Model of Local Interaction with Congestion

In the spirit of [Schelling, 1969, Schelling, 1971] and, more recently, [Blume, 1993, Blume, 1995] and [Morris, 2000][D1], we account for the spatial dimension of (pairwise) social interactions in reduced-

[D1] See [Blume et al., 2005] for an excellent survey on local-interaction games.

form by assuming that (i) interacting units be pre-assigned specific locations onto a segment of given length[D2], and that (ii) interactions be *local* in the sense that the physical distance between two interacting units entirely determines the intensity of their interaction.

D.2.1 Setup

Consider a stylized environment populated by a *finite* number $M \gg 2$ of non-strategic individuals – the *units* –, that interact within a closed space – the *room* – whose spatial extension is parameterised by $W > 0$ – its *width*. Each unit is exogenously assigned to one of two (mutually exclusive) subpopulations: that of the Infected (I) – indexed by $j = 1, 2, \ldots$ – and that of the Healthy (H) – indexed by $h = 1, 2, \ldots$ Indicating with $\bar{I}$ and $\bar{H}$ the total number of infected and healthy units, respectively, the identity

$$\bar{I} + \bar{H} = M$$

must therefore hold. Infected units are infective, and social interactions with them entail, for healthy individuals, a risk of contagion proportional to physical proximity – the closer the contact, the higher the probability of infection, *ceteris paribus*. Social interactions with healthy units are completely risk-free. Locations $\ell \in [0, W]$ in the room are (arbitrarily) ordered from left to right, and assigned separately to the units in each subpopulation according to their indexes – that is, $\ell = 0$ is the first available location, $\ell = W$ is the last one, the unit $j = 1$ gets the first location available to the infected subpopulation, the unit $h = 1$ gets the first location available to the healthy one, etc... Finally, within subpopulations, locations are assigned according to two criteria: (**C1**) all units must be evenly distributed along the entire width W; (**C2**) all distances between neighbouring units must be identical. Formally, it is immediate to check that, indicating with ℓ_j and ℓ_h the locations of the j-th infected and of the h-th healthy unit, respectively, the pair of equations

$$\ell_{j=1} = 0 \tag{D.1a}$$

$$\ell_{j+1} = \ell_j + \frac{W}{\bar{I} - 1} \tag{D.1b}$$

[D2] This is the simplest modelling strategy available, that we adopt in this Appendix for the sake of analytical tractability. Alternative, more complex approaches are available – among others: (i) interactions on fully specified networks – see e.g. [Herrmann and Schwartz, 2020] and [Block et al., 2020]; (ii) interactions on lattices – see e.g. [Bak et al., 1993]; (ii) random walks with matching in two-dimensional spaces – see e.g. [Bisin and Moro, 2022a, Bisin and Moro, 2022b].

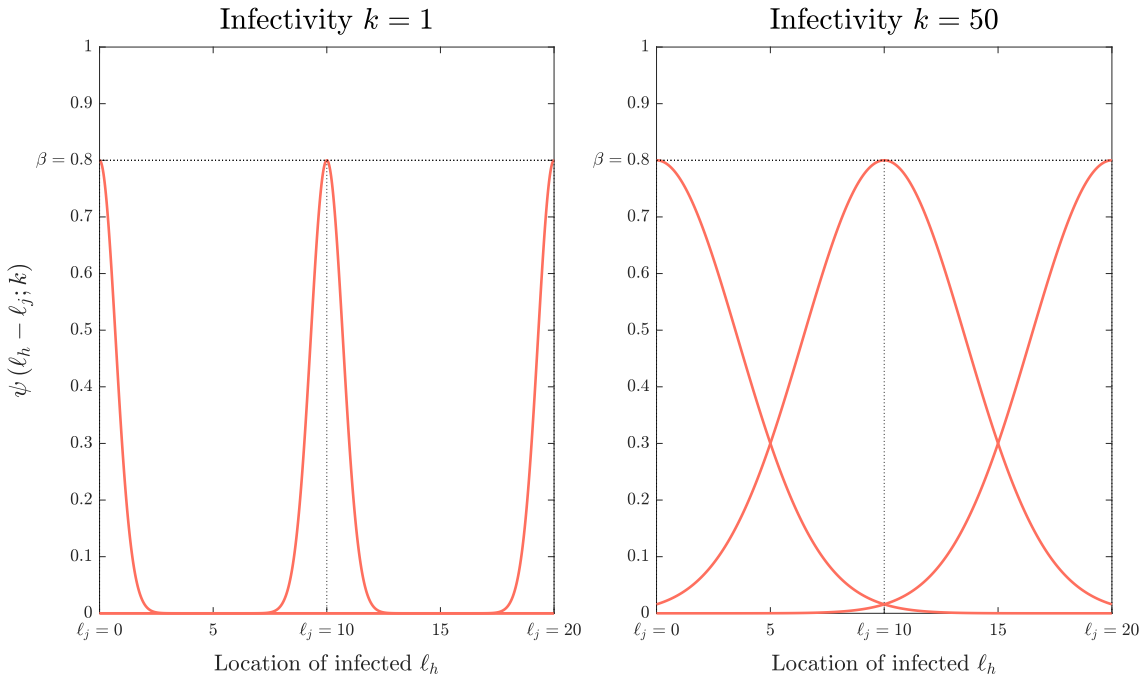

Figure D.1. Pairwise probability of infection $\psi(\ell_h - \ell_j)$ as a function of ℓ_h – with $\bar{I} = 3$ infected units within a room of width $W = 20$. Contagiousness at zero-distance is $\beta = 0.8$. Comparison between low infectivity ($k = 1$, left-hand side) and high infectivity ($k = 50$, right-hand side).

for the infected subpopulation, and, similarly, the pair of equations

$$\ell_{h=1} = 0 \tag{D.2a}$$

$$\ell_{h+1} = \ell_h + \frac{W}{\bar{H} - 1} \tag{D.2b}$$

for the healthy subpopulation, allocate locations consistently with the criteria (**C1**)-(**C2**).

D.2.2 Contagion

In Subsection D.2.1 we assumed that the probability of contagion upon interaction with an infected decay with the physical distance between the interacting units. To impose additional structure on the model, we assume further that the decay be *exponential*. Formally, indicating with $\psi_{h,j} \in [0, 1]$ the probability of infection from an infected unit in location ℓ_j faced by a healthy unit in location ℓ_h, we assume that

$$\psi_{h,j} = \psi(\ell_h - \ell_j) = \beta \exp\left\{ -\frac{1}{k}(\ell_h - \ell_j)^2 \right\}, \tag{D.3}$$

with $\beta \in (0, 1]$, and where the coefficient $k > 0$ scales the infectivity of the disease – the larger k, the larger the distance at which an infected unit may infect a healthy one with non-zero probability. Note that

$$\psi(0) = \beta ,$$

so that β can be interpreted as the probability of infection in a zero-distance contact – i.e. in the closest possible interaction between units. Figure D.1 provides a graphical representation.

Note that, *a priori*, any of the $\bar{I}$ infective units in the room can infect any of the $\bar{H}$ healthy ones. Taking the perspective of the generic h-th healthy unit, therefore, any (location-based) interaction with an infected, if considered in isolation, is akin to a Bernoulli experiment – whose outcome is contagion with probability $\psi_{h,j}$. However, an healthy unit can be actually infected *at most once*. As a consequence, for any given location ℓ_h, the probability of infection *ex ante* is the outcome of a collection of $\bar{I}$ Bernoulli experiments that are *not* independent. In particular, it must hold that

$$\Pr\left(h \text{ infected by } j' \,|\, h \text{ infected by } j \neq j'\right) = 0$$

for every $h \in \{1, 2, \ldots, \bar{H}\}$ and every $j \neq j'$ with $j, j' \in \{1, 2, \ldots, \bar{I}\}$. We derive the marginal probabilities of our non-standard statistical experiment starting from the probability space of a standard binomial experiment with $\bar{I}$ Bernoulli trials, and normalizing the corresponding probabilities by the total probability mass of the outcomes that can occur with non-zero probability in our experiment. Such events are of two types:

(A) the healthy unit h is infected by the infective unit j and not by any of the other infective units $\neq j$;

(B) the healthy unit h remains healthy – i.e. it is *not* infected by any of the $\bar{I}$ infective units in the room.

The corresponding (total) probability mass $\Sigma(\ell_{h=\bar{h}})$ is therefore

$$\boldsymbol{\Sigma}(\ell_{h=\bar{h}}) = \underbrace{\sum_{j=1}^{\bar{I}} \left[\psi_{\bar{h},j} \prod_{j' \neq j} \left(1 - \psi_{\bar{h},j'}\right) \right]}_{\text{Tot. probability mass events of type (A)}} + \underbrace{\prod_{j=1}^{\bar{I}} \left(1 - \psi_{\bar{h},j}\right)}_{\text{Tot. probability mass event (B)}} \tag{D.4}$$

so that the overall *ex ante* probability of contagion faced $\hat{\Psi}\left(\ell_{h=\bar{h}}\right)$ by the generic healthy unit $h=\bar{h}$ in located at $\ell_{\bar{h}}$ can be written as

$$\hat{\Psi}\left(\ell_{h=\bar{h}}\right) \;=\; \frac{\sum_{j=1}^{\bar{I}}\left[\psi_{\bar{h},j}\prod_{j'\neq j}\left(1-\psi_{\bar{h},j'}\right)\right]}{\sum_{j=1}^{\bar{I}}\left[\psi_{\bar{h},j}\prod_{j'\neq j}\left(1-\psi_{\bar{h},j'}\right)\right] \;+\; \prod_{j=1}^{\bar{I}}\left(1-\psi_{\bar{h},j}\right)} \quad , \tag{D.5}$$

that is the probability of being infected by one of the $\bar{I}$ infective units when being located at $\ell_{\bar{h}}$.

D.2.3 Estimated Probability of Contagion

Before entering the room, a healthy unit knows both how many infected units are in the room ($\bar{I}$) and where they are located (positions ℓ_j). However, it ignores which of the $\bar{H}$ locations ℓ_h available to healthy units it will be assigned to, once in the room. As a consequence, it ignores, too, the (location-dependent) probability of contagion it will face, once in the room. Since locations are randomly allocated, however, each al(location) is equally likely *a priori*. Collapsing all the structural parameters that define the environment in which interactions occur into a single vector $\mathbf{\Omega}$, the probability of contagion $\mathbf{\Psi}\left(\bar{h};\mathbf{\Omega}\right)$ estimated *a priori* by the generic healthy unit $h=\bar{h}$ that is about to enter a room (i) of width W, (ii) with $\bar{I}$ infective units evenly distributed along W, and (iii) with $\bar{H}$ locations available to healthy units evenly distributed along W, can be written as

$$\mathbf{\Psi}\left(\bar{h};\mathbf{\Omega}\right) \;=\; \frac{1}{\bar{H}}\left[\sum_{h=1}^{\bar{H}}\hat{\Psi}\left(\ell_h\right)\right] \quad , \tag{D.6}$$

with $\hat{\Psi}\left(\ell_h\right)$ the (expected) probability of contagion when located at ℓ_h defined by (D.5).

D.3 Scenario Analysis

To be able to decide whether or not to observe social-distancing norms, the rational, strategic agents that play the interaction game outlined in the main text are called to anticipate other's social behaviour in order to properly assess the risk of contagion in interactions. In his/her mind, every agent is called to perform a sort of "scenario analysis" by asking: "what is the risk of contagion I would face by interacting socially when M other agents are going to do the same?" What if, instead,

the interacting mass is $M' > M$?" And so on... In this subsection, we take the perspective of one of the non-strategic units that populate this toy model, and let it perform the same scenario analysis. By following its estimates *ex ante* – before entering the room – of the probability of infection it will face *ex post* – once in the room –, we are able to see how they vary with (i) the total number of interacting units $M = \bar{H} + \bar{I}$, (ii) the total number of infective units $\bar{I}$, and (iii) the width W of the room. The scenario analysis performed by the unit substantiates in keeping track of its estimates *ex ante* of the probability of contagion upon interaction in the room, $\boldsymbol{\Psi}\left(\bar{h}; \boldsymbol{\Omega}\right)$, as additional units are assumed to enter into the room. The process that governs the new arrivals is known, and entails that, at every iteration, a cohort of 10 units enters the room. The share $\delta_I \in [0, 1]$ of infective units in each cohort is assumed to be (i) stable over iterations, and (ii) known to the unit that performs the estimation – hence, to the modeller that keeps track of it! We simulate two distinct "worlds": one in which the share of infected units within the room remain constant as new cohorts arrive; the other, in which such share decrease at every iteration. The first world approximates the best-response dynamics of the social-interaction game played by *forgetful* citizens, where individual actions are always strategic substitutes – see Sections 2 and 3). The second world approximates the best-response dynamics of the social-interaction game played by citizens that are aware of their epidemiological states, where individual actions can be both complements and substitutes depending on the magnitude of the congestion externality – see Section 5.

D.3.1 Constant Share of Infective Units in New Arrivals

In this world, every new cohort that enters the room has a stable composition, with $\delta_I \in (0, 1)$ its (constant) share of infective units. As new cohorts arrive, the room gets more and more crowded, but the share of infective units in the room remains constant *by construction*. This controlled simulation is intended as a robustness check for the argument

> *If the group-specific prevalence remains constant or increases as more an more individuals interacts, then the risk of infection unambiguously increases in the total mass of interactions.*

that rationalises, in the main text, the formal result that individual decisions to comply with social-distancing norms are always strategic substitutes when agents ignore their epidemiological status. We calculate the probability of infection $\boldsymbol{\Psi}\left(h; \boldsymbol{\Omega}\right)$ for several (fixed) shares of infective units δ_I and

several values of the width W.

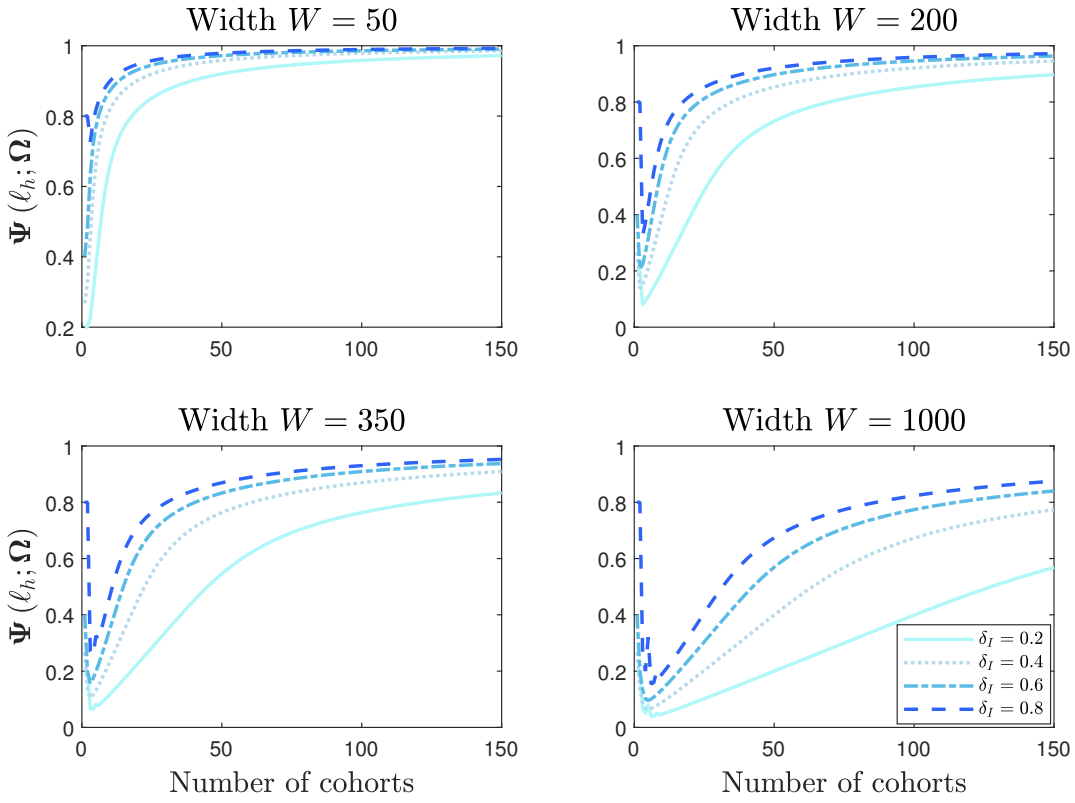

Figure D.2. Probability of infection $\mathbf{\Psi}(h; \mathbf{\Omega})$ estimated for an increasing number of cohorts with constant shares δ_I.

The three figures correspond to three sets of initial conditions. It is immediate to check that, except for the initial iterations, the estimated probability of infection steadily increases as the number cohort cumulates in the room. The width W parameterises how quickly congestion occurs, and can therefore be considered as a proxy of the ϕ parameter that scales the congestion externality in the main text. The evolution of $\mathbf{\Psi}(\Omega)$ is stable across all the several value of W, suggesting that the substitutability-only result when the group-specific disease-prevalence is non-decreasing in the interacting mass may be quite robust.

D.4 Decreasing Share of Infective Units in New Arrivals

In this world, every new cohort that enters the room contains healthy units only – i.e. $\delta_I = 0$. The number of infective units in the room is therefore equal to that imposed via initial conditions,

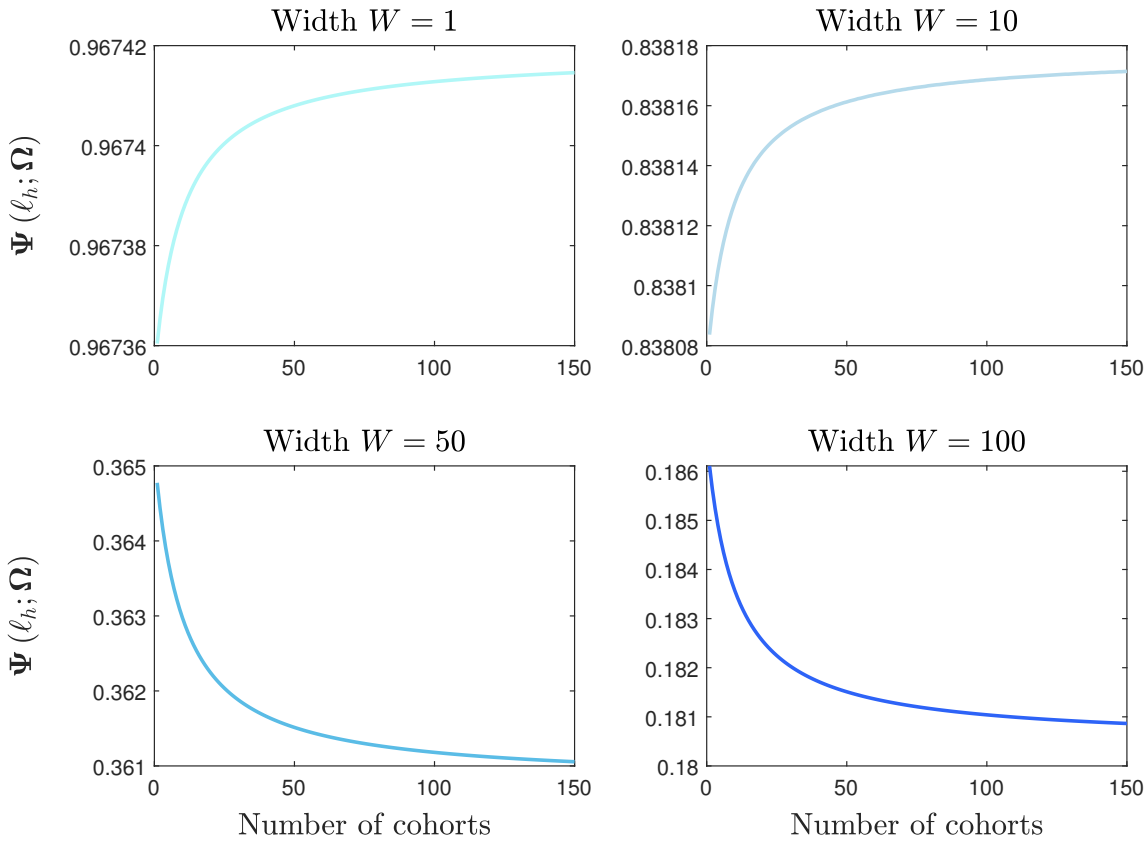

Figure D.3. Blabla

and the disease prevalence in the room steadily decreases as new cohorts arrive. This controlled simulation is intended as a robustness check for the argument

If the group-specific prevalence decreases as more an more individuals interacts, then the risk of infection increases or decreases depending on the relative magnitude of the congestion externality

that rationalises, in the main text, the formal result that individual decisions to comply with social-distancing norms can be both strategic substitutes and complements when every agent is aware of his/her epidemiological state, and conditions on that his/her decisions about social interactions.