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ROTATION AS CONTAGION MITIGATION

Abstract

We study rotation schemes that govern individuals' activities within an organization during an epidemic. We optimize the frequency of rotation and degree of cross-mixing of the rotating subpopulations. Frequency affects risk over the length of diffusion within the infected subpopulation until the organization detects and/or reacts to the infection. If the reaction time is short, then such risk is undesirable since the growth of the prevalence is initially convex in time. Frequent rotation, which acts as insurance against exposure time risk, is then optimal. Infrequent rotation becomes optimal if the organization reacts slowly. Mixing of the rotating subpopulations is detrimental because it increases the share of interactions between sick and healthy individuals. However, the effect of mixing is small if the terminal prevalence is low in the absence of mixing.

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Rotation as Contagion Mitigation*

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Abstract

To prevent the spread of a disease an organization obeys social distancing restrictions and thus limits the number of its members physically present on a given day. We study rotation schemes in which mutually exclusive groups are active on different days. The frequency of rotation affects risk over the duration of diffusion prior to the time the organization is able to react to the infection. If this reaction time is speedy, then such risk is undesirable since prevalence is initially convex in time. Then, frequent rotation acts as insurance against exposure-time risk and is optimal. Infrequent rotation becomes optimal if the organization reacts slowly. Cross-mixing of the rotating subpopulations is detrimental because it increases contacts between sick and healthy individuals. However, the effect of mixing is small if the terminal prevalence is low in the absence of mixing.

1 Introduction

At the outset of a disease outbreak, the main instrument available to decision-makers to contain the epidemic is to regulate and shape meeting rates. Strict social distancing measures are highly successful in flattening the contagion curve. However, they are costly economically and socially and they are not viable for a prolonged time. It is therefore important to design smarter mechanisms that can shape social interaction to minimise the likelihood of new outbreaks and, at the same time, resume and maintain economic activities over time.

This paper focuses on how to organize group rotation. We consider the following problem. An organization has $2K$ members but, due to mitigation measures like social distancing, can

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only accommodate the presence of K members per day. One individual gets infected at random but the infection is initially not observable. The infection diffuses within an active group until the organization reacts at which time the infected individuals are counted and isolated. It takes time for the organization to react due to the presence of asymptomatic individuals (e.g. in schools (Viner et al., 2020)), delayed display of symptoms and a variety of organizational frictions.[1] Taking this reaction time as given we explore the best way to rotate groups in order to minimize the expected terminal infection.

We show that optimal rotation schemes are extreme. First, rotation should be *strict*: once the organization has been divided into two groups, these groups should continue to be rotated without cross-mixing. Second, optimal rotation among these groups is either very frequent with small persistence or very infrequent with high persistence. In particular, frequent rotation maximizes risk sharing of the length of exposure to the infection across the two groups. When detection speed is slow relatively to contagion then risk sharing is desirable and frequent rotation is the optimal. Otherwise the policy-maker prefers to concentrate risk and infrequent rotation is preferred.

The key intuition comes from the observation that the diffusion process has two phases. Initially the infection grows approximately exponentially. As the disease has spread sufficiently enough within a group, infection starts growing at a decreasing rate. Precisely, if the *prevalence*, i.e. the infected fraction among active group members, is p, then the (expected) number of new infections will be

$$g(p) = p(1-p),$$

in line with the standard logistic-growth process, see Figure 1. The growth function g is hump-shaped so that when prevalence is still low the growth rate is increasing, whereas when prevalence is already high the growth rate declines.

Figure 1 can be used to illustrate the forces that determine the optimal rotation scheme. Consider *strict rotation*: the organization is divided into two equally sized groups A and B and activity alternates between them without any cross-mixing. If group A includes the originally infected member then it will have experienced some contagion and its prevalence p_A will be larger the more frequently it has been active. If the original infection occurred in group B then group A will have a prevalence of zero, with both these possibilities being equally likely.[2]

[1] As a concrete example, an Israeli high school that has reopened in early May 2020, has closed due to recurring infection in about a month later, at which point the count of infected students and teachers has reached 130. See `https://www.npr.org/sections/coronavirus-live-updates/2020/06/03/868507524/israel-orders-schools-to-close-when-covid-19-cases-are-discovered?t=1592383905674`

[2] It is also possible that there has yet to be any initial infection but in this case, the decision of how to proceed with rotation going forward has no consequence.

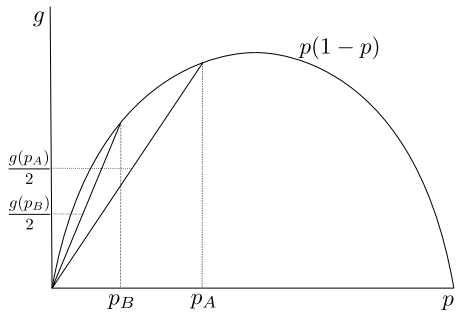

Figure (a) Frequent Rotation

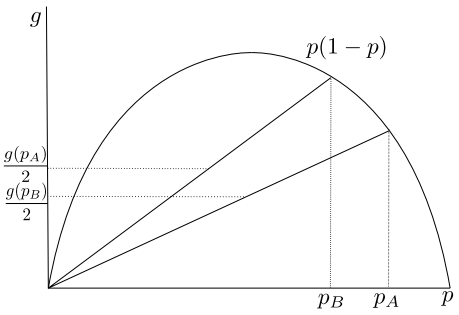

Figure (b) Infrequent Rotation

Figure 1: Strict Rotation

If the diffusion is still in the early phase, as depicted in Figure 1a, then activating the group which has been less active, here group B, will result in a smaller expected number of new infections (in particular $g(p_B)/2$ versus $g(p_A)/2$). Thus, in the early phase *frequent rotation* minimizes the growth of the infection by ensuring that over all short time intervals the two groups have approximately equal shares of activity.

By contrast if the infection has reached the second phase, as depicted in Figure 1b, then it is the group which has been *more* active that generates a smaller number of new infections; in a sense, this group has got closer to the local "herd immunity". It is therefore optimal to repeatedly re-activate the group which has already accumulated the majority of activity. That is, rotation should stop.

However, the timing of the initial infection is unobserved by the policy-maker so she cannot condition her activation policy on which of these two cases applies. Nevertheless, we show below that the optimal policy has a bang-bang property depending on parameters. Frequent rotation is optimal when the organization reacts fast and when the environment is not too infectious. Otherwise it is optimal to rotate very infrequently with large persisetence. Frequent rotation is optimal when the organization will spend most of the time in the first phase of diffusion. Hence, there is an important complementarity between frequent rotation and other policy instruments available to the decision-maker like testing within the organization and interventions that decrease the rate of person-to-person transmission.[3] The size of each group also matters. Increase in the size of a group, keeping the within-group rate of transmission fixed, favours frequent rotation.

[3]See Ely et al. (2020) for the formulation of the test-allocation problem for the heterogeneous tests portfolio and the derivation of the monotonicity properties of the optimal test allocations in special settings.

The discussion thus far has been confined to strict rotation policies. A related mechanism explains why strict rotation is preferred over policies that mix previously rotated groups.

For illustration, suppose that frequent strict rotation between groups A and B has been followed up to time t. If the contagion is underway then each group is equally likely to have one of two prevalence levels: zero prevalence (if the infection occurred in the other group) or some positive prevalence level p. Therefore the *expected* number of new infections resulting from continuing with strict rotation and activating, say A again, is $g(p)/2$.

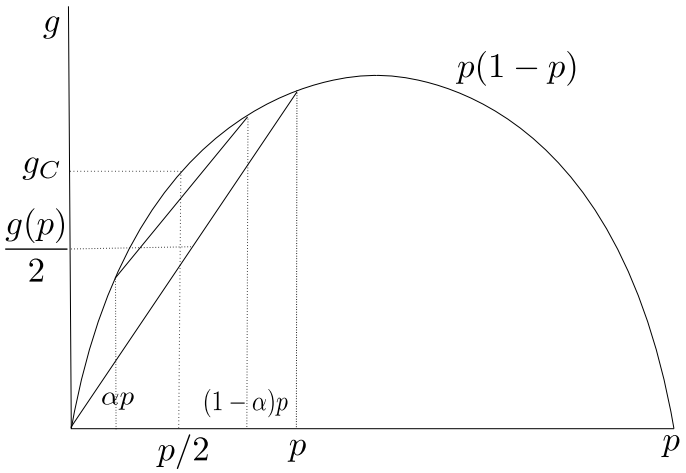

Figure 2: Mixing increases contagion.

Consider instead activating a group consisting of some fraction α of the members from A and $(1-\alpha)$ fraction of the members of B. The prevalence of the composite group will be either αp or $(1-\alpha)p$, each with equal probability, and so the *expected* prevalence level equals $p/2$. This is exactly the same as the expected prevalence level of group A or B. However, due to the strict concavity of the contagion rate, the composite group would yield a strictly higher expected number of new infections, see Figure 2.

A similar argument applies to deviations from any strict rotation policy, the key reason being that rotation maximizes the correlation in the infection states of group members, generating a mean-preserving spread in the group-aggregate random prevalence. The strict concavity of the contagion rate means that the policy-maker is risk-loving with respect to such spreads.

Our explanation allows us to quantify the adverse effect of the groups' mixing. When the growth function g can be well approximated by a linear function for the relevant range of the prevalence levels, then the effect of mixing is small. Using this, we show that when the

organization does not react too slowly, so that the terminal prevalence is not too large, then prevention of mixing has a second-order effect relative to the choice over the frequency of rotation. It follows that the optimality of frequent rotation is robust to some mixing across groups that could occur because of implementation mistakes.[4]

These intuitions are all based solely on a myopic goal of reducing new infections in the short run. A policy-maker who wishes to minimize the number of infections at the random time in which symptoms appear is willing to trade off an increase in infections in the short run in return for a reduction in infections later. Our formal analysis considers a fully optimal dynamic policy which indeed confronts such tradeoffs.

Karin et al. (2020) propose rotation of the economic activities as a safe path to reopening the economy.[5] We share with Karin et al. (2020) the motivation of understanding rotation mechanisms in the diffusion of disease, and our work is complementary to theirs. Their focus and recommendation over the rotation's frequency differ from those in our paper. They exploit particular biological facts about the length of the covid-19 incubation and infectiousness periods to design a cycle in which each person is active about one third of the times so that she is likely to be isolated during her possible infectious period. We, instead, focus on uncertainty over the exposure period and show that the frequency of rotation has large effects even if the timing of infectiousness is not carefully exploited. This is an adequate approach when there are organizational frictions and, therefore, organizations cannot be trusted to react immediately to the first displays of symptoms. Vice versa, there is no uncertainty over the length of the diffusion of the disease in Karin et al. (2020). Additionally, we require that half of the individuals are active at any time, and thus the policy experiment evaluated by Karin et al. (2020) is infeasible in our model. Finally, we explain the role of aggregate uncertainty over infections when we study the effect of mixing members of the rotating groups; aggregate uncertainty is absent in Karin et al. (2020). Relatedly, Akbarpour et al. (2020) show, in a SIR-based network model calibrated to two US agglomerations, that rotation of workforce has strong contagion-mitigation effect with small disruption costs.

Recent contributions in epidemiology, computer science and economics investigate targeted lock-downs and other social-distancing measures typically within variants of the SIR diffusion model, see, e.g., Acemoglu et al. (2020), Eichenbaum et al. (2020) and Brother-

[4]We consider only mixing of distinct groups that both rotate at a same frequency. Akbarpour and Jackson (2018) show, in a SIR framework, that heterogeneity in the frequency of activity speeds up diffusion. Their effect arises because those who alternate their activity frequently have a high chance of contracting the disease, whereas those with low frequencies are more likely to spread the disease once they get infected. This suggests that mixing of several groups that rotate at distinct frequencies may be detrimental.

[5]See Alon et al. (2020) for the economic branch of the project by Karin et al. (2020).

hood et al. (2020). We take a complementary micro-level approach and our analysis uncover some mechanisms that could allow decision-makers to re-design the workplace to increase organizational resilience to new Covid-19 outbreaks. We do not attempt to model the whole economy, and instead take the infection rate outside of the studied organization as given.

2 Example

A member of a large organization has been infected with a contagious disease at a random point in time. Let us assume that, initially, the disease spreads exponentially in the predominantly healthy organization; infections double every 5 days.[6] The organization reacts with a delay of 30 days from the initial infection; afterwards all its infected individuals are counted in mass testing. This scenario concludes with $2^6 = 64$ infections.

Let us, instead, divide the organization into two equally sized groups and let, at each instance, one group actively attend the organization whereas the other group be isolated. As an illustration we consider three schemes that differ in the frequency of rotation. First, under the simplest such scheme, a same half of the organization is active at any point of time, and the remaining half is perpetually isolated.[7] This scheme leads to $(1+2^6)/2 = 32.5$ infections, since the disease spreads only if the exogenously infected individual happens to be in the active half. Second, we rotate the two groups monthly. In this case, the infected group will be active for the exposure time of $1, 2, \ldots, 30$ days depending on the random time the initial infection has occurred. If the arrival of the initial infection is uniformly distributed over a large interval, then the number of the doubling cycles that occur before the organization reacts is uniformly distributed between 0 and 6. Thus, the expected number of infections is $\int_0^6 2^s \frac{ds}{6} \approx 15.2$. Third, an organization that rotates its two halves daily exposes the infected group to 15 active days for sure, that is, to 3 doubling cycles, which leads to $2^3 = 8$ infections only. Such frequent rotation is optimal.

Frequency of rotation determines a distribution of the exposure time for which the disease spreads in the infected half of the organization. Since it is initially unknown which half has been infected, the mean exposure time is 15 days for any rotation scheme. The schemes, however, differ in the risk over the exposure time. Permanent activation of the same group induces the extreme risk (six cycles or none), monthly rotation induces an intermediate risk and daily rotation no risk at all. If the goal of the decision-maker is to reduce the expected number of infections and the growth is convex, then the decision-maker is risk-averse with

[6] He et al. (2020) report time-span of 5.8 days between symptom onsets of successive infection generations of Covid-19 and the basic reproduction number of 2.2 to 2.5.

[7] In schools of many countries only certain grades of students attend the school after its reopening whereas other grades remain home-schooled, see Melnick and Darling-Hammond (2020).

respect to the exposure time; in this case frequent rotation allows for perfect insurance. If the infectious spread becomes large relative to the half of the organization, then the prevalence becomes concave with respect to the exposure time. In this case, the decision-maker is risk-seeking with respect to the exposure time, and activating one group perpetually is optimal.

Rotating individuals may wish to swap their attendance schedules, or the implementation of a rotation scheme may be imperfect. We show that the mixing of the two groups increases the disease prevalence. To see this, note that, without mixing, the health statutes of any two members of the same group are positively correlated. Such correlation is beneficial because the disease spreads only when two individuals with distinct health statuses interact. Mixing of the two groups undermines the correlation, and hence prevalence increases with the frequency of the swaps.

Perhaps surprisingly though, the effect of mixing is small unless a large proportion of the population gets infected. Indeed, whenever prevalence remains low until the organization reacts, most of the contacts of an infected individual are healthy peers, and hence the role of the correlation is limited. Using this argument, we provide an upper bound on the adverse effect of mixing in Proposition 3. For instance, if 10 people of an organization of size 100 get infected under a frequent rotation scheme in absence of mixing, then a flow of swaps of any intensity would lead to infection of less than one additional individual. Thus, in case of low final prevalence rates, regulation of swaps is secondary to the choice of the rotation frequency.

3 Rotation Problem

We begin by considering strict rotation policies. The organization is divided into two groups, $i \in \{0, 1\}$ each with measure $K > 1$.[8] At each point of continuous time $t \geq 0$, one of the two groups is active and the remaining group is isolated. A disease spreads in the currently active group according to a process specified below, and there are no transmissions among members of the inactive group. A *rotation schedule* specifies which group is active at each point in time. Formally, a rotation schedule is a map $r : \{0, 1\} \times \mathbf{R}_+ \longrightarrow [0, 1]$ such that $\sum_i r(i, t) = 1$ for each t. The rotation schedule governs the measure of time $\int_t^{t'} r(i, s)ds$ for which the group i has been active between any $t < t'$.

For instance, a rotation schedule such that $r(0, t) = 1$ for $t \in [0, \delta) \cup [2\delta, 3\delta) \cup [4\delta, 5\delta) \ldots$ and $r(1, t) = 1$ for $t \in [\delta, 2\delta) \cup [3\delta, 4\delta) \cup [5\delta, 6\delta) \ldots$ rotates the two groups each δ units of time, starting with activating the group 0. A rotation schedule $r(i, t) = 1/2$ for all t specifies

[8] The model readily extends to rotating three or more groups though we do not attempt to study trade-offs related to the number of the active individuals.

that both groups have been active for a same amounts of time within any time interval. The latter schedule can be approximated by the first one by choosing a small interval of rotation δ. In general, a rotation schedule with interior $r(i,t) \in (0,1)$ is a continuous-time abstraction that can be approximated by a frequent rotation of the two groups such that group i is active approximately $r(i,t)$ share of the time in a neighborhood of each point of time t.

All individuals are healthy at $t = 0$. At a random point of time t^*, an infection occurs in one of the groups, with each group equally likely to be infected.[9] To describe the uncertainty over the infected group, we introduce a state of the world $\omega \in \{0,1\}$ each occurring with probability half. We let $p_t^i(\omega) \in [0,1]$ denote the prevalence of the disease in group i in state ω at t – it is the probability that a random member of the group i is infected in state ω at t. We assume that $p_{t^*}^i(\omega) = 1/K$ for $i = \omega$ and $p_{t^*}^i(\omega) = 0$ for $i \neq \omega$. That is, measure 1 of individuals of the group $i = \omega$ gets infected at t^* and all the individuals from the other group remain healthy. By assuming that exactly one group gets infected, we implicitly assume that these exogenous infection events are rare so that the probability that both groups get infected within a relevant time period is negligible.

The disease prevalence in each group follows the logistic growth whenever the group is active for $t \geq t^*$,

$$\dot{p}_t^i(\omega) = \gamma r(i,t) p_t^i(\omega) \left(1 - p_t^i(\omega)\right). \tag{1}$$

The justification for equation (1) is standard and based on the uniform pairwise matching of active individuals. The parameter $\gamma > 0$ summarizes both the flow of pairwise contacts and the probability that a transmission occurs when healthy and infected individuals interact. The share of contacts that occur between a healthy and infected individuals is $p_t^i(\omega)\left(1 - p_t^i(\omega)\right)$. For a short time interval $[t, t+\Delta]$, the group i has been active approximately for $r(i,t)\Delta$ units of time, which has lead to the measure $\gamma r(i,t) p_t^i(\omega)\left(1 - p_t^i(\omega)\right)\Delta$ of new infections. Since there is no interaction between the two groups, only the prevalence in the infected group is nontrivial, whereas prevalence in the other group remains zero. We discuss mixing of the two groups in Section 5.

The disease evolution terminates at time $t^* + R$, at which point the organization reacts to displayed symptoms and counts all the infected via testing; the parameter R measures the delay between the initial infection and the organization's reaction. The reaction time R is a fixed parameter which accounts for the time before symptoms appear, the frequency of testing and the speed with which the organization responds.

The *rotation problem* is to choose a rotation scheme that minimizes the expected disease

[9] Though we assume that t^* is drawn from a distribution, the properties of this distribution are irrelevant for our result, and thus we do not introduce a notation for it.

prevalence at the point of the reaction,

$$\min_r \mathrm{E}\, p^{\omega}_{t^*+R}(\omega), \tag{2}$$

where the expectation is with respect to the date of infection t^* and the infected group ω. The rotation scheme r affects the prevalence via equation (1).

3.1 Reformulation

We notice that the terminal prevalence in the infected group depends only on the measure of time this group has been active between t^* and $t^* + R$; it is independent from all other details of the rotation scheme. To make this precise, let the *loss* $\ell(t)$ given by

$$\ell(t) = \frac{e^{\gamma(t-b)}}{1 + e^{\gamma(t-b)}},$$

be the solution to the logistic differential equation

$$\dot{\ell}(t) = \gamma \ell(t)(1 - \ell(t)) \text{ with } \ell(0) = 1/K.$$

That is, $\ell(t)$ is the prevalence in a population in which the disease spreads without interruptions for t units of time, starting with measure one of infections in a group of size K. The summary parameter

$$b = \frac{1}{\gamma} \ln(K - 1)$$

is the inflexion point; $\ell(t)$ is convex below and concave above b, and half of the group is infected at $t = b$.

Let *exposure time* $\tau = \int_{t^*}^{t^*+R} r(\omega, t)dt$ be the measure of time for which the infected group ω has been active between t^* and $t^* + R$; it is a random variable with stochasticity due both to the random infection time t^* and uncertainty over the infected group ω.

Lemma 1. *The random prevalence, once the organization reacts, is* $p^{\omega}_{t^*+R}(\omega) = \ell(\tau)$.

Proof of Lemma 1. The lemma follows from integration of equation (1) with respect to t from t^* to $t^* + R$ and change of the variable from t to $\hat{t} = \int_0^t r(\omega, s)ds$. □

Let F be the cumulative distribution function of the exposure time, τ.

Lemma 2. *Exposure time,* τ*, of the infected group has mean* $R/2$ *and its distribution is symmetric around* $R/2$*:* $F(\tau) = 1 - F(R - \tau)$ *for each* $\tau \in [0, R]$.

Proof of Lemma 2. The first statement follows from the second one. To prove the second statement, let the random variable $\tau^i = \int_{t^*}^{t^*+R} r(i,t)dt$ represent the length of time in which the group i has been active between t^* and $t^* + R$. Since one of the two groups is active at any point of time, i.e. $\sum_i r(i,t) = 1$, we have that $\tau^0 + \tau^1 = R$ for sure. The infected group ω is the group 0 or 1 with equal probabilities. Thus, we have for any $T \in [0, R]$,

$$\begin{aligned} F(T) &= \Pr(\tau \le T) \\ &= \frac{1}{2}\left(\Pr(\tau^0 \le T) + \Pr(\tau^1 \le T)\right) \\ &= \frac{1}{2}\left(\Pr(\tau^1 \ge R - T) + \Pr(\tau^0 \ge R - T)\right) \\ &= \Pr(\tau \ge R - T) = 1 - F(1 - T). \end{aligned}$$

□

Since the distribution F of the exposure time is symmetric around $R/2$, we have $\mathrm{E}_F\, \ell(\tau) = \frac{1}{2}\,\mathrm{E}_F\left[\ell(\tau) + \ell(R - \tau)\right]$, suggesting the following *relaxed rotation problem*:

$$\max_F \mathrm{E}_F\left[\ell\left(\tau\right) + \ell\left(R - \tau\right)\right] \tag{3}$$

$$\text{s.t.:}\ \ \mathrm{E}_F\,\tau = \frac{R}{2}. \tag{4}$$

Problem (3) is relaxed in that we do not require the distribution F to be symmetric nor we require the distribution of the exposure time to be implementable via a rotation scheme. It turns out, however, that the solution to the relaxed problem is in fact symmetric and implementable via a rotation scheme. Thus the solutions of the two problems correspond to each other.

4 Optimal Rotation Scheme

The constraint (4) highlights that the choice over the rotation schemes has no impact on the mean exposure time but it can, however, influence the risk over the length of the exposure time. Frequent symmetric rotation eliminates the risk: it implies that the exposure time is close to $R/2$ for sure. Very infrequent rotation, on the other hand, induces nearly maximal risk: the exposure time then attains values 0 or R with probabilities near half each. Whether risk over the exposure time is preferable depends on the curvature of the objective function in the relaxed problem (3).

Depending on the parameters, logistic growth function $\ell(t)$ is convex and then concave, or only concave, see Figure 3. The initial convex part arises due to approximately exponential

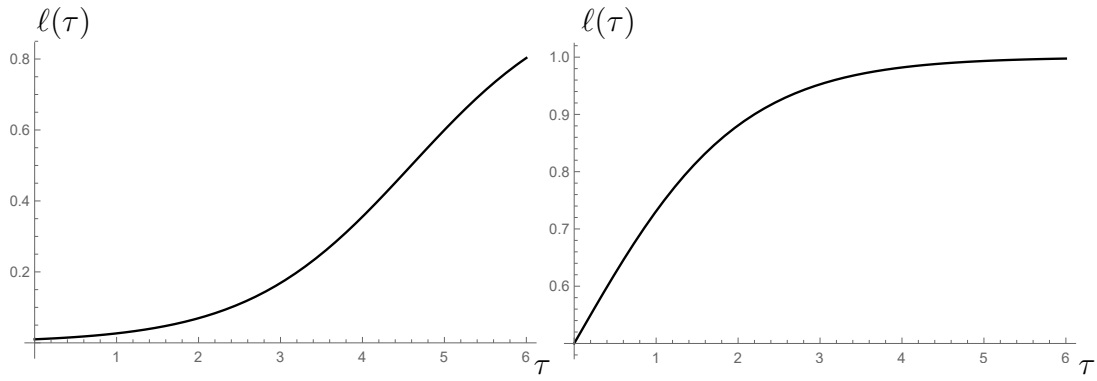

Figure 3: Function $\ell(\tau)$ for a large group size on the left-hand side ($K = 100$) and for a small group on the right-hand side ($K = 2$); $\gamma = 1$ in both graphs.

spreading in the predominantly healthy population. The logistic growth becomes concave once the population is sufficiently saturated with the disease when prevalence exceeds half.

The two phases of the logistic growth have opposite implications for the optimal choice over the rotation schemes. When the organization's reaction time, R, is short relative to the group size K, then $\ell(\tau)$ is convex for $\tau \in [0, R]$ and, therefore, risk over the exposure time is undesirable. In this case, frequent rotation is optimal. When the logistic growth function is concave for all τ, then an optimal scheme induces risk over the exposure time by always activating the same group. It turns out that one of these two rotation schemes is optimal also for all intermediate cases for which the logistic growth is first convex and then concave.

Lemma 3.

1. *If it takes more than half of the reaction time to infect half of the group, $R/2 < b$, then $\ell(\tau) + \ell(R - \tau)$ is U-shaped: it decreases for $\tau < R/2$ and increases for $\tau > R/2$.*

2. *If it takes less than half of the reaction time to infect half of the group, $R/2 > b$, then $\ell(\tau) + \ell(R - \tau)$ is hump-shaped: it increases for $\tau < R/2$ and decreases for $\tau > R/2$.*

Proof of Lemma 3. If $R/2 < b$, then $\ell'(\tau) < \ell'(R-\tau)$ for $\tau < R/2$, and thus $\ell(\tau) + \ell(R-\tau)$ decreases in τ on $[0, R/2]$. If $R/2 > b$, then $\ell'(\tau) > \ell'(R - \tau)$ for $\tau < R/2$, and thus $\ell(\tau) + \ell(R - \tau)$ increases in τ on $[0, R/2]$. The result then follows from the symmetry of ℓ. □

See Figure 4 for the shape of $\ell(\tau) + \ell(R-\tau)$. The next proposition characterises optimal rotation schemes.

Proposition 1.

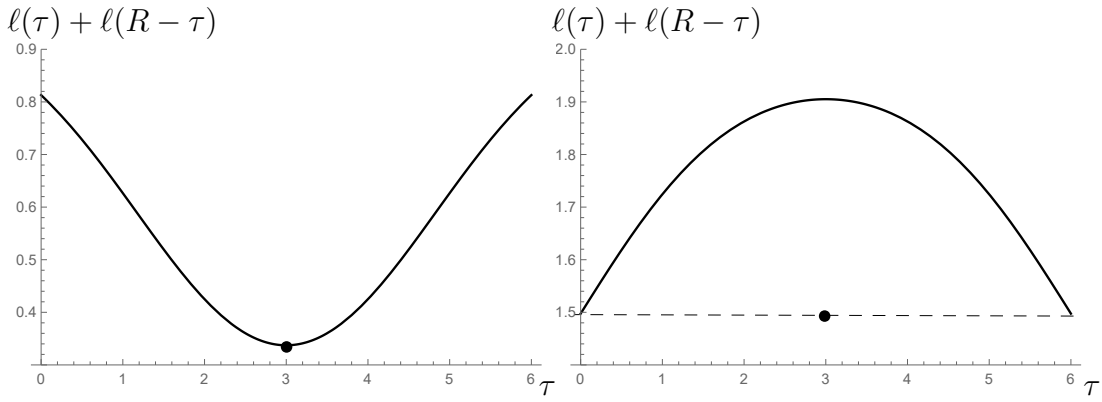

Figure 4: Function $\ell(\tau) + \ell(R-\tau)$ for a large group size on the left-hand side ($K = 100$) and for a small group on the right-hand side ($K = 2$); $\gamma = 1$ and $R = 6$ in both graphs.

1. *If it takes more than half of the reaction time to infect half of the group, $R/2 < b$, then the frequent rotation, $r(0,t) = r(1,t) = \frac{1}{2}$ for all t, is optimal.*

2. *If it takes less than half of the reaction time to infect half of the group, $R/2 > b$, then it is optimal to activate the same group at all times: for either $i = 0$ or $i = 1$, $r(i,t) = 1$ for all t.*

Proof of Proposition 1. When $R/2 < b$, then, the objective in the relaxed rotation problem (3) is U-shaped. By the standard concavification argument, the optimal distribution F that solves problem (3) assigns probability 1 to $\tau = R/2$.[10] This distribution is implementable via the rotation scheme $r(i,t) = 1/2$ for both i and all t.

When $R/2 > b$, then the objective in the relaxed rotation problem is hump-shaped. By the concavification again, F that solves the relaxed rotation problem assigns probability half to $\tau = 0$ and R each. This distribution is implementable via the rotation scheme such that $r(i,t) = 1$ and $r(1-i,t) = 0$ for all t, for one of the two groups i. □

The scheme $r(i,t) = 1$ and $r(1-i,t) = 0$ for all t activates only one of the two groups. When such asymmetric treatment of the two groups is undesirable, this scheme can be approximated by infrequent rotation of both groups. With this in mind, we refer to $r(i,t) = 1$ and $r(1-i,t) = 0$ for all t as to the *infrequent rotation*, and to $r(i,t) = 1/2$ for all t as to the *frequent rotation*.

Let us normalize the parameter γ to $\ln 2$, which is equivalent to setting the unit of time to the doubling time in the early stage of spreading (starting from a low prevalence value p_0,

[10]Since we are solving a minimization problem, and concavification technique, as in Kamenica and Gentzkow (2011), is applied to maximization problems, the concavification must be applied to $-\ell(\tau) - \ell(R-\tau)$.

the growth is initially exponential, $p_t \approx p_0 e^{\gamma t}$). Recalling that $b = \frac{1}{\gamma}\ln(K-1)$, Proposition 1 implies that frequent rotation is optimal for reactions times that do not exceed the multiple $\sqrt{2}\ln(K-1)$ of the doubling time. That is, frequent rotation is optimal if the reaction time does not exceed 0, 3.1, 6.5 and 9.8 multiples of the doubling time for the group size of $K = 2$, 10, 100, and 1000, respectively. In particular, infrequent rotation is optimal for the group size $K = 2$ regardless of the reaction time because half of the population is infected immediately at t^* and thus the logistic growth is concave in the whole relevant period in this case.[11]

Corollary 1. *Frequent rotation is favoured by:*

1. *A short organizational reaction time, i.e., for any $\gamma > 0$ and $K > 2$, frequent rotation is optimal for sufficiently small R;*

2. *A low infectiousness, i.e., for any R and $K > 2$, frequent rotation is optimal for sufficiently low γ;*

3. *A large group's size, i.e., for any $\gamma > 0$ and $R > 0$, frequent rotation is optimal for sufficiently high K.*

That is, when the organization's reaction is fast, the infection spreads slowly, and the group is large, then prevalence is a convex function of the exposure time for most of the relevant interval. Then, frequent rotation optimally eliminates risk in the exposure time.[12]

5 Mixing of the Rotating Groups

We continue to consider two groups, $i = 0, 1$, each with population of measure K. As before, measure one of the individuals from one of the two groups, $i = \omega$, is exogenously infected at a random time t^*, where $\omega = 0$ or 1 with equal probabilities. We let the two groups follow the frequent symmetric rotation scheme, $r(i,t) = 1/2$ for both i and all $t > 0$.

Unlike in the previous sections, the two groups mix. That is, there is a continuous flow of intensity $\alpha > 0$ of individuals from group 0 to group 1 and vice versa. We postulate that

[11] The insight that infrequent rotation of pairs is optimal carries on to a discrete setting. Assume that one out of two individuals is infected initially and that the healthy individual is infected with probability γ in each discrete time period. Then the expected number of infected individuals, $2 - (1-\gamma)^t$, is concave in t.

[12] We have made a tacit assumption that the expected number of contacts of each individual per unit of time does not depend on the group size, K. If the flow of contacts scales up with K, then the effect of the group size on the optimal rotation frequency would become ambiguous. An increase of the group size, K, would have two opposing effects: (i) keeping the speed of spreading constant, it takes longer to saturate the group, but (ii) increase in K would make the spreading of the disease faster, which favours saturation.

the prevalence rates $p_t^i(\omega)$ evolve for this setting in a system of differential equations,

$$\begin{aligned} \dot{p}_t^i(\omega) &= p_t^i(\omega)\left(1-p_t^i(\omega)\right)+\alpha\left(p_t^{1-i}(\omega)-p_t^i(\omega)\right) \text{ for each } i \text{ and } \omega, \\ p_{t^*}^i(\omega) &= 1_{i=\omega}/K, \end{aligned} \tag{5}$$

where, for sake of simplicity, we have set $\gamma = 2$ so that $\gamma r(i,t) = 1$. The second summand of equation (5) captures the fact that, in a short interval of time Δ, a fraction $\alpha\Delta$ of members of the $1-i$ group migrate to group i (and vice-versa) and, therefore, the difference in prevalence across the two groups determines the net effect of mixing on the prevalence in the group i.

We conveniently reformulate the system (5) using a change of variables. Recall that $p_t^i(\omega)$ is the probability that a random member of the group i is infected at t when the initially infected group has been ω. We let

$$\pi_t^i = \frac{1}{2}\sum_{\omega} p_t^i(\omega)$$

be the probability that a random individual from the group i is infected at time t, where this probability is evaluated ex ante before the state ω has realized. Due to symmetry of the system, π_t^i is independent of the group i, and thus we omit the upper index. The total measure of infections across both populations at t is $2K\pi_t$, and therefore π_t is the variable of our interest.

Let

$$c_t^i = \frac{1}{2}\sum_{\omega}\left(p_t^i(\omega)\right)^2 - \left(\pi_t\right)^2$$

be the ex-ante covariance of the health statuses of any two individuals from the group i at time t. Again, due to the symmetry, c_t^i does not depend on i, and thus we omit the upper index. Upon the time of the initial infection, this covariance is positive due to the common infection shock to all the group's individuals. Thereafter, the covariance evolves due to mixing and new infections.

The next result states that (i) growth of the aggregate prevalence decreases with covariance and that (ii) covariance diminishes with mixing of the two groups.

Lemma 4. *The aggregate infection π_t and in-group covariance c_t evolve in the system*

$$\dot{\pi}_t = \pi_t\left(1-\pi_t\right) - c_t \tag{6}$$

$$\dot{c}_t = c_t\left(2-4\pi_t\right) - 4\alpha c_t \tag{7}$$

with the initial condition $\pi_{t^*} = \frac{1}{2K}$ and $c_{t^*} = \frac{1}{4K^2}$.

See Appendix for the proof. Equation (6) implies that within-group covariance lowers the growth rate of the prevalence, ceteris paribus, for any fixed π_t. This is because, keeping the expected infection fixed, covariance lowers the share of healthy-sick contact pairs. The term $-4\alpha c_t$ in equation (7) captures the fact that covariance dissipates with mixing.

Proposition 2. *Mixing increases the aggregate prevalence across both groups. That is, aggregate prevalence* $\pi_t(\alpha) = \sum_i p_t^i(\omega; \alpha)$ *increases in* α *for any* t *and each state* ω*.*

Proof of Proposition 2. It suffices to prove that $\pi_t(\alpha)$ increases in α for each $t > t^*$. Let $\alpha' > \alpha$. It must be that $c_t(\alpha') < c_t(\alpha)$ for all $t > t^*$. To see this, note that $\dot{c}_{t^*}(\alpha) > \dot{c}_{t^*}(\alpha')$, and thus we have that $c_t(\alpha) > c_t(\alpha')$ for a right neighborhood of t^*. For sake of contradiction, consider the lowest time $t^{**} > t^*$ such that $c_t(\alpha') = c_t(\alpha)$. We have that $c_t(\alpha) > c_t(\alpha')$ for all $t \in (t^*, t^{**})$. Thus, $\dot{\pi}_t(\alpha) < \dot{\pi}_t(\alpha')$ for all $t \in (t^*, t^{**})$, and hence $\pi_{t^{**}}(\alpha) < \pi_{t^{**}}(\alpha')$. Thus, $\dot{c}_{t^{**}}(\alpha) > \dot{c}_{t^{**}}(\alpha')$ and therefore $c_t(\alpha)$ intersects $c_t(\alpha')$ from below at t^{**}, which establishes contradiction.

Since $c_t(\alpha') < c_t(\alpha)$ for all $t > t^*$, equation (6) implies that $\pi_t(\alpha')$ cannot intersect $\pi_t(\alpha)$ from above, as needed. □

It is a common intuition that mixing infected groups is harmful. Our explanation that highlights the role of the covariance of the health statuses allows us to assess the adverse effect quantitatively. It turns out that when the terminal prevalence at t^*+R is small without mixing, then the adverse effect of mixing is negligible. To understand this, first, recall that the logistic growth model is well approximated by an exponential growth model and thus,

$$\dot{p}_t^i(\omega) \approx p_t^i(\omega) - \alpha \left(p_t^{1-i}(\omega) - p_t^i(\omega) \right)$$

for each group i and state ω at low prevalence levels. Since this system of differential equations is linear, it holds after we take expectation with respect to ω. Hence, using its symmetry, we get that $\dot{\pi}_t \approx \pi_t$ for the aggregate prevalence irrespectively of the mixing intensity α.

Mixing thus affects prevalence only if the nonlinearities in the logistic growth process become significant. This occurs when the prevalence is high enough so that the saturation effect becomes sizable and infected individuals interact with other infected individuals with notable probabilities. Only in that scenario covariances of health statutes, and the impact of mixing on them, become relevant.

The next result bounds the excess prevalence caused by mixing uniformly across all values of α. Equation (6) implies that prevalence $\pi_t(\alpha)$ is bounded across all α from above by process $\dot{\pi}_t = \pi_t(1 - \pi_t)$ with initial condition $\pi_{t^*} = \frac{1}{2K}$. This process is equivalent to

complete mixing of both groups immediately upon the event of the exogenous infection, and then having the disease spread in both groups. The bound on the excess prevalence in the proposition is obtained by comparing this process with the process that entails no mixing.

Proposition 3. *Excess prevalence* $\pi_{t^*+s}(\alpha)/\pi_{t^*+R}(0)$ *due to mixing of the two groups with intensity* α *is at most*

$$\frac{2K-2}{2K\left(1-\pi_{t^*+R}(0)\right)-1} = 1 + o\left(\pi_{t^*+R}(0) - \frac{1}{2K}\right).$$

Thus, when prevalence $\pi_{t^*+R}(0)$ in absence of the mixing does not significantly exceed the initial level of $\frac{1}{2K}$, then mixing has a negligible impact. The analysis of this session has two important implications. First, the value of frequent rotation, when optimal, is not significantly compromised by possible mixing of the two groups. Second, it suggests that if the decision-maker wants to activate more than half of the organization at each point in time, then frequent rotation with some mixing is still an effective way to contain outbreaks.

6 Discussion

We have imposed a number of simplifying assumptions such as deterministic reaction time and logistic growth process. The result that frequent rotation is optimal when prevalence growth is convex (before the organization reacts) extends to stochastic reaction times. That is, let the reaction time be a random variable, and assume that prevalence is convex on its support. Then, frequent rotation is optimal because any other rotation scheme induces a distribution of the exposure time that is a mean-preserving spread of the distribution induced by the frequent rotation. This holds irrespectively of the distribution of the initial infection time t^* and of the reaction time R since the mean-preserving-spread comparison holds conditional on each realization of t^* and R.

The assumption of the logistic growth has allowed us to characterize optimal rotation frequency even when prevalence growth is both convex and concave within the organization's reaction span. We discuss a minor generalization of our result to highlight the specific properties of the logistic growth that we have used. Let $\dot{p}_t = g(p_t)$ be the growth rate of prevalence as a function of its current level; e.g., $g(p) = p(1-p)$ for the logistic growth. When g is non-negative and hump-shaped on $[0,1]$ and symmetric around $1/2$, then the resulting prevalence p_t is convex in t when $p_t < 1/2$, concave for $p_t > 1/2$ and antisymmetric around the inflexion point. Since these are the properties of $\ell(\tau)$ that we have used to derive Proposition 1, the result extends. Ultimately, we can define $\ell(\tau)$ as the expected prevalence for the exposure time τ and let this be the primitive of the model that can be empirically

estimated for a given class of organizations. We advise for frequent rotation as long as this function is convex within the reaction time.

Finally, the result on adverse effect of mixing is implied by concavity of the growth function g, and the insight that mixing has an only negligible effect when terminal prevalence is low would extend to a general class of concave functions g with bounded second derivative.

A Appendix

Proof of Lemma 4. Equation (6) follows from (5) by taking expectation with respect to ω.

Let us prove (7). For future reference, note from its definition that covariance

$$c_t^i = \frac{1}{4}\left(p_t^i(0) - p_t^i(1)\right)^2. \tag{8}$$

The symmetry of the system implies that $p_t^0(0) = p_t^1(1)$ and $p_t^0(1) = p_t^1(0)$ for each t. Thus, we drop the upper index and write for $p_t(0)$ and $p_t(1)$ for $p_t^0(0)$ and $p_t^0(1)$. System (5) and the symmetry imply

$$\begin{aligned} \dot{p}_t(0) &= p_t(0)(1-p_t(0)) + \alpha\left(p_t(1) - p_t(0)\right) \\ \dot{p}_t(1) &= p_t(1)(1-p_t(1)) + \alpha\left(p_t(0) - p_t(1)\right). \end{aligned}$$

Let us use a transformation of variables. Recall $\pi_t^0 = \frac{p_t(0)+p_t(1)}{2}$, drop the upper index and write π_t. Similarly, let $\Delta_t = \frac{p_t(0)-p_t(1)}{2}$. After the transformation,

$$\begin{aligned} \dot{\pi}_t &= \pi_t(1-\pi_t) - \Delta_t^2 \\ \dot{\Delta}_t &= \Delta_t\left(1 - 2\pi_t - 2\alpha\right). \end{aligned}$$

Since $c_t^0 = c_t = \Delta_t^2$, we have, $\dot{c}_t = 2\Delta_t\dot{\Delta}_t$. This, combined with the last inline equation gives (7). □

Proof of Proposition 3. Let $\ell(t;\pi^*) = \frac{\pi^* e^t}{\pi^* e^t + 1 - \pi^*}$ be the solution to $\dot{\pi}_t = \pi_t(1-\pi_t)$ with the initial condition $\pi_0 = \pi^*$. When there is no mixing, $\alpha = 0$, then prevalence $\pi_{t^*+R}(0)$ evaluated R units of time after the initial infection satisfies $\ell(R;1/K)/2$ since the infection spreads only in the infected group and the initial prevalence in this group equals $1/K$. Equation (7) implies that $c_t(\alpha)$ is nonnegative for all α and all t. Thus, for any α, equation (6) implies that prevalence $\pi_{t^*+R}(\alpha)$ with mixing flow α is at most $\ell(R;1/2K)$. Thus, the excess prevalence $\pi_{t^*+R}(\alpha)/\pi_{t^*+R}(0)$ is at most $\frac{\ell(R;1/2K)}{\ell(R;1/K)/2}$. The proposition follows from expressing $\ell(R;1/2K)$ as a function of $\ell(R;1/K)/2$. □

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