Act Now or Forever Hold Your Peace: Slowing Contagion with Unknown Spreaders, Constrained Cleaning Capacities and Costless Measures

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Abstract

What can be done to slow contagion when unidentified healthy carriers are contagious, total isolation is impossible, cleaning capacities are constrained, contamination parameters and even contamination channels are uncertain? Short answer: reduce variance.

I study mathematical properties of contagion when people may be contaminated by using successively devices, such as restrooms, which have been identified as a potential contamination channel for COVID19. The expected number of exposures (at least one previous user was already contaminated and is thus a "spreader") and new contaminations (which may increase with the number of spreaders among previous users and may also decrease with time) are always convex functions of the number *n* of users. As a direct application of Jensen inequality, contamination can be reduced at no cost by limiting the variance of *n*.

The gains from optimal use and cleaning of the devices can be substantial in this baseline framework: with a 1% proportion of (unknown) contaminated people, cleaning one device after 5 uses and the other after 15 uses increases contamination by 26 % with respect to the optimal organization, which is cleaning each device after 10 uses. The relative gains decrease when the proportion of spreaders increases. Thus, optimal organization is more beneficial at the beginning of an epidemic, providing additional reason for early action during an epidemic (the traditional reason, which is first-order, is that contamination is approximately exponential over the expansion phase of an epidemic).

These convexity results extend only partially to simultaneous use situations, since the exposure function becomes concave above a threshold which decreases with the proportion of spreaders: once again, this calls for early action. Simultaneous use is the framework most often analyzed in the network literature, which may explain why the above convexity results have been overlooked.

When multiple spreaders increase the probability of contamination, the degree of convexity depend on the precise effects of each additional spreader. With linear probabilities, the expected contamination curves are semi-parabolas, both for successive and simultaneous use. For other inverse link functions, convexity is always ensured in the successive use case but must be determined case by case for simultaneous use.

Keywords

Epidemic, Coronavirus, contagion, spreader, silent spreader, healthy carrier, successive use, directed network, asymptomatic transmission, airborne transmission, fomite, half-contamination function, geometric distribution, binomial distribution, convexity, Jensen inequality

JEL Classification

I12, I18, L23, M50

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1. Introduction

Even during deadly epidemics, the functioning of vital institutions (hospitals, systemic public administrations, private firms providing food or medicines and medical devices, NGOs…) require many people to work on-site together.

Some of these people may be sick and likely to contaminate their healthy colleagues without presenting any noticeable symptom: they are unknown spreaders[2]. To what extent healthy carriers spread a disease is an epidemic-specific question (*cf.* § 2.1).

I contribute to analyzing the contamination issue from an organizational perspective: how to use limited resources ("devices" used by many people, and cleaning capacities) optimally? I focus on the case where people may be contaminated if they use successively the same device. Typical devices are restrooms, which have been identified as a potential contamination channel for COVID19 (*cf.* § 2.1), but also access hatches, elevators, etc.

An interesting literature analyzes the steady-state. I rather focus on slowing contagion, which may be a priority when facing deadly epidemics. Encompassing slowing contagion techniques into a wider framework to analyze steady-state consequences is left for future research.

I consider the speed of contagion for given parameters such as the proportion of spreaders and I do not consider dynamic loop effects. More precisely, I focus on the limited framework of single use and I disregard more dynamic interactions. Thus, the focus of the present paper is static; encompassing the results provided here would be an interesting and useful extension.

The optimization techniques identified here contribute to slow contamination with existing means, which is crucial since epidemics often induce a shortage of required equipment[3]. They rely on the convexity of exposures with respect to the number of successive users. This convexity stems from two simple facts. First, when more people use a device, the probability that at least one of them is a spreader increases[4]. Second, the expected number of healthy people exposed if a spreader has used the device also increases.

As a result of this convexity, Jensen inequality induces the simple and general recommendation of minimizing the variance of the number of users, when the average number cannot be reduced. For instance, if it is impossible to clean toilets more often than once every ten users on average, one can at least organize the use and cleaning of restrooms so that each one is cleaned indeed after ten users, rather than one restroom after five users and another after fifteen users. Indeed, due to convexity, the gains from cleaning one restroom sooner (after five rather than ten users) are limited, while the losses from waiting to clean the other restroom (after fifteen rather than ten users) are much higher.

I find that the effects of better organization can be substantial for proportions of spreaders below 2% to 5%, that is over the expansion phase of a major epidemic: the simple measures recommended here must be implemented accordingly.

The convexity results do not generalize to the simultaneous case. Indeed, local concavity happens above a number of users which decreases when the proportion of spreaders increase. Still, convexity is verified when the proportion of spreaders is low, and therefore better organization is beneficial. This provides additional argument for early action.

If the number of spreaders to which a healthy user is exposed affects her probability of contamination, convexity depends on the inverse link function which relates the number of spreader to the conditional

[2] Unknown spreaders are untested contaminated people who do not present and did not present symptoms beforehand. Indeed, for SARS-CoV-2, Chang *et al* (March 27, 2020) find viral load 1 to 8 days after the end of symptoms. Rothe *et al*. (March 5, 2020) present similar observation on one patient.

[3] World Health Organization (Feb. 27, 2020), Centers for Disease Control and Prevention (Feb. 29, 2020)

[4] Note that the expected number of spreaders also increases (see sections 5.2 and 6).

probability of contamination. With linear probabilities, relative effects of better organization are much larger than with the single spreader baseline model.

Quite often, people's individual behavior may naturally implement optimal use. For instance, subway and train users may avoid overcrowded coaches since they would be more likely to be infected there. When they try to limit their individual probability of being infected, they may also limit the number of people that they are likely to contaminate. Overall, this limits the variance of the number of travelers by coach and may limit new contaminations[5]. Unfortunately, for successive use, individuals usually ignore how many people have already used each device, so that they are unable to make optimal individual choices and therefore to reduce overall variance.

Also, quite surprisingly, major organizations seem to disregard convexity issues and the resulting need to prevent crowding, as shown by transportation industry examples during the COVID19 epidemic. For instance, in the United States, the borders closure was announced with short notice and arrivals limited to 13 airports, inducing massive crowdings. In France, the repatriation of more than 240 000 nationals by the major airline in March 2020 was implemented with the objective of filling planes. The 70 % reduction in the number of trains in the Parisian subway by the end of March 2020 made it impossible to maintain the minimal recommended distance between passengers.

The rest of the paper is as follows. Section 2 reviews the literature. Section 3 describes the framework. Section 4 shows that the expected numbers of exposures and new contaminations are always and everywhere convex functions of the number of people using a device successively. Section 5 quantifies the potential gains from convexity for successive use when contamination depends on exposure to (at least) a single spreader. Section 6 provides a similar analysis for simultaneous use and shows local concavity, typically for a high proportion of spreaders and a lot of users. The latter two sections provide a polar case where the potential for concavity is maximal. Conversely, section 7 analyze the linear probability model in which each additional spreader increases linearly the probability of contamination, which is the opposite polar case with the highest potential for convexity. In this framework, convexity holds of course for the successive framework (as is clear from the general results presented in section 4), but also for the simultaneous framework. Section 8 introduce alternative inverse link functions and analyzes the corresponding framework, which may be considered as intermediate between the two polar cases examined just before. Section 9 analyzes robustness and limitations of the results. Section 10 concludes and suggests extensions. A simple extension to a two-types of device framework is provided in an appendix.

2. Previous Literature

2.1 Medical literature

The relevance of the present analysis relates to the transmission of a disease by asymptomatic, presymptomatic, subclinical or only mildly sick patients[6]. In the rest of this article, I use "asymptomatic transmission" to cover all these particular cases. Lipsitch *et al.* (2003) underline that asymptomatic transmission hamper usual control measures. The measures proposed here may therefore be crucial.

Asymptomatic transmission is epidemic-specific and may induce lasting scientific debates (Leung, N. H. L., C. Xu, D. K. M. Ip, and B. J. Cowlin, 2015). Asymptomatic forms of SARS-CoV-1 are documented by Wilders-Smith *et* al. (2005). The potential for asymptomatic transmission is documented for MERS (Omrani A. S. *et al*., 2013**)** and SARS-CoV-2 (Bai Y, L. Yao, T. Wei *et al*., February 21, 2020, Rothe *et al*., March 5, 2020, and Zou L, F. Ruan, M. Huang *et al*., March 19, 2020, Santarpia *et al.*, March 26, 2020).

[5] As noted before, in the simultaneous case, convexity is not general so that reducing variance is not always beneficial. However, I show in sections 6, 7 and 9 that convexity overcomes local concavity for low proportions of spreaders, as well as for many frameworks in which the probability of contaminations depend on the number of spreaders to which an individual is exposed.

[6] Human-to-human transmission of SARS-CoV-2 is identified by Q. Li, X. Guan, P. Wu, X. Wang, B. Cowling, B. Yang, M. Leung, Z. Feng *et al*. (January 31, 2020).

If viruses survive long enough, people can get contaminated by using successively the same device. Doremalen, Bushmaker, Morris *et al.* (March 17, 2020) find that SARS-CoV-1 and SARS-CoV-2 can remain on plastic and steel up to days. The presence of SARS-CoV-2 in toilets (typically used successively) is found by Ong *et al*. (March 4, 2020) and Santarpia *et al.* (March 26, 2020).

Long-range airborne contamination, as opposed to short-range droplets contamination (Tellier *et al.* (2019) changes the meaning and scope of the convexity results presented here: the simultaneous case becomes very relevant and "cleaning" may correspond to a given period without use, such as night. The potential for airborne contamination is reviewed by Tellier *et al.* (2019) and has been found for MERS (Kim S. H., S. Y. Chang, M. Sung *et al*., 2016), SARS-CoV-1 (Booth T. F., B Kournikakis, N. Bastien, J. Ho, J Kobasa, L. Stadnyk *et al.*, 2005) and SARS-CoV-2 (Doremalen *et al.* and Santarpia *et al*., *op. cit.*).

2.2 Public health and mathematical literature

Public health decisions have sometimes proven to be effective in slowing contamination, in combination with medical treatments and testing. For instance, during the COVID19 epidemic, the Chinese lockdown (World Health Organization, February 28, 2020, and Kupferschmidt and Cohen, March 2, 2020) and the South Korean test and trace policy (Normile, March 17, 2020) seem to have been successful at curbing COVID19 contamination by the end of March 2020.Non-phamaceutical interventions may have prevented 21 000 to 120 000 deaths in 11 European countries (Flaxman, S., S. Mishra, A. Gandy, S. Bhatt, N. M. Ferguson *et al.,* March 30, 2020).

Mathematical tools have been used to analyze epidemics since the 18th century (Bernouilli, D., 1766), Dietz K. and J. Heesterbeek, 2002). The literature is based either on probability and combinatory computation (the present analysis is an example), on diffusion processes using for instance partial differential equations, of on the more recent theory of graphs/networks[7].

In the network literature, M. O. Jackson and B. W. Rogers (2007) analyze the effects of mean-preserving spreads degrees distributions, and show that an mean-preserving increase in the dispersion of degrees (similar to the increase in the variance of the number of users in the present paper) increase the average neighbor infection rate[8]. However, the sign of effect of higher dispersion on the average infection rate may depend on the form of the network and, in a family of structures including scale-free and random networks, depends on the speed of contamination.

M. O. Jackson and D. Lopez-Pintado (2013) analyze the effects of homophily, which is the structure where nodes (people) with common characteristics are more likely to have connections. They show that all things being equal, higher homophily allows initial spreading of a disease.

A more specific mathematical literature on curbing epidemics focuses on the optimal use of limited equipment. For instance, sequential dynamic resource allocation can be used to allocate limited medical resources to identified patients and limit contagion under constrained information (Fekom, M., N. Vayatis and A. Kalogeratos, September 9, 2019). The present analysis considers a case where even less information is available since spreaders are unknown.

Although the convexity results are rather robust to changes in the proportion of unknown contaminated people, this proportion is relevant to assess the quantitative effect of the organizational improvements proposed in the present paper, and is crucial to implement the optimal cleaning allocation over different types of devices. The literature has rather focused on forecasting the number of symptomatic patients, since this information is directly relevant for the health system in an epidemic (Alvarez, L., March 28, 2020, and Flaxman, S., S. Mishra, A. Gandy, S. Bhatt, N. M. Ferguson *et al.*, March 30, 2020)[9]. Still, using basic assumptions, it is possible to derive the number of asymptomatic patients.

[7] 'graph' is used by mathematicians, 'network' is used by social scientists and computer scientists

[8] Note that Jackson and Rogers use "contamination rate" for the speed of contamination, while I use this expression for the proportion of contaminated people in the whole population.

[9]. Websites providing forecasts include Alvarez: https://sites.google.com/site/luisalvarezsite/covid-19-italy-france and Verrardi : http://homepages.ulb.ac.be/~vverardi/COVID_19.html

3. Framework

I consider all healthy people to be susceptible to infection. Extension to the SIR case where healed patients are immune is straightforward and is detailed in section 9.2.

Contamination happens through the successive use of a device (a "device" can be a restroom, a coffee machine, an access hatch…) by both unidentified sick and healthy people. A large number of people use a limited number of devices. This large number of people makes sampling with replacement offers a decent modelling approximation. *n* is the average number of people using a device between two full cleanings of the device. I neglect integer part problems. If *n*=1, the contamination risk is null, so I focus on cases with *n*>1. Cleaning constraints, measured by *n*, decrease with cleaning capacities and increase with the number of people using the device.

Full cleaning is the cleaning intervention that breaks with probability one any chain of contamination from a spreader to a healthy person. If the device can be fully cleaned between each use, no contamination happens. I focus on the realistic case where such systematic full cleaning is not possible.

If full cleaning is impossible, convexity results may remain relevant but their interpretation is modified. For instance, in case of aerosol contamination, many institutions are unable to clean the air in closed rooms. Still, if the virus aerosol half-life is low enough, so that one night may constitute a natural full cleaning of the air, then the convexity results may apply to the number of people entering a room over one day[10].

The population is divided between spreaders and healthy users. Spreaders are all people who are already contaminated; they are unknown, because otherwise, they would be isolated and cured for their own protection and the protection of their colleagues. Healthy users are susceptible to infection. Extension to the SIR-like case where healed people are immune is straightforward and is discussed in section 9.

α is the exogenous proportion of unknown spreaders in the population using a device. α can vary over time, but I neglect dynamic aspects.

β is the conditional probability for a healthy person to get contaminated, provided that a contaminated person has used the device since the last cleaning:

$$[1] \quad \beta = P(\,x\ gets\ contaminated \mid x\ is\ healthy,\ \ at\ least\ 1\ spreader\ has\ used\ the\ device\,)$$

In many cases, β can be reduced by individual and collective hygienic behaviors. However, in the case of aerosol contamination, reducing β is more difficult. In section 5.1, I analyze the simultaneous use of a room (office, production line…). Then the definition of β is modified accordingly:

$$[2] \quad \beta = P(\,x\ gets\ contaminated \mid x\ is\ healthy,\ \ at\ least\ 1\ spreader\ shares\ the\ room\,)$$

In sections 5 and 6, I assume that the number of spreaders has no effect on the conditional probability of contamination. Still, in sections 7 to 9, I consider alternative processes in which each additional spreader increases linearly the probability of contamination, in line with the literature. Then, β is :

$$[3] \quad \beta = P(\,x\ gets\ contaminated \mid x\ is\ healthy,\ \ 1\ more\ spreader\ has\ used\ the\ device\,)$$

$$[4] \quad \beta = P(\,x\ gets\ contaminated \mid x\ is\ healthy,\ \ 1\ more\ spreader\ shares\ the\ room\,)$$

I consider here the static case where each user uses a device only once, which extends simply to the simultaneous case. I neglect the effect of time between users. In the real world, an increase in the time-span between the contamination of a device and its use by a healthy person is likely to affect the probability of contamination of this person. The different assumptions in my framework corresponds

[10] Reminder: half-life on plastic or stainless steel is higher than aerosol half-life for SARS-CoV-2 so that a natural cleaning of the air does not ensure a similar cleaning of these surfaces.

to intensive use of a device, so that this assumption is a decent approximation in the case of a long or infinitesimal half-life, but is unrealistic otherwise[11].

This framework can be interpreted in network models as follows. Graph 1 illustrates the successive use case. First step is the exogenous stochastic contamination of all users outside of the process analyzed here. For instance, workers can get contaminated at home (children, friends) or in the transportation system when coming to work, and so on. Each worker gets contaminated with probability α.

Second step is the potential contamination analyzed in the present study. In the left panel, workers 1 to 4 use the same device, and workers 5 and 6 use another device. Worker 1 can contaminate users 2 to 4, user 2 can contaminate user 3 and 4, and user 3 can contaminate user 4, who cannot contaminate anyone because she is the last user. User 5 can contaminate user 6, who cannot contaminate anyone. The main result of the present study is that the network displayed in the right panel will always decrease expected exposure and contamination

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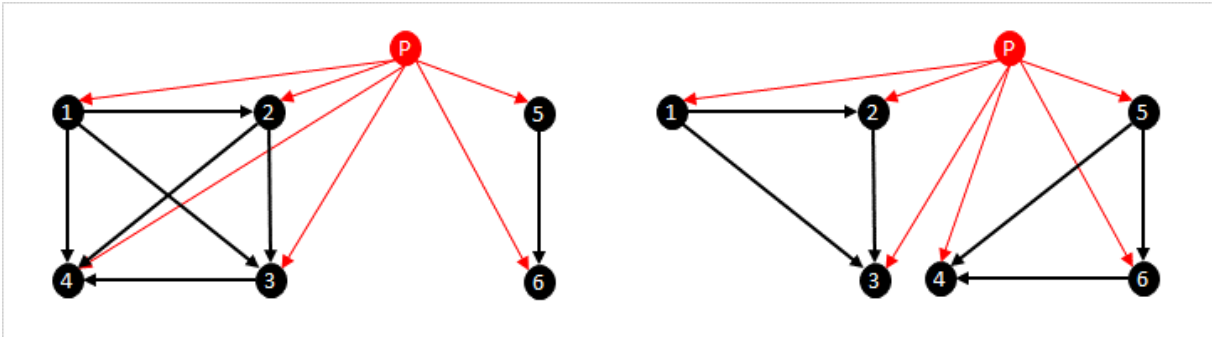
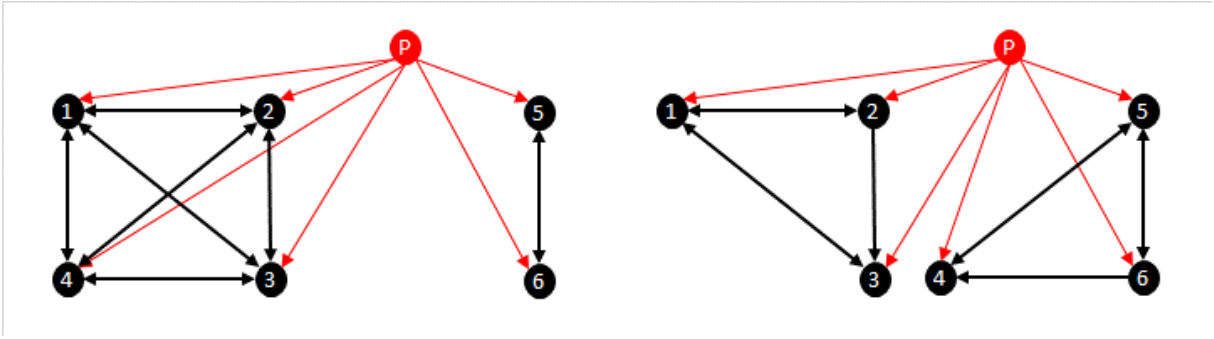


Graph 1: network representation of successive use - unequal use (left) and equal use (right)

Graph 2 displays the simultaneous case, which is analyzed in most of the literature. General theoretical results presented in section 4 do not generalize to this framework, which may explain why they have been overlooked. Still, in many practical case, "better organization" described by the right panel is beneficial.



Graph 2: network representation of simultaneous use - unequal use (left) and equal use (right)

I use "exposure" or "single-spreader" for frameworks where contamination depends on at least one (previous or simultaneous) spreader (sections 5 and 6). I allow for additional effects of additional spreaders in sections 7 to 9.

[11] A simple example may clarify these points. Assume a device is used by many people so that the no-use time between two people is 10 minutes. If the half-time is one minute, the number of viruses will have been divided by 1024 when the next person comes in. And if the half-time is very long, waiting a bit will make negligible difference. Conversely, if the half-time is similar to the no-use time, then waiting may make a big difference. According to Doremalen, Bushmaker, Morris *et al.* (*op. cit.*), the aerosol half-life of SARS-CoV-2 is 1.1 to 1.2 hours and the time between two users may matter. Whether in normal conditions, the virus will stay aerosol in a closed room or fall rapidly to the round is, to the best of my knowledge, an open question at the time of writing.

4. General convexity results for successive use

Theorem 1:

In the successive use framework, if the *ex ante* probability of contamination is the same for all users, the exposure function, which measures the number of healthy (susceptible) users exposed to contamination by at least one contaminated person, is convex.

Proof:

(1) In the successive use framework, additional exposure due to an additional user is the probability of exposure of this user if she is healthy. This probability is the probability that at least one previous user was already contaminated.

(2) The probability that a given user is already contaminated is strictly positive but strictly lower than one, thus the probability that at least one previous user is contaminated is strictly increasing in the number of previous users.

Combining (1) and (2), and given that the probability that the new user is healthy is constant, the additional exposure due to an additional user is strictly increasing with the number of previous users, i.e. the exposure function is convex. □

Theorem 2:

In the successive use framework, if the *ex ante* probability of contamination is the same for all users, the expected contamination function, which measures the expected number of newly contaminated people depending on the number of already contaminated users, is convex.

Proof:

(1) In the successive use framework, the expected contamination due to an additional user is the probability of contamination of this user if she is healthy. This probability is an increasing function of the number of previous users that were already contaminated.

(2) Since the *ex ante* probability that each user is sick is strictly positive, the expected number of spreaders among previous users is a strictly increasing function of the number of previous users.

Combining (1) and (2), and given that the probability that the new user is healthy is constant, the additional probability of contamination due to an additional user is strictly increasing with the number of previous users, i.e. the expected contamination function is convex. □

Theorem 3: In the successive use framework, if the *ex ante* probability of contamination is the same for all users, if the effect of a spreader on the probability of contamination of a subsequent user decrease with the time between them in a way that is consistent over time, then the probability of contamination function is convex.

Note that an effect that is decreasing in the time interval between a spreader and a healthy user in a way that is consistent over time typically covers any exponential decay framework.

Proof:

(1) In the successive use framework, the expected contamination due to an additional user is the probability of contamination of this user if she is healthy. This probability is an increasing function of the number of previous users that were already contaminated.

(2) Since the decreasing effect due to the time interval between a spreader and a healthy user is consistent over time, then in expectation, the cumulated effects of potential spreaders 1 to n-1 on user n is the same as the cumulated effect of potential spreaders 2 to n on user n+1. However, user n+1 is also affected by the potential additional effect of user 1 in case user 1 is a spreader.

Combining (1) and (2), and given that the probabilities that users n and n+1 are healthy are equal, the additional probability of contamination of user n+1 is higher than the additional probability of contamination of user n, i.e. the expected contamination function is convex. □

Convexity of expected exposures and new contaminations with respect to *n* reflects two facts: (1) if many people use the same device, the probability that at least one of them is contaminated increases, (2) additionally, the number of people exposed to contamination obviously increases with the number of people using the device.

Corollary 1:

In successive use frameworks with identical devices, if the same number of people uses each device between to full cleanings, then expected exposures and contamination are reduced with respect to cases of heterogeneous use with the same average number of users.

Proof:

Let $E[c_n]$ be the expected number of exposures or new contaminations when n people use a device between two full cleanings. We can write $f(n) = E[c_n]$. Since, $f(n)$ is convex, Jensen inequality writes: $f(E(n)) < E((f(n))$. We define as n the average number of people that use a device between two cleanings. n* derives directly from exogenous parameters such as the number of users and the cleaning capacities; thus, n* can be considered as fixed exogenously. We get:*

$$[5] \qquad f(n^*) < E[f(n)] = E\big[E[c_n]\big] = E[c_n] \text{ □}$$

That is, we can minimize the expected number of contaminated people by ensuring that each device is used by exactly *n** people between two cleanings (no more and no less), where n* is the average number of people using each device between two cleanings. Empirically, the effect of small deviations (*e.g.* +/- 1) is small except if *n** is also very small. Conversely, larger deviations have substantial effects.

Theorems 1 to 3 and corollary 1 do not generalize to the simultaneous use framework, and I provide counterexamples in sections 6 (contamination by a single spreader case) and 7.2 (multiple spreaders with increasing probability of contamination), although in practice convexity matters in many cases also for simultaneous sue so that "better organization" reducing the variance of the number of users makes it possible to slow contamination down. Successive use corresponds to the directed network framework, while simultaneous use corresponds to the undirected network framework which is analyzed in most of the literature. This may explain why, to the best of my knowledge, the general convexity results detailed in theorems 1 to 3 have not been identified yet.

5. Successive use with contamination by a single spreader

Convexity is a general feature of exposures and expected contamination function in the successive use case. Still, the relevance of organizational measures aimed at reducing the variance of the number of users depends on the degree of convexity. In the present section, I tackle this issue for contamination by (at least) one spreader, *i.e.* when additional spreaders do not increase the probability of contamination. I show that the gains from better organization can be substantial, especially when the proportion of spreaders in the population is low.

5.1 The convexity of the exposure function

The first person who uses the device after a full cleaning cannot be contaminated. Provided that this person is sick, which happens with probability α, and the second user is healthy, which happens with probability (1-α), this second user can be contaminated with probability β. Thus the unconditional probability of contamination of the second user is $\beta(1-\alpha)\alpha$. Similarly, the probability that the third user is contaminated is $\beta(1-\alpha)(1-(1-\alpha)^2)$, and so on. The expected number of contaminated people when exactly *n* people use the same device between two full cleanings is:

[6] $$E[c_n] = \beta(1-\alpha)\sum_{i=2}^{n}\left(1-(1-\alpha)^{i-1}\right)$$

where β is defined by [1]. With $r = 1-\alpha$, [5] simplifies to:

[7] $$E[c_n] = \beta r\left(n-1+r\frac{r^{n-1}-1}{\alpha}\right)$$

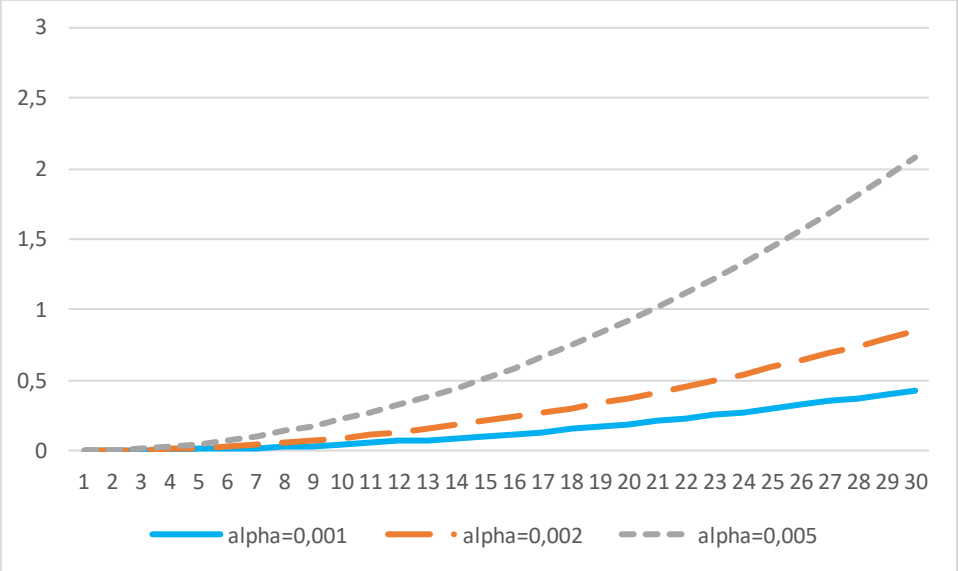
which is clearly increasing in n[12]. For n>=2, [5] induces:

[8] $$E[c_n] - E[c_{n-1}] = \beta(1-\alpha)(1-(1-\alpha)^{n-1})$$

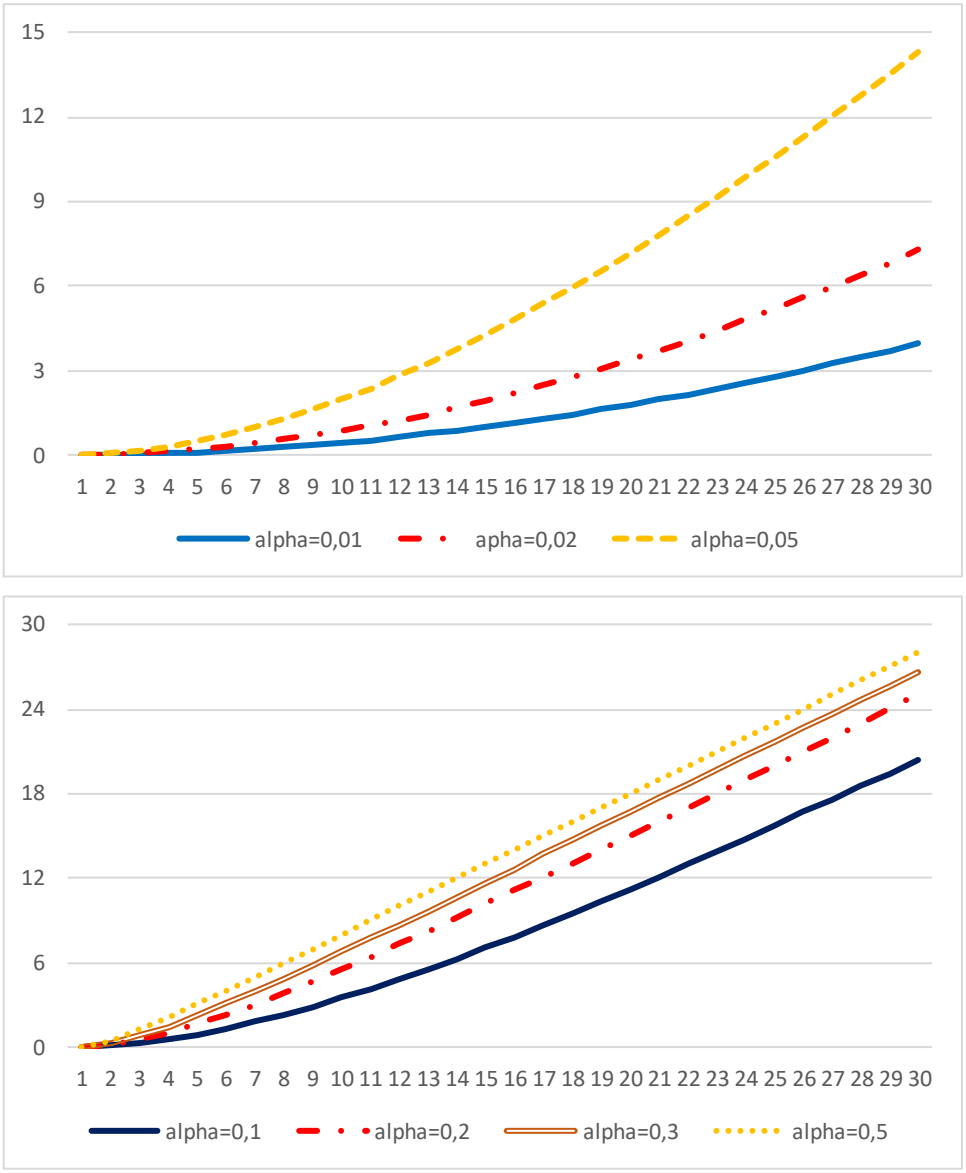
which is also increasing in n so that $E[c_n]$ is a strictly convex function of n.

In [6], the sum over n is the average number of people exposed to contamination because they use a device after at least one spreader. Graph 3 displays this component, which depends only on α and n. $\beta(1-\alpha)$ is merely a scaling constant determining the conditional probability that a person using a contaminated device gets contaminated indeed.

Convexity is clear for the lowest values of α (upper graph). It is less visible for higher values (lower graph), partially as an effect of the change in scale, but also and more fundamentally because for higher values of α, the probability that at least one user is a spreader increases very fast. As soon as it is close to one, the number of exposures increases almost one-to-one (and thus linearly) with the number of users, so that there is less to gain locally over convexity. For α=0.3, it takes only 5 first users for the probability of device contamination to exceed 0.8, and it takes only seven users for α=0.2.



[12] I define static use as the single use of a device by each person. Given the assumptions, the fact that a spreader uses a device repeatedly between two cleanings is not relevant. Conversely, if a healthy person uses a device between two cleanings, she may be contaminated the first time and thus cannot be "re-contaminated" afterwards. However, if α and β are small, the probability that the individual is contaminated the first time is small, especially because if she has time to use the device twice between two cleanings, then it is likely that the first time happens "at the beginning of the queue", *i.e.* just after a cleaning, when the probability of contamination is low. Thus I can neglect this aspect by approximation.



Graph 3: expected number of exposures to a contaminated device, depending on the number of uses between two full cleanings and the proportion of unknown contaminated users (successive use)

Thus, the gains from better organization of the use and cleaning of a device are greater when the proportion of spreaders α is low, which is typically the case at the beginning of an epidemic.

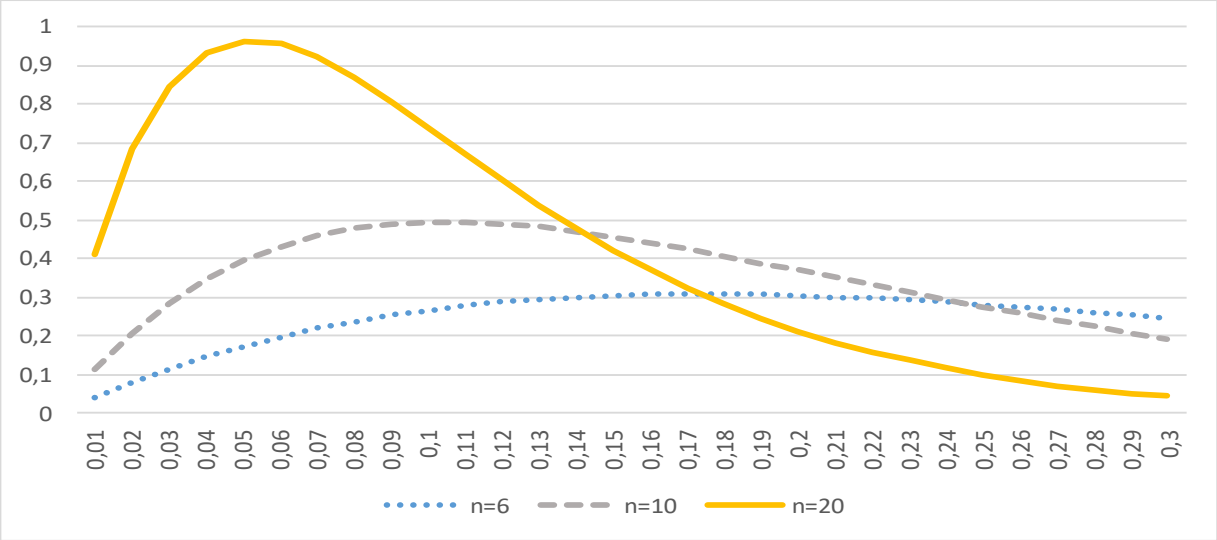
5.2 Relative and absolute gains from optimal use and cleaning

Table 1: relative increase in exposure due to a mean-preserving deviation of $n/2$

α \ n	6	10	20
0.001	29,9%	27,6%	26,0%
0.002	29,8%	27,4%	25,6%
0.005	29,5%	26,9%	24,7%
0.01	28,9%	26,1%	23,1%
0.02	27,9%	24,4%	20,2%
0.05	24,8%	20,1%	13,4%
0.1	20,4%	14,2%	6,6%

0.2	13,2%	6,7%	1,4%
0.3	8,1%	2,9%	0,3%

Table 1 displays the relative increase in exposures due to suboptimal organization for different values of α for three examples. The idea is to get the increase in contamination for exogenously given number of people, number of devices, and cleaning capacities. I compare the optimal case (n users use the device between two cleanings) to a mean-preserving deviation in which one device is cleaned after $n/2$ and another device is cleaned after $3n/2$ uses. I present the increase in exposures due to this deviation. The overall picture is that the decrease in exposure from better organization is high for small α. When α exceeds 2%, the relative effect decreases strongly.



Graph 4: absolute increase in exposure due to a mean-preserving deviation of $n/2$, depending on α

Graph 4 displays the absolute additional exposures due to mean-preserving deviations. The x-axis is the proportion of unknown spreaders in the population (α). The y-axis is the absolute increase in the number of contamination due to bad organization. Each curve corresponds to the absolute additional exposure induced by a mean-preserving deviation of +/- n/2. For instance, the solid yellow line corresponds to the case n^*=20; for a proportion of unknown spreader equal to 0.01, cleaning one restroom after ten users and the other after thirty users increases the expected number of healthy users by 0.4, when compared to cleaning both restrooms after twenty users.

The effect of optimal organization reaches a peak for low values of α, especially if the cleaning capacity is limited (high n). For n=20, the mean-preserving deviation has maximal effect (0.964 exposure) for α=0.05[13].

Such threshold is well below any "collective immunity" threshold. Second, α can only be estimated with a large confidence interval, using for instance large-scale tests, or using the number of deaths, severe cases or symptomatic cases combined with assumptions of the speed of propagation. Third, the rate of contamination during the expansion phase of an epidemic is approximately exponential. Fourth, the peak happens for lower values of α when n is higher. High n corresponds to stringent constraints on cleaning and may correspond to many realistic cases.

Finally, these results are encouraging and show the possibility of slowing contamination down at no cost (except administrative costs) over the first phase of an epidemic. Still, whenever possible, cleaning constraints must also be reduced, *e.g.* by reducing the number of workers, by increasing cleaning

[13] At the time of completing this article, a study published by Imperial College (Flaxman, S., S. Mishra, A. Gandy, S. Bhatt, N. M. Ferguson *et al.*, March 30, 2020)) evaluates α=0.03 by end March 2020 in France.

capacities[14], or, if the cleaning calendar is fixed by nature (*e.g.* every night for aerosol contamination) by increasing the number of available devices[15].

6. Simultaneous use

The baseline case (uniform exposure if at least one user is a spreader) can be extended to the case of simultaneous use. If *n* people work in the same office, the expected number of new contaminations is:

$$[14] \qquad E[c_n] = \beta \sum_{i=1}^{n-1} i.\, C_n^i (1-\alpha)^i\, \alpha^{n-i}$$

where *β* is defined by equation [2], $C_n^i = \frac{n!}{i!(n-i)!}$, and for each piece of the sum, *i* is the number of healthy people susceptible to contamination, and *n-i* is the number of spreaders. Note that for ease of computation, the number of spreaders and healthy users (*i* and *n-i* in section 5) are inverted in the present section.*α* is the same as before (proportion of unknown spreaders) while *β* is now the probability of contamination of a healthy person sharing a room, over a given time interval, as defined by equation [2].

The sum over *i* is, *modulo* the last missing term in *n*, the computation of the expected value of the binomial distribution *B*(*n*,1-*α*), which is equal to *n*(1- *α*). Thus

$$[15] \qquad E[c_n] = \beta[n(1-\alpha) - n(1-\alpha)^n]$$

$E[c_n] - E[c_{n-1}]$ can be computed from [15]. Another method is to use direct combinatory reasoning. When an additional person *n* works in a room, additional contamination may happen either if person *n* is a spreader while none of the other users is, or if at least one of the other user is a spreader and person *n* is not. Thus:

$$[16] \qquad E[c_n] - E[c_{n-1}] = \beta.[\propto (n-1).(1-\propto)^{n-1} + (1-\propto).(1-(1-\propto)^{n-1})]$$

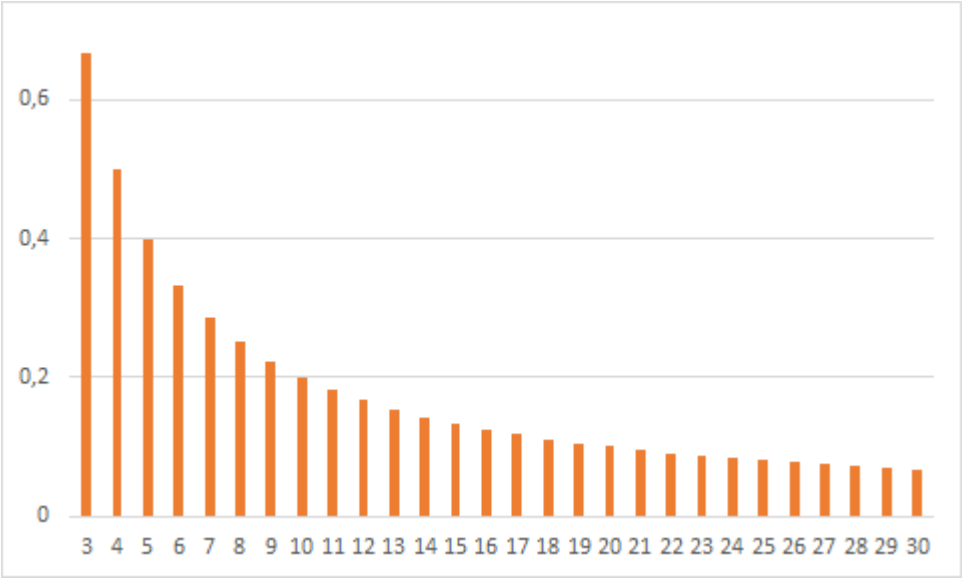
[16] is not always increasing in *n* and is actually decreasing for high values of *α* and *n* Thus, [14] is not convex. The intuition is the following, from equation [16]: when *α* is high enough, the probability that none of the first *n*-1 users is a spreader (*i.e.* $(1-\alpha)^{n-1}$) decreases exponentionally with the number of users, which eventually more than counteracts the increasing potential effect of an infected newcomer on *n*-1 incumbents. Mathematically, $(n-1).(1-\propto)^{n-1}$ decreases with respect to *n* when *α* and *n* are large enough. Since the other part of the bracket converge to 1-*α* (if 0<*α*<1), we get:

Theorem 4: $For\ simultaneous\ use\ , \forall \alpha, \exists \hat{n}\ (\alpha)\ such\ that\ \forall n \geq \hat{n}\ (\alpha), E[c_n]\ is\ convex\ in\ n$

Graph 5 displays the value of *α* as a function of $\hat{n}\ (2 \leq \hat{n} \leq 30)$. When *α* is higher, $E(c_n)$ becomes concave for lower *n*. Note that $E(c_n)$ cannot be concave for *n*<3. Indeed, $E(c_n)$ is null for *n*=0 or 1, and strictly positive for *n*=2. For *α*=2/3, concavity of the number of exposure is verified for all *n* higher than 3. For *α* =0.1, concavity is verified for all *n* higher than 10.

[14] For instance, for *α*=0.05, the absolute gain due to the optimal organization is 0.96. but for the same value of *α*, the gain from loosening the cleaning constraint and reducing *n* from 20 to 18 reduces absolute exposure by 1.23. Once again, it is not always possible to reduce cleaning constraints.

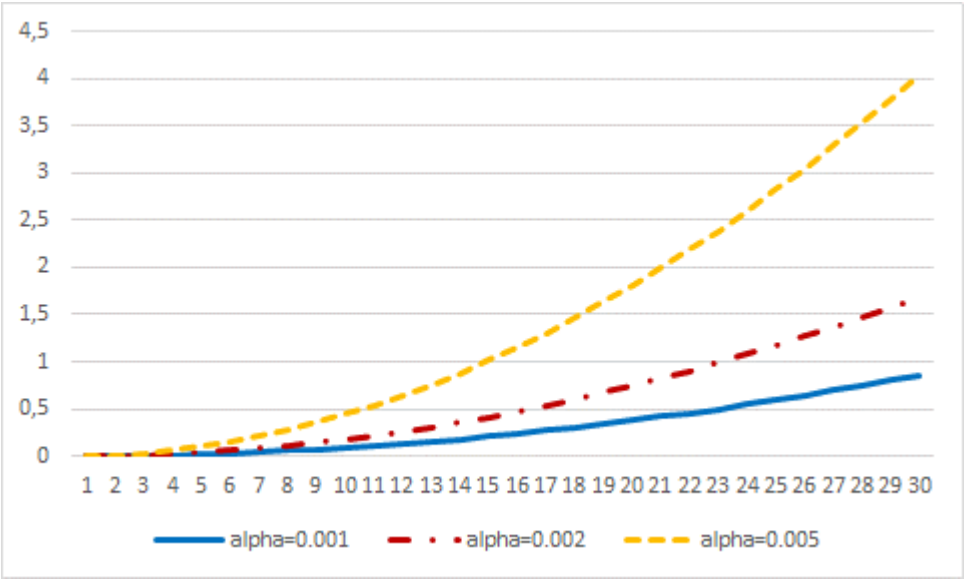
[15] Unfortunately, the latter result is not trivial and is disregarded in many occasions. For instance, during the COVID19 epidemic, many anecdotal examples in large organizations show a concentration of cleanings on a reduced number of still available restrooms (while many restrooms are closed). This organization is innocuous if contamination happens through fomites, but very detrimental in case of airborne contamination.

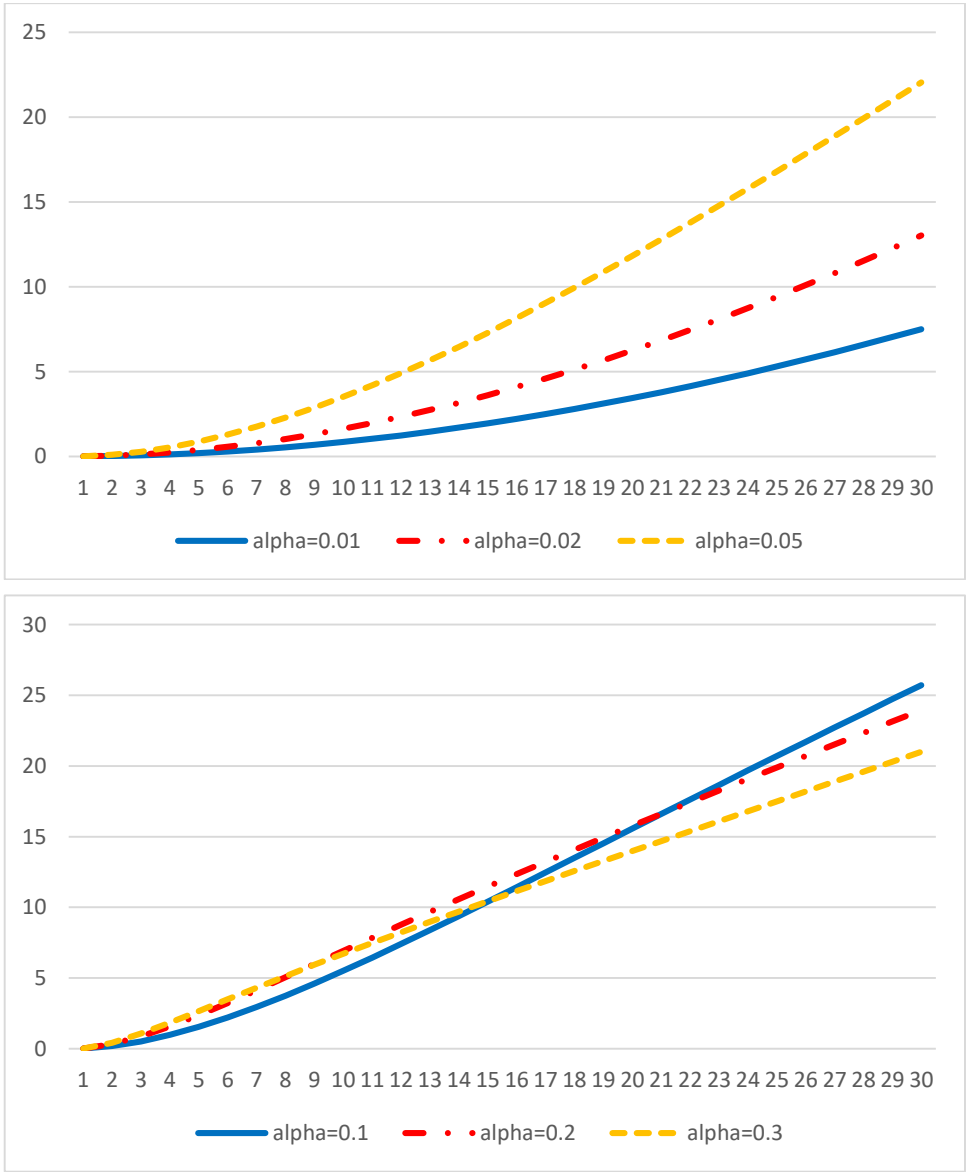


Graph 5: values of α determining the concavity threshold $\hat{n}$

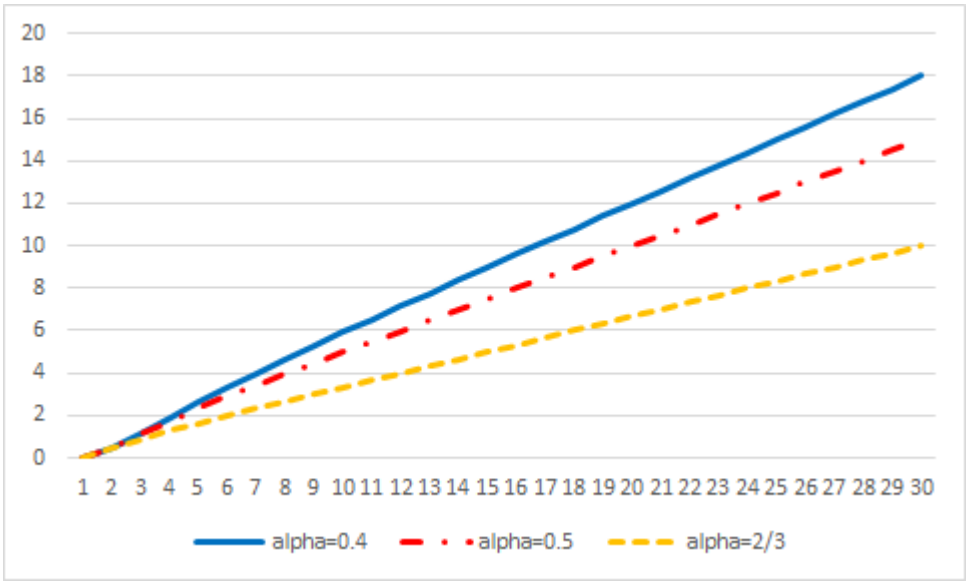
Thus, in this framework, reducing variance is only efficient for low values of α and n. Whether reducing variance can be detrimental (rather than only inefficient) depends on the degree of concavity.

Graph 6 displays the exposure curves for the simultaneous case.





Graph 6: expected number of exposures to a contaminated device, depending on the number of users and the proportion of unknown contaminated users (simultaneous use)



Graph 6 (continued)

The curves crossing in the graph for α equal to 0.1, 0.2 and 0.5 reflects that as α increases, a higher proportion of people are already sick and thus cannot be contaminated.

Even for high values of α, concavity remains limited, and for large n, the curves converge to affine functions so that there is not much to lose or gain locally from concavity. This is why the negative values in Table 3 are low in absolute terms, especially for intermediate values of n (that is, for the same deviations as those presented in Table 1).

Table 3: relative increase in the number of expected exposures due to mean-preserving deviations

	Small *n*				**Intermediate *n***			**global**
α	*3 +/- 1*	*3 +/- 2*	*4 +/- 1*	*4 +/- 2*	*6 +/- 3*	*10 +/- 5*	*20 +/- 10*	***10 +/- 9***
0.001	16.6%	66.5%	8.3%	33.2%	29.8%	27.5%	25.8%	**89.1%**
0.002	16.6%	66.3%	8.3%	33.1%	29.7%	27.3%	25.3%	**88.3%**
0.005	16.5%	65.8%	8.2%	32.8%	29.2%	26.5%	23.8%	**85.8%**
0.01	16.3%	65.0%	8.0%	32.2%	28.4%	25.2%	21.5%	**81.8%**
0.02	15.8%	63.4%	7.8%	31.0%	26.8%	22.8%	17.4%	**74.2%**
0.05	14.6%	58.5%	6.9%	27.7%	22.4%	16.5%	8.1%	**54.9%**
0.1	12.6%	50.8%	5.6%	22.6%	15.9%	8.5%	0.3%	**31.8%**
0.2	8.9%	36.7%	3.3%	13.6%	6.2%	-0.1%	-2.1%	**7.8%**
0.3	5.5%	24.2%	1.4%	6.4%	0.3%	-2.6%	-0.9%	**-1.2%**
0.4	2.5%	13.3%	0.0%	1.0%	-2.7%	-2.3%	-0.2%	**-4.0%**
0.5	0.0%	4.2%	-0.9%	-2.7%	-3.5%	-1.4%	0.0%	**-4.8%**
2/3	-2.8%	-7.4%	-1.3%	-5.1%	-2.4%	-0.3%	0.0%	**-5.0%**

Closer examination of the exposures formulas and curves reveals two facts. First, for high α, concavity occurs mainly for very small n, since for larger n the exposure curves converge very fast toward affine functions. Second, for high α, the slope of the latter affine functions is rather flat. The combination of initial concavity and subsequent flat slope provides scope for global concavity.

As regards initial concavity, I compute small deviations for small n (left panel of table 3). For very high α, local concavity can be meaningful. For instance, having one person in a room and five in another reduces exposures by 7.4% with respect to the three/three organization. Still, the reverse holds, and with much larger difference, when α is lower than 0.4.

As regards global concavity, I compute large deviations presented in the last column of table 3. Concavity can be meaningful: having one person in a room and 19 in another reduces exposure by 5% if α=2/3. Note that this result only means that all people in the "big" room are exposed for sure, while the lonely one is not. Still, the reverse holds for α lower than 0.2, with larger benefits for equal distribution. Gains can be substantial for small n and small changes in n. Note that in the simultaneous use case, implementing precise small changes in n is realistic in many settings.

As a conclusion, although local and global concavity exists for simultaneous use with high values of α, this concavity is much less meaningful than the convexity observed for lower α. Overall, and except if α is known to be very high, reducing the variance of simultaneous occupation of offices and other rooms is beneficial. Still, concavity above a threshold which decreases with α provides further argument for early organizational action during an epidemic.

7. Additional effect of multiple spreaders: the linear probability case

The exposure framework (one spreader suffices and additional spreaders do not increase the probability of contamination) provides a simple mathematical polar case. Still, the literature often links exposure to many spreaders with a higher probability of contamination.

7.1 Successive use when the probability of contamination increases with the number of spreaders

I have considered the case where the probability of contagion depends only on the fact that at least one previous user is already contaminated. But the number of users may also have an effect. If $\beta \leq {}^{1}/_{n-1}$, the conditional contamination rate can be approximated with the linear probability model in which each spreader increases linearly the probability of contamination. In this case, for 1<=*i*<*n*, user *i*+1 is exposed to a number of previous spreaders which is:

[11] $$E[\#\ spreaders(i)] = \sum_{j=1}^{i} j.C_i^j \alpha^j (1-\alpha)^{i-j}$$

Where $C_i^j = \frac{i!}{j!(i-j)!}$. Thus, the expected number of new contaminations when *n* users use successively a device is:

[12] $$E[c_n] = \beta(1-\alpha)\sum_{i=1}^{n-1}\sum_{j=1}^{i} j.C_i^j \alpha^j (1-\alpha)^{i-j}$$

Where *β* is defined by equation [3]. The sum over *j* is the computation of the expected value of the binomial distribution B(*i*,*α*), which is equal to *iα*, so that:

[13] $$E[c_n] = \beta(1-\alpha)\sum_{i=1}^{n-1} i\alpha$$

$$= \beta\alpha(1-\alpha)\frac{(n-1)(n-2)}{2}$$

Thus [13] is a parabola, is convex and actually more convex than [5] for large realistic values of *n*. This provides additional argument to decrease the variance of *n*. Of course, decreasing the expected *n* remains a first best, whenever possible.

Table 4: relative increase in exposure due to a mean-preserving deviation of *n*/2

n	6	10	20
	45.0%	34.7%	29.2%

Note that with linear probabilities, the relative gains do not depend upon *α*. However, linear probabilities are considered here for tractability. If *α* is high, the number of spreaders increases quickly with *n* so that, unless *β* is arbitrarily low, linear probabilities quickly sum above one. With *logit* or *probit* probabilities, which may be more realistic in many setups, the relative effects may be slightly lower, but are likely to depend on *α*, as in the baseline case presented above.

7.2 Simultaneous use with linear probability

The risk of contamination is likely to increase with the simultaneous number of spreaders in a room. If $\beta \leq {}^{1}/_{n-1}$, the risk of contamination can be approximated with the linear probability model in which each spreader increases linearly the probability of contamination:

[17] $$E[c_n] = \beta\sum_{i=1}^{n-1} i.(n-i)C_n^i(1-\alpha)^i\,\alpha^{n-i}$$

$$= \beta\sum_{i=1}^{n} i.(n-i)C_n^i(1-\alpha)^i\,\alpha^{n-i}$$

$$= \beta n \sum_{i=1}^{n} i.C_n^i(1-\alpha)^i\alpha^{n-i} - \beta \sum_{i=1}^{n} i^2 C_n^i(1-\alpha)^i\alpha^{n-i}$$

where β is defined by equation [4]. The two sums correspond to the first and second moments of a binomial distribution $B(n, 1-\alpha)$. Thus:

$$E[c_n] = \beta n^2(1-\alpha) - \beta[n\alpha(1-\alpha) + n^2(1-\alpha)^2]$$

which, after a series of simplifications, gives

$$[18] \qquad E[c_n] = \beta\alpha(1-\alpha)n(n-1)$$

and therefore:

$$[19] \qquad E[c_n] - E[c_{n-1}] = 2\beta\alpha(1-\alpha)(n-1)$$

[19] is increasing in *n* so that [18] is convex (which is also directly noticeable). Note that [19] can be obtained by direct combinatory reasoning. Over a sample of *n*-1 users, on average there are (*n*-1)(1-α) healthy people, and the n^{th} user is a spreader with probability α, so that with the linear probability model, the increase in contamination over the *n*-1 first users due to the arrival of user *n* is $\beta\alpha(1-\alpha)(n-1)$. Furthermore, over the first *n*-1 users, the expected number of spreaders is (*n*-1)α and the n^{th} is healthy with probability (1-α) so that he gets contaminated with probability $\beta\alpha(1-\alpha)(n-1)$. The addition of both effects provides equation [19].

8. Multiple spreaders with alternative inverse link functions

8.1 Inverse link functions

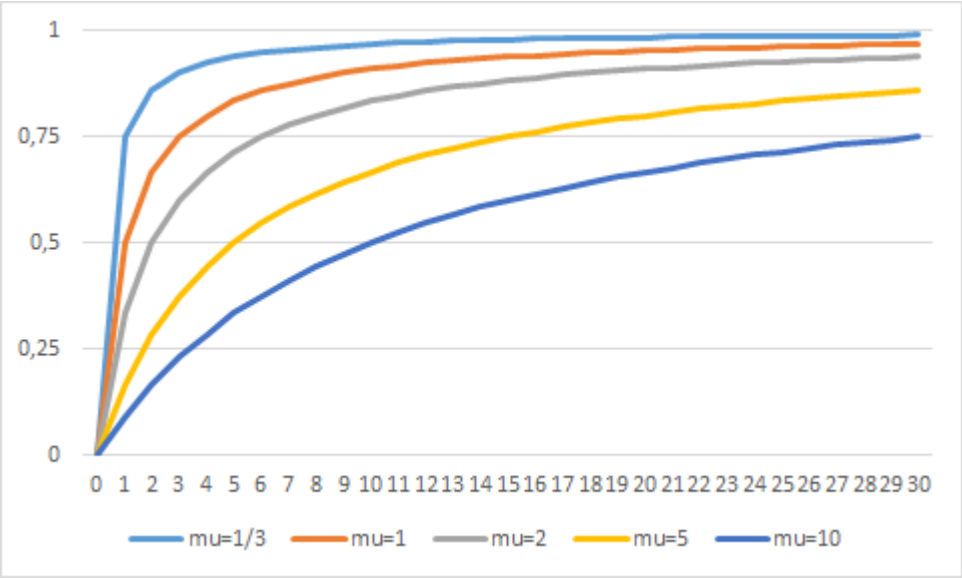
The linear probability model is valid at best only locally. An obvious limitation of this model is that for high values of α, β and *n*¸ the "probability" of contamination provided by this model is higher than one. This problem is common and in order to solve these limitations, "inverse link functions" also called "mean functions" can be defined. I suggest two inverse link functions, which define the conditional probability of contamination when *i* spreaders have used a device (successive use) or share the room (simultaneous use). The first inverse link function is:

$$[20] \qquad \beta(\mu, i) = \frac{i}{i+\mu}$$

For any μ in $\mathbf{R}^+$, $\beta(\mu, i)$ is increasing in *i*, $\beta(\mu, 0) = 0$ (provided that $\beta(0,0)$ is defined to be 0) and $lim_{i\to+\infty}\beta(\mu, i) = 1$, thus $\beta(\mu, i)$ is a good candidate for an inverse link function (which is sometimes called a mean function). An interesting property of this family of functions is:

$$[21] \qquad \forall\, \mu \in (0, +\infty), \beta(\mu, \mu) = \frac{1}{2}$$

that is, μ defines the number of spreaders for which conditional contamination is equal to one half. Thus, μ can be called the *half-contamination parameter*, and by extension, $\beta(\mu, i)$ is called the *half-contamination function* and noted $\beta_{hc}(\mu, i)$. Graph 5 displays the half-contamination function for μ=1/3 (which corresponds to a probability of contamination of 0.75 for one spreader), μ=1, μ=2, μ=5 and μ=10.



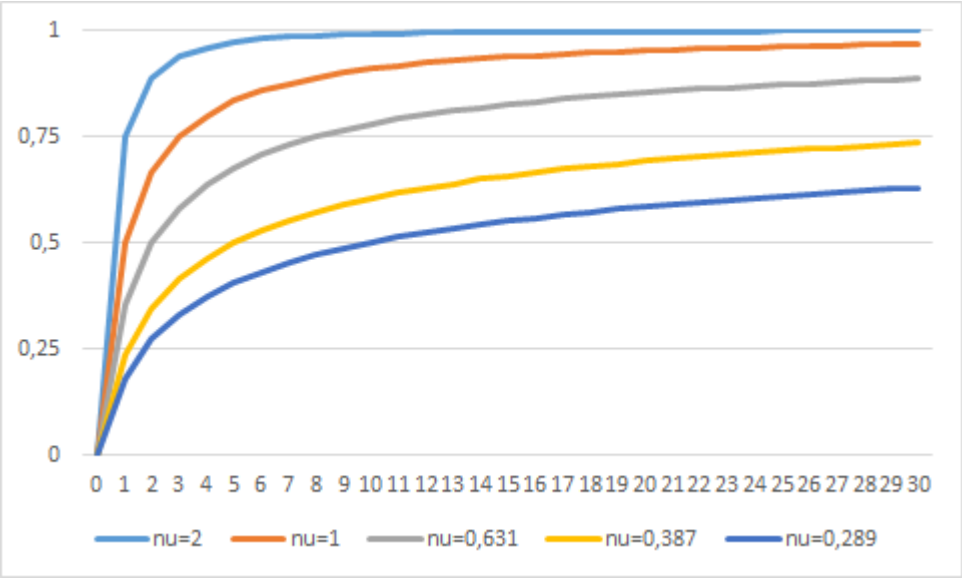
Graph 7: half-contamination inverse link function

Another natural inverse link function is:

$$[22] \qquad \beta_{exp}(\nu, i) = 1 - \left(\frac{1}{1+\nu}\right)^{i}$$

Which can be called the exponential inverse link function. For any *v* in $\mathbf{R}^{+}$, $\beta_{exp}(\nu, i)$ is increasing in *i*, $\beta_{exp}(\nu, 0) = 0$ and $lim_{i\to+\infty}\beta_{exp}(\nu, i) = 1$. $\beta_{exp}(\nu, i)$ is increasing with respect to *v*; thus *v* is a speed of contamination parameter.

Graph 8 displays the exponential inverse link function for parameters calibrated so that one spreader induces a probability of contamination of 0.75 (v=2), one spreaders induces a probability of 0.5 (v=1), and the probability of contamination is one-half for contacts with 2, 5 and 10 spreaders (respectively, $\nu \approx 0.631$, $\nu \approx 0.387$ and $\nu \approx 0.289$), in order to compare with the curves displayed in Graph 7.



Graph 8: exponential inverse link function

8.2 Successive use, effects of multiple spreaders with inverse link functions

With successive use, the expected number of new contaminations when *n* people use the same device is (using the half-contamination inverse link function with parameter μ):

[23]
$$E[c_n] = (1-\alpha)\sum_{i=1}^{n-1}\sum_{j=1}^{i}\beta_{hc}(\mu,j)C_i^j\alpha^j(1-\alpha)^{i-j}$$
$$= (1-\alpha)\sum_{i=1}^{n-1}\sum_{j=1}^{i}\frac{j}{j+\mu}C_i^j\alpha^j(1-\alpha)^{i-j}$$

Table 5 displays the relative increases in the expected number of contaminations when the conditional probability of contamination is determined by a half-contamination inverse link function with parameter μ=1 (left panel) and μ=5 (right panel). As with contamination by a single spreader (section 5, table 1), the relative gains from better organization decreases when the proportion α of contaminated people (spreaders) increases.

However, the effect of higher α is less noticeable than in the single spreader case. This confirms empirically that the inverse link frameworks are in-between the single-spreader case, where α has a strong effect, and the linear probability case, where α has no effect. Economically, this means that the effect of better organization decreases more slowly when additional spreaders have an effect than when only the first spreader affects the probability of contamination of subsequent users.

Accordingly, the effect of better organization is lower than in the single spreader case when α is low, and higher when α is high. In table 5, bolded cells correspond to cases where local convexity is higher than in the single spreader case, so that the effect of better organization are higher.

Table 5:
relative increase in expected contaminations due to a mean-preserving deviation of *n*/2 – half-contamination inverse link function

	μ=1			μ=5		
α *n*	6	10	20	6	10	20
0.001	21,4%	22,6%	23,6%	21,4%	22,7%	23,7%
0.002	21,3%	22,5%	23,4%	21,4%	22,6%	23,6%
0.005	21,1%	22,2%	22,8%	21,3%	22,5%	23,4%
0.01	20,8%	21,7%	21,7%	21,2%	22,3%	22,9%
0.02	20,2%	20,7%	19,9%	20,9%	21,8%	**22,1%**
0.05	18,5%	18,0%	**15,3%**	20,1%	**20,6%**	**19,8%**
0.1	16,0%	**14,3%**	**10,2%**	19,0%	**18,7%**	**16,7%**
0.2	11,9%	**9,2%**	**5,2%**	**16,8%**	**15,7%**	**12,4%**
0.3	**8,9%**	**6,2%**	**3,1%**	**15,0%**	**13,3%**	**9,6%**

Using the exponential inverse link function with parameter *v*, the expected number of contamination is:

[24]
$$E[c_n] = (1-\alpha)\sum_{i=1}^{n-1}\sum_{j=1}^{i}\beta_{exp}(\mu,j)C_i^j\alpha^j(1-\alpha)^{i-j}$$
$$= (1-\alpha)\sum_{i=1}^{n-1}\sum_{j=1}^{i}\left(1-\left(\frac{1}{1+v}\right)^j\right)C_i^j\alpha^j(1-\alpha)^{i-j}$$

Table 6 displays the relative increases in the expected number of contaminations when the conditional probability of contamination is determined by a exponential inverse link function with parameter *v*=1 (left panel) and *v*=0.387 (right panel). These two cases correspond respectively to the cases where contamination happens with probability one-half if a healthy user follows one spreader (μ=1 with the half-contamination inverse link function) and after five spreaders (μ=5 with the half-contamination inverse link function). Results are qualitatively similar to the half-contamination inverse link function.

Table 6:
relative increase in expected contaminations due to a mean-preserving deviation of *n*/2 – exponential inverse link function

	v=1			*v* =0.387		
α *n*	6	10	20	6	10	20
0.001	21,3%	22,6%	23,5%	21,4%	22,6%	23,6%
0.002	21,3%	22,4%	23,2%	21,3%	22,5%	23,4%
0.005	21,0%	22,0%	22,4%	21,2%	22,3%	22,9%
0.01	20,6%	21,4%	21,2%	20,9%	21,8%	22,2%
0.02	19,9%	20,2%	19,1%	20,4%	21,1%	**21,1%**
0.05	17,8%	17,2%	**15,0%**	19,2%	19,6%	**19,9%**
0.1	15,1%	14,0%	**12,0%**	18,0%	**18,8%**	**19,5%**
0.2	11,9%	**11,2%**	**8,2%**	**17,5%**	**18,7%**	**13,7%**
0.3	**10,5%**	**9,5%**	**4,3%**	**17,9%**	**16,7%**	**6,7%**

8.3 Simultaneous use, multiple spreaders with inverse link functions

In the single-spreader simultaneous use framework, concavity happens above a number of users that decreases when the proportion of spreaders increase (section 6). Conversely, in the linear-probability simultaneous use framework, the proportion of spreaders does not matter (section 7).

As already noticed, the frameworks with the half-contamination or exponential inverse link functions are in-between these two polar cases. With a low half-contamination parameter μ or a high speed of contamination parameter *v*, the probability of contamination can be arbitrarily close to that of the single-spreader framework. Conversely, with high μ or low *v*, this probability can be arbitrarily close to the linear probability framework. Thus, whether the contamination function is convex or not is a parameter-dependent issue.

When users share a room simultaneously, and the conditional probability of contamination is determined by a half-contamination inverse link function, the expected number of new contaminations is:

[25]
$$E[c_n] = \sum_{i=1}^{n-1} \beta_{hc}(\mu, i)(n-i)C_n^i\,\alpha^i(1-\alpha)^{n-i}$$
$$= \sum_{i=1}^{n} \frac{i}{i+\mu}(n-i)C_n^i\,\alpha^i(1-\alpha)^{n-i}$$

For this framework Table 7 displays the relative increase in the expected number of contaminations due to non-optimal organization, as defined in section 6, Table 3. Each panel of table 7 corresponds to a value of the half-contamination parameter: μ=1/3 in the upper panel (one spreader induces a 0.75 probability of contamination), μ=1 in the central panel, and μ=5 in the lower panel.

As in the single-spreader case of section 6, the benefits of better organization are higher for low proportions of spreaders in the population. For low values of the half-contamination parameter, the results are rather similar to those of the single-spreader model, and “better organization” is detrimental when the proportion of spreaders is high. For high values of the half-contamination parameter, the effect of the proportion of spreader is less noticeable and remains positive even for high proportions of spreaders, which gets closer to the linear probability case where this parameter is fully irrelevant.

Table 7: relative increase in the number of expected contaminations due to mean-preserving deviations – half-contamination inverse link function

μ=1/3	**Small *n***				**Intermediate *n***			**global**
α	*3 +/- 1*	*3 +/- 2*	*4 +/- 1*	*4 +/- 2*	*6 +/- 3*	*10 +/- 5*	*20 +/- 10*	***10 +/- 9***
0.001	16,6%	66,5%	8,3%	33,2%	29,9%	27,6%	25,9%	89,3%
0.002	16,6%	66,4%	8,3%	33,1%	29,7%	27,3%	25,4%	88,5%
0.005	16,5%	66,0%	8,2%	32,8%	29,3%	26,7%	24,2%	86,4%
0.01	16,3%	65,2%	8,1%	32,3%	28,6%	25,6%	22,2%	82,9%
0.02	16,0%	63,8%	7,8%	31,4%	27,3%	23,5%	18,6%	76,4%
0.05	14,9%	59,7%	7,1%	28,5%	23,4%	18,0%	10,4%	59,5%
0.1	13,2%	53,1%	6,0%	24,1%	17,8%	10,9%	2,9%	38,9%
0.2	10,0%	40,8%	4,0%	16,2%	9,2%	2,7%	-0,8%	15,8%
0.3	7,0%	29,8%	2,3%	9,8%	3,5%	-0,5%	-0,7%	5,7%
0.4	4,4%	20,2%	1,0%	4,8%	0,3%	-1,2%	-0,3%	1,3%
0.5	2,1%	11,9%	0,1%	1,2%	-1,3%	-1,0%	-0,2%	-0,7%
2/3	-0,7%	1,0%	-0,6%	-2,2%	-1,6%	-0,5%	-0,1%	-2,2%
μ=1	**Small *n***				**Intermediate *n***			**global**
α	*3 +/- 1*	*3 +/- 2*	*4 +/- 1*	*4 +/- 2*	*6 +/- 3*	*10 +/- 5*	*20 +/- 10*	***10 +/- 9***
0.001	16,6%	66,6%	8,3%	33,3%	29,9%	27,6%	26,0%	89,4%
0.002	16,6%	66,4%	8,3%	33,2%	29,8%	27,4%	25,6%	88,9%
0.005	16,5%	66,1%	8,2%	32,9%	29,5%	26,9%	24,7%	87,2%
0.01	16,4%	65,6%	8,1%	32,6%	28,9%	26,1%	23,1%	84,5%
0.02	16,1%	64,5%	7,9%	31,8%	27,9%	24,4%	20,2%	79,3%
0.05	15,3%	61,2%	7,4%	29,6%	24,8%	20,1%	13,4%	65,8%
0.1	14,0%	56,0%	6,5%	26,1%	20,4%	14,2%	6,6%	48,4%
0.2	11,4%	46,3%	4,9%	19,8%	13,2%	6,7%	1,4%	27,1%
0.3	9,1%	37,5%	3,5%	14,5%	8,1%	2,9%	0,3%	15,9%
0.4	6,9%	29,5%	2,4%	10,1%	4,6%	1,1%	0,0%	9,8%
0.5	5,0%	22,5%	1,5%	6,6%	2,4%	0,4%	0,0%	6,2%
2/3	2,4%	12,7%	0,5%	2,6%	0,6%	0,0%	0,0%	2,9%
μ=5	**Small *n***				**Intermediate *n***			**global**
α	*3 +/- 1*	*3 +/- 2*	*4 +/- 1*	*4 +/- 2*	*6 +/- 3*	*10 +/- 5*	*20 +/- 10*	***10 +/- 9***
0.001	16,7%	66,6%	8,3%	33,3%	30,0%	27,7%	26,2%	89,8%
0.002	16,6%	66,6%	8,3%	33,3%	29,9%	27,6%	26,0%	89,5%
0.005	16,6%	66,4%	8,3%	33,2%	29,8%	27,4%	25,6%	88,8%
0.01	16,5%	66,2%	8,3%	33,0%	29,5%	27,0%	24,9%	87,6%
0.02	16,4%	65,7%	8,2%	32,7%	29,1%	26,3%	23,6%	85,3%
0.05	16,1%	64,3%	7,9%	31,7%	27,7%	24,3%	20,1%	78,8%
0.1	15,5%	62,0%	7,5%	30,1%	25,6%	21,3%	15,6%	69,5%
0.2	14,4%	57,6%	6,8%	27,2%	21,9%	16,4%	9,8%	55,1%
0.3	13,3%	53,4%	6,1%	24,4%	18,7%	12,8%	6,5%	44,7%
0.4	12,2%	49,4%	5,4%	21,9%	16,0%	10,1%	4,6%	36,9%
0.5	11,2%	45,6%	4,8%	19,6%	13,7%	8,1%	3,4%	31,1%
2/3	9,6%	39,7%	4,0%	16,3%	10,6%	5,8%	2,2%	24,0%

Using the exponential inverse link function with parameter *v*, this expected number of new contaminations is:

[26]
$$E[c_n] = \sum_{i=1}^{n-1} \beta_{exp}(\mu, i)(n-i)C_n^i\,\alpha^i(1-\alpha)^{n-i}$$
$$= \sum_{i=1}^{n} \beta_{exp}(\mu, i)(n-i)C_n^i\,\alpha^i(1-\alpha)^{n-i}$$

Table 8 displays the same results as table 7, but for the case with exponential rather than half-contamination inverse link function. Results are similar for both types of inverse link functions, except that in the "exponential" case, "better organization" is always beneficial for small *n* but is detrimental for high *n* event when the speed of contamination parameter is low.

9. Robustness and limitations

9.1 Robustness and misspecification

The convexity results presented here are quite general. In particular, they must be taken into account in the dynamic case: even with repeated uses, at each time the cross-section variance of *n* must be kept low.

The framework used in the present paper assumes that all non-spreaders are susceptible to contamination. The extension to a SIR-like model where healed people are immune is straightforward: either immune people are known, and they must be excluded from computation, or only their proportion is known. In the latter case, let *h* be the proportion of immune (healed) people in the population of non-spreaders. Then [**5**] becomes:

[27]
$$E[c_n] = \beta(1-\alpha)(1-h)\sum_{i=2}^{n}\left(1-(1-\alpha)^{i-1}\right)$$

Other equatiosare modified accordingly. Note that for the sake of simplicity, I have defined *h* as a proportion of non-spreaders, not as a proportion of the total population. Conversely, if *h* affects the proportion of spreaders, all equations would have to be modified accordingly.

9.2 Variance of the contamination rate and probability of an outbreak

Reducing the variance of exposure in order to slow contagion is a new idea. Actually, in a slightly different context, Lipsitch *et al.* (2003) show that for a given average rate of contamination (number of people infected by each spreader), if the number of cases in the total population is limited to a handful, then a higher variance of the contamination rate reduces slightly the probability of an outbreak. Given this apparent contradiction, the difference between the present framework and the analysis of Lipsitch *et al.* must be explained.

First, the decrease in the probability of an outbreak identified by Lipsitch *et al.* is quantitatively limited, and relies on the fact that the number of cases in the total population is very limited: with ten spreaders, an outbreak is almost certain whatever the variance.

Second, and more importantly, a higher variance reduces the probability of an outbreak but not the expected number of contaminated people. Actually, Lipsitch *et al*. document that over the 201 first SARS cases in Singapore, 103 were contaminated by just 5 people. Thus, there is no real contradiction between the present analysis and that of Lipsitch *et al*., who do not assert that higher variance would be beneficial in any sense.

Table 8: relative increase in the number of expected contaminations

due to mean-preserving deviations – exponential inverse link function

v=2	**Small *n***				**Intermediate *n***			**global**
α	*3 +/- 1*	*3 +/- 2*	*4 +/- 1*	*4 +/- 2*	*6 +/- 3*	*10 +/- 5*	*20 +/- 10*	***10 +/- 9***
0.001	16,6%	66,5%	8,3%	33,2%	29,8%	27,5%	25,8%	89,1%
0.002	16,6%	66,3%	8,3%	33,1%	29,7%	27,2%	25,3%	88,2%
0.005	16,5%	65,8%	8,2%	32,7%	29,2%	26,4%	23,8%	85,7%
0.01	16,2%	64,9%	8,0%	32,1%	28,3%	25,2%	21,5%	81,6%
0.02	15,8%	63,2%	7,7%	31,0%	26,7%	22,8%	17,6%	74,1%
0.05	14,6%	58,3%	6,9%	27,6%	22,4%	16,9%	10,5%	56,5%
0.1	12,5%	50,6%	5,6%	22,6%	16,5%	10,8%	6,8%	39,2%
0.2	8,9%	36,9%	3,5%	14,5%	9,4%	6,8%	3,1%	26,5%
0.3	5,8%	25,8%	2,1%	9,2%	6,8%	5,8%	-1,2%	20,2%
0.4	3,4%	17,1%	1,4%	6,3%	6,7%	3,6%	-2,7%	12,8%
0.5	1,8%	10,8%	1,4%	5,8%	7,1%	0,5%	-2,2%	5,8%
2/3	1,5%	6,0%	2,7%	8,6%	5,3%	-3,3%	-0,9%	-1,5%
v=1	**Small *n***				**Intermediate *n***			**global**
α	*3 +/- 1*	*3 +/- 2*	*4 +/- 1*	*4 +/- 2*	*6 +/- 3*	*10 +/- 5*	*20 +/- 10*	***10 +/- 9***
0.001	16,6%	66,5%	8,3%	33,2%	29,9%	27,5%	25,9%	89,2%
0.002	16,6%	66,4%	8,3%	33,1%	29,7%	27,3%	25,4%	88,5%
0.005	16,5%	65,9%	8,2%	32,8%	29,3%	26,6%	24,2%	86,3%
0.01	16,3%	65,2%	8,1%	32,3%	28,6%	25,6%	22,3%	82,9%
0.02	15,9%	63,8%	7,8%	31,3%	27,2%	23,6%	19,2%	76,7%
0.05	14,9%	59,6%	7,1%	28,5%	23,6%	18,9%	14,0%	62,7%
0.1	13,2%	53,1%	6,1%	24,4%	19,0%	14,5%	10,9%	50,0%
0.2	10,2%	41,9%	4,4%	18,1%	13,9%	11,5%	4,1%	39,5%
0.3	7,8%	33,2%	3,4%	14,3%	12,4%	8,9%	-1,9%	29,3%
0.4	6,0%	26,8%	3,1%	12,7%	12,0%	4,6%	-3,4%	17,7%
0.5	5,0%	22,7%	3,3%	12,8%	11,0%	0,0%	-2,7%	8,0%
2/3	5,5%	20,8%	4,4%	15,2%	6,0%	-4,5%	-1,0%	-1,1%
v=0.387	**Small *n***				**Intermediate *n***			**global**
α	*3 +/- 1*	*3 +/- 2*	*4 +/- 1*	*4 +/- 2*	*6 +/- 3*	*10 +/- 5*	*20 +/- 10*	***10 +/- 9***
0.001	16,6%	66,6%	8,3%	33,3%	29,9%	27,6%	26,0%	89,5%
0.002	16,6%	66,5%	8,3%	33,2%	29,8%	27,5%	25,7%	89,0%
0.005	16,5%	66,2%	8,2%	33,0%	29,5%	27,0%	24,9%	87,6%
0.01	16,4%	65,7%	8,2%	32,6%	29,0%	26,3%	23,8%	85,4%
0.02	16,2%	64,7%	8,0%	32,0%	28,2%	25,2%	22,4%	81,8%
0.05	15,5%	62,0%	7,5%	30,2%	26,2%	23,1%	21,7%	76,0%
0.1	14,4%	58,0%	7,0%	27,9%	24,3%	22,6%	20,6%	74,6%
0.2	12,8%	52,2%	6,3%	25,6%	24,3%	22,6%	7,2%	70,0%
0.3	11,8%	49,0%	6,4%	25,8%	25,6%	17,0%	-2,8%	51,0%
0.4	11,6%	48,3%	7,0%	27,6%	25,3%	8,1%	-4,9%	29,4%
0.5	12,2%	49,9%	7,9%	30,3%	21,9%	0,1%	-3,7%	13,3%
2/3	15,3%	57,2%	9,7%	34,1%	10,7%	-6,7%	-1,2%	-0,2%

10. Conclusion and future research

The homogenous use and cleaning of a limited number of devices used successively by many people limits contamination when some users are unknown contagious carriers of a virus. That is, it is optimal that the same (or at least a very similar) number of people uses each device between two cleanings. This result is based on the fact that when many people use the same device, the probability that some of them are infected increase and the number of people they can infect also increases, so that the number of exposures to contamination is a convex function of the number of successive users. This result is robust to parameters such as the proportion of spreaders or the conditional probability of contamination. For realistic parameters values at the start of an epidemic, reducing heterogeneity in use and cleaning reduces substantially the speed of contamination. However, for this baseline framework, the effects tend to decrease when the proportion of contaminated people increase.

Convexity results extend only partially to the simultaneous use framework, for which I evidence concavity above a number of users that decreases with the proportion of spreaders. This provides additional argument for early organizational action in times of epidemic.

I also consider the case where more spreaders increase the conditional probability of contamination. Convexity tends to increase, at least with the linear probability model. This model, which is used for tractability, may lack realism if the proportion of spreaders is high. Further work, using for instance logit or probit transforms, may be in order here.

Surprisingly enough, these variance considerations, as well as the simple need to reduce the average number of people using a device or sharing a space, are often disregarded even during deadly epidemics. Also, the previous literature points to the beneficial effects of higher variance on the risk of an outbreak, but to the best of my knowledge, not to its detrimental effects on the expected number of contaminations.

Although crucial, these variance considerations are second-order with respect to the need for decreasing the average number of users and more generally to the need for public health policies of social distancing, test and track, or others, in times of epidemic. Their advantage is to be implementable in times of high uncertainty with very scarce information. In later phases, they may help slow contamination and, for instance, make test and trace policies more efficient. Note that if the research question addressed here is the speed of contamination rather than the final steady state, these variance considerations could be embedded in a more general framework to assess their contribution to decreasing the contamination rate R_t below one during epidemics.

The meaning of the results presented here depends on the channels of contamination at work in a specific epidemic. Cleaning constraints may be imposed by the ratio of the number of people and uses to the cleaning capacity over a given period. In that case, and if the time span between two users does not matter, the number of devices is of minor relevance. Conversely, if cleaning constraints are time-related (*e.g.* natural cleaning happens over night as regards aerosol risk of contamination) or if the time-span between two users matters, then increasing the number of devices decreases exposure. These topics deserve consideration in future work.

Dynamic analysis (people using the devices or sharing the offices/production lines repeatedly) is required in future research and may include topics like constant nominal allocation of people to devices and constant running order. The case of different types of devices and the optimal organization with constant nominal allocation (*e.g.* the potential optimality of having the same and/or nested nominal allocations of a group of people to different types of devices) deserves consideration.

11. references

Alvarez, L. (March 28, 2020), "A model to forecast the evolution of the number of COVID-19 symptomatic patients after drastic isolation measures", *mimeo*.

Bai Y, Yao L, Wei T, F. Tian, D.-Y. Jin, L .Chen and M. Wang (February 21, 2020), "Presumed asymptomatic carrier transmission of COVID-19", *JAMA*. (Epub ahead of print)

Bernoulli, D. Essai d'une nouvelle analyse de la mortalité causée par la petite vérole. Mem. Math. Phys. Acad. R. Sci. Paris. 1766:1–45

Booth T. F., B. Kournikakis, N. Bastien, J. Ho, D. Kobasa,L. Stadnyk, Y. Li, M. Spence, S. Paton, B. Henry, B. Mederski, D. White, D. E. Low, A. McGeer, A. Simor, M. Vearncombe, J. Downey, F. B. Jamieson, P. Tang and F. Plummer (2005) "Detection of airborne severe acute respiratory syndrome (SARS) coronavirus and environmental contamination in SARS outbreak units", J Infect Dis. Vol. 191, pp.1472–1477.

Centers for Disease Control and Prevention (February 29, 2020), "Strategies for Optimizing the Supply of N95 Respirators",

Chang, D. G. Mo, X. Yuan, Y. Tao, X. Peng, F.Wang, L. Xie, L. Sharma, C. S. Dela Cruz, E. Qin (March 27, 2020), "Time Kinetics of Viral Clearance and Resolution of Symptoms in Novel Coronavirus Infection,", *American Journal of Respiratory and Critical Care Medicine.*

Dietz K. and J. Heesterbeek (2002), Daniel Bernoulli's epidemiological model revisited. Math. Biosci.; vol.180, pp. 1–21.

Doremalen, N. van, T. Buskmaker, D. H. Morris, M. G. Holbrook, A. Gamble, J. L. Harcourt, N. J. Thornburg; S. I. Gerber, J. O. Lloiyd-Smith, E. de Wit and V. J. Munster (March 17, 2020), "Aerosol and Surface Stability of SARS-CoV-2 as Compared with SARS-CoV-1", New England Journal of Medicine, DOI: 10.1056/NEJMc2004973

Fekom, M., N. Vayatis and A. Kalogeratos (September 9, 2019), "Sequential Dynamic Ressource Allocation for Epidemic Control", *arXiv*

Flaxman, S., S. Mishra, A. Gandy, S. Bhatt, N. M. Ferguson, H. J. T. Unwin, H. Coupland, T. A. Mellan, H. Zhu, T. Berah, J. W. Eaton, P. N. P. Guzman, N. Schmit, L. Callizo, K. E. C. Ainslie, M. Baguelin, I. Blake, A. Boonvasiri, O. Boyd, L. Cattarino, C. Civarella, L. Cooper, Z. Cucunubá, G. Cuomo-Dannenburg, A. Dighe, B. Djaafara, L. Dorigatti, S. van Elsland, R. FitzJohn, H. Fu, K. Gaythorpe, L. Geidelberg, N. Grassly, W. Green, T. Hallett, A. Hamlet, W. Hinsley, B. Jeffrey, D. Jorgensen, E. Knock, D. Laydon, G. Nedjati-Gilani, P. Nouvellet, K. Parag, L. Siveroni, H. Thompson, R. Verity, E. Volz, P. G. T. Walker, C. Walters, H. Wang, Y. Wang, O. Watson, C. Whittaker, P. Winskill, X. Xi, A. Ghani, C. A. Donnely, S. Riley, L. C. Okell, M. A. C. Vollmer (March 30, 2020), "Report 13 - Estimating the number of infections and the impact of non-pharmaceutical interventions on COVID19 in 11 European countries", *MRC Centre for Global Infectious Disease Analysis*.

Jackson, M. O. and D. Lopez-Pintado (2013), "Diffusion and Contagion in Networks with Heterogeneous Agents and Homophily", *Network Science*,vol.1, n° 1, pp. 49-67.

Jackson, M. O. and B. W. Rogers (2007), "Relating Network Structure to Diffusion Properties through Stochastic Dominance", *The B.E. Journal of Theoretical Economics*, vol. 7, n° 1.

Kim S. H., S. Y. Chang, M. Sung, J. H. Park, H. B. Kim, H. Lee, J.-P. Choi, W. S. Choi, and J.-Y. Min (2016), "Extensive viable Middle East respiratory syndrome (MERS) coronavirus contamination in air and surrounding environment in MERS isolation wards", Clin Infect Dis. Vol. 63, pp.363–369.

Kupferschmidt, K. and J.Cohen (Mar. 2, 2020), "China's aggressive measures have slowed the coronavirus. They may not work in other countries", *Science*

Leung, N. H. L. C. Xu, D. K. M. Ip, and B. J. Cowlin (November 2015), "The fraction of influenza virus infections that are asymptomatic: a systematic review and meta-analysis", Epidemiology; vol. 26, n°6, pp 862–872. doi:10.1097/EDE.0000000000000340

Li, Q., X. Guan, P. Wu, X. Wang, L. Zhou, Y. Tong, R. Ren, K. S. M. Leung, E. H. Y. Lau, J. Y. Wong, X. Xing, N. Xiang, Y. Wu, C. Li, Q. Chen, D. Li, T. Liu, J. Zhao, M. Liu, W. Tu, C. Chen, L. Jin, R.Yang, Q. Wang, S. Zhou, R. Wang, H. Liu, Y. Luo, Y. Liu, G. Shao, H. Li, Z. Tao, Y. Yang, Z. Deng, B. Liu, Z. Ma, Y. Zhang, G.Shi, T. T. Y. Lam, J. T. Wu, G. F. Gao, B. J. Cowling, B. Yang, G. M. Leung and Z. Feng (January 31, 2020), "Early Transmission Dynamics in Wuhan, China, of Novel Coronavirus–Infected Pneumonia", New England Journal of Medicine, DOI: 10.1056/NEJMoa2001316

Lipsitch, M., T. Cohen, B. Cooper, J. M. Robins, S. Ma2, L. James, G. Gopalakrishna, S. K. Chew, C. C. Tan, M. H. Samore, D. Fisman and M. Murray (2003), "Transmission Dynamics and Control of Severe Acute Respiratory Syndrom", *Science*, vol. 300(5627), pp. 1966–1970. doi:10.1126/science.1086616

Normile, D. (March 17, 2020) "Coronavirus cases have dropped sharply in South Korea. What's the secret to its success?", *Science*, doi:10.1126/science.abb7566

Omrani AS, Matin MA, Haddad Q, Al-Nakhli D, Memish ZA, Albarrak AM. (2013), "A family cluster of Middle East respiratory syndrome coronavirus infections related to a likely unrecognized asymptomatic or mild case". Int J Infect Dis., vol. 17, pp. 668–72.

Ong, S. W. X.; Y. K. Tan, P. Y. Chia, T. H. Lee, O. T. Ng, M. S. Y. Wong and K. Marimuthu, (March 4, 2020), "Air, Surface Environmental, and Personal Protective Equipment Contamination by Severe Acute Respiratory Syndrome Coronavirus 2 (SARS-CoV-2) From a Symptomatic Patient", JAMA, doi:10.1001/jama.2020.3227

Rothe, C., M. Schunk, P. Sothmann, G. Bretzel, G. Froeschl, C. Wallrauch,. T. Zimmer, V. Thiel, C. Janke, W. Guggemos, M. Seilmaier, C. Drosten, P. Vollmar, K. Zwirglmaier, S. Zange, R. Wölfel, M. Hoelscher, (March 5, 2020), "Transmission of 2019-nCoV Infection from an Asymptomatic Contact in Germany", *New England Journal of Medicine*

Santarpia, J. L., D. N. Rivera, V. Herrera, M. J. Morwitzer, H. Creager, G. W. Santarpia, K. K. Crown, D. M. Brett-Major, E. Schnaubelt, M. J. Broadhurst, J. V. Lawler, St. P. Reid and J. J. Lowe (March 26, 2020), "Transmission Potential of SARS-CoV-2 in Viral Shedding Observed at the University of Nebraska Medical Center", *mimeo*

Tellier, R., Y. Li, B. J. Cowling and J. W. Tang (2019), "Recognition of aerosol transmission of infectious agents: a commentary"*BMC Infectious Diseases* volume 19, Article number: 101

Wilder-Smith, A., M. D. Teleman, B. H. Heng, A. Earnest, A. E. Ling and Y. S. Leo, (2005), "Asymptomatic SARS Coronavirus Infection among Healthcare Workers, Singapore", Emerging Infectious Disease, Vol. 11, n° 7.

World Health Organization (February 28, 2020), "Rational use of personal protective equipment for coronavirus disease 2019 (COVID-19) - Interim Guidance"

World Health Organization (February 28, 2020), "Report of the WHO-China Joint Mission on Coronavirus Disease 2019 (COVID-19)".

Zou L., F. Ruan, M. Huang, L. Liang, H. Huang, Z. Hong, J. Yu, M. Kang, Y. Song, J. Xia, Q. Guo, T. Song, J. He, H. L. Yen, M. Peiris and J. Wu (Marc 19, 2020), "SARS-CoV-2 viral load in upper respiratory specimens of infected patients", *New England Journal of Medicine* DOI: 10.1056/NEJMc2001737

Appendix

Optimal cleaning with two types of devices

In the successive use case, it is interesting to assess the optimal cleaning strategy when two (or more) types of devices are used, for instance, restrooms and coffee machines. I express the cleaning costs in terms of the change in the number of users of a device that can be obtained by allocating one marginal "unit" of cleaning capacity to this type of device. That is, the reallocation of a marginal unit of cleaning capacity from user 2 to user 1 decreases n_1 by Δn_1 and increases n_2 by Δn_2[16]. An intuitive notation of the costs k_1 and k_2 of cleaning respectively devices of types 1 and 2 is:

$k_1 = \frac{1}{|\Delta n_1|}$ and $k_2 = \frac{1}{|\Delta n_2|}$

At the optimum, the gains from allocating a marginal unit of cleaning to a device of type 1 must equal the losses from deallocating a marginal unit of cleaning from a device of type 2. $E[c_{n1}] - E[c_{n1-1}]$ and $E[c_{n2}] - E[c_{n2-1}]$ provides decent approximation of the absolute value of changing respectively n_1 and n_2 by one unit. If α is small enough and if n_1 and n_2 are large enough, these differences can also represent decent approximation in the neighborhood of n_1 and n_2. Thus:

$$[A-1] \qquad |\Delta n_1|.\{E[c_{n1}] - E[c_{n1-1}]\} = |\Delta n_2|.\{E[c_{n2}] - E[c_{n2-1}]\}$$

or:

$$[A-2] \qquad \frac{k_1}{k_2} = \frac{E[c_{n1}] - E[c_{n1-1}]}{E[c_{n2}] - E[c_{n2-1}]} = \frac{\beta_1(1-\alpha)(1-(1-\alpha)^{n_1-1})}{\beta_2(1-\alpha)(1-(1-\alpha)^{n_2-1})}$$

First, note that given the assumption of initial exogenous contamination, and since I disregard the endogenous dynamics, α is the same for both types of devices. On the right-hand-side, what changes between both devices is the conditional probability of contamination β and the number of users n. Also, the numerator and the denominator are increasing functions of n, so that from [10], the optimal (constrained) n_i increases with k_i.

[16] Note that costs are expressed in marginal number of users, *i.e.* in unit of time*use intensity, not time alone.