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# Risk-taking behavior and its impact on treatment, vaccination and diagnosing

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**Abstract**

We present results from two recent mathematical biology studies that address the effect of risk-taking behavior on epidemic models. We show that when risk-taking prevails over risk-avoidance, it is possible to show a detrimental and counterintuitive effect from increasing vaccination and treatment rates over certain critical time intervals. In addition, when risk-taking has a cumulative effect within the population, a lower transmission rate from diagnosed infected individuals may prevent the intended effect of vaccination.

**Introduction**

In this article we discuss results obtained through mathematical modeling techniques about the effect of behavioral factors in the spread of infectious diseases. Specifically, we address the concept of risk-assessment that a typical individual undertakes when faced with an epidemic and how his/her behavior may influence not only the course of the epidemic but also the role of traditionally positive measures, including vaccination, treatment, and/or diagnosing.

During an infectious disease individuals may analyze the risk of transmission and either take or ignore precautionary measures to avoid it. This response is complex and depends on many factors such as: fear of symptoms, mode of transmission, and the availability and efficacy of a treatment or vaccine. Many studies show that people's response to transmission risks change with various factors (1). Efficacious drugs make people worry less about the presence of common pathogens when they are known to be treatable. On the other hand, knowledge about the severity of symptoms (measured by the number of infectious people, virulence, lack of effective treatment) may cause individuals to raise their guard and reduce their exposure risk. Two terms are coined in relation to this behavioral paradigm: disease optimism (increasing risk-taking behavior as a response to "good news") and disease pessimism (decreasing risk-taking behavior as a response to "bad news".

Study about disease pessimism appears more often in the literature (2). Most such studies analyze how sensitive the epidemic is with respect to risk behavior, a typical question being: in what situation does a small change in the risk-taking factor causes a dramatic change in the outcome of the epidemic? Another question frequently posed is related to the influence of risk-taking in the value of the epidemic reproductive number, a mathematical quantity that predicts whether a disease initially rare in the population will spread and cause an epidemic.

In general, it is intuitive to imagine that beneficial measures (treatment, vaccination, or diagnosing) and risk-taking behavior act in the opposite way and should partially cancel each other. For

example, whatever reduction in the number of new cases provided by partial vaccination can be canceled by an increase in risk-taking behavior due to disease-optimism caused by the very existence of such vaccine. However, two recent studies (which form the focus of this article) have shown that under certain circumstances, risk-taking behavior can also change a normally positive measure into a negative one. For example, (3, 4) demonstrates that it is possible to obtain a worse epidemic outcome when vaccination, treatment, or avoidance of infection by diagnosed people are present and/or increased. Essentially, this model shows that if risk-taking is not mitigated, it is possible to obtain a "moral paradox" in which the outcome of the epidemic is actually improved by NOT increasing measures that are normally expected to be beneficial.

Since the concept of the epidemic reproductive number was used extensively in these studies, we devote the following (2nd) section of this paper to a definition and a brief description of this mathematical quantity, which serves as a standard tool for predicting whether an infectious disease increases within the population. We also provide a standard example in the Appendix.

In section 2 from Discussion we describe the results from (3), whereby increasing vaccination and treatment may be detrimental to epidemic control if disease-optimism prevails. This result is achieved by analyzing how the epidemic reproductive number changes when risk behavior is incorporated in the transmission process.

In section 3 from Discussion we focus on the results from (4) that describe a situation where cumulative risk-taking may counter the lower transmission rate resulting from those who have undergone diagnosis. That is, a positive and desirable attitude of diagnosed individuals in taking steps for avoiding disease transmission may actually contribute to an worse outcome of the disease at the population level. We show that this result is possible due to a particular form the epidemic reproductive number takes in this situation.

**Discussion**

*1. The epidemic reproductive number*

A key feature of most infectious diseases is the mechanism of transmission from infected to healthy individuals, when the latter are exposed to infection (also known as susceptible). In order to quantify how the disease will evolve in the affected population one needs, an understanding of the transmission route and the duration of the infection is helpful.

The standard approach in modeling an epidemic is to view the situation from the perspective of one typical infected individual. If the population is well mixed, that is, if there is equal probability in coming into contact with any member of the population, we can assume that this infected individual comes into contact with a fraction of the total population at any given time. Furthermore, there is a probability that each of these contacts is with a susceptible but uninfected individual. Taking into account the number of infected and susceptible individuals, one then arrives at a quantity that gives the number of new infections per unit of time:

*Definition:* the epidemic reproductive number, denoted by R0, represents the number of secondary cases of infections created by a typical infectious individual in a susceptible population.

The term reproductive is used because each infectious individual, by infecting others, acts as a reproductive means for the pathogen in terms of individuals who become infected. Just as with biological reproduction, an infectious individual who, on average, infects more than one healthy person will produce a net increase in the number of infected people, thus spreading the disease. For this reason, most standard results are expressed mathematically as: if $R_0$ is less than one the disease will decline and disappear, whereas if it is greater than one the infection will increase and, eventually, persist in the population until conditions change. The larger $R_0$ is above 1, the faster the number of new cases will appear. A large value of $R_0$ suggests that disease-containing measures need to be applied fast and early.

For this article, it is important to point out that $R_0$ is expected to have a logical dependence on other epidemic related parameters. For example if a treatment or vaccine is available then $R_0$ is expected to decrease as treatment/vaccine rates are increased. This outcome is normally expected because fewer new infections should occur with more treatment or vaccination. However, it is precisely this relationship that may actually be reversed once the effect of risk-taking behavior is incorporated into that part of the model that captures the probability of transmission.

## 2. Risk-taking behavior dependent on the knowledge of the epidemic

One possible behavioral influence on the contact rate among individuals within a population is given by the number of infected, treated or vaccinated individuals in the population. Also known as intuitive epidemiology (5), the idea is that a high burden of infectious individuals may cause people to fear more the transmission risk and take precautionary measures. On the other hand, knowledge that a high number of individuals received successful treatment or are vaccinated sends the message that the disease is easy to manage, curable or easier to avoid altogether. In the context of the HIV epidemic, such a phenomenon has already become a concern. Blower et. al. (6) have shown that, regarding HIV transmission, increases in risk-taking are positively correlated with the availability of treatment. Yet treatment or even vaccination is, in general, only partially effective and as a result of an overconfidence in their efficacy, the risk-taking behavior could compensate any potential benefit of such treatment/vaccine.

In (3), this phenomenon is captured in the models by making the transmission rate dependent on the infected, treated or vaccinated individuals. In doing so, disease transmission will increase if the number of treated and vaccinated individuals increases (disease optimism); at the same time, disease-transmission will decrease if the number if infectious individuals gets larger (disease pessimism). The concern is, of course, what happens if the disease optimism prevails, especially if the protection of the treatment/vaccine is only partial.

The main result in (3) namely that disease transmission increases happens under the condition that disease-optimism prevails. If true, even by a slight margin, the epidemic reproductive number ($R_0$) may no longer reduce in value even when the vaccination rate increases (as normally expected), assuming an initially low vaccination rates. This model suggests that the population needs to be vaccinated well above a certain threshold value (dependent on the disease conditions) in order to ensure that further increases in vaccination are beneficial. This phenomenon occurs because the contact rate may actually increase due to risk-taking before the effect of vaccination protection is manifested in the population.

A similarly counter-intuitive effect happens when treatment rates increase, the only difference being that the detrimental effect happens if the treatment rate is increased in a narrow range of values (also dependent on the disease specifics). The corresponding interpretation is given below.

*Remark*

In the presence of risk taking it may be better to increase the treatment rate either from zero to a low value or beyond some greater threshold value. Increasing treatment to a value between these two possibilities has an effect similar to that of actually increasing the number of secondary infections.

We conclude this section with the observation that, although it may be difficult to quantify the risk-taking dependence on treated and/or vaccinated individuals, to obtain such outcomes requires a predominance of risk-taking over risk-avoidance. In other words, one only needs to decide which is stronger in the average persons behavior: either confidence in the number of vaccinated/treated individuals or fear of the number of infected individuals.

*3. The cumulative effect of risk-taking on the diagnosing process*

The process of identifying who has the disease, that is, diagnosing, is considered another positive step during an epidemic. The rationale is that, when accompanied by strong educational and moral

messages, a diagnosed infectious individual will take measures to avoid spreading the disease to others. Indeed, strong advertising programs geared toward convincing sexually active individuals to undergo regular testing remain a hallmark in the fight against sexually transmitted infections.

In the context of risk-taking, the delay between becoming infected and being diagnosed creates the possibility that two high risk individuals can come into contact with each other. If so, a cumulative effect of risk-taking may happen with a surprising effect based on the role of the diagnosed individuals. We illustrate this effect using the following example analyzed in (4). Suppose susceptible individuals are vaccinated with a partially effective vaccine, meaning that vaccinated individuals are partially protected (i.e. they have a reduced chance of getting the disease). We also assume that individuals who are vaccinated (either healthy ones or ones infected but not yet diagnosed) have a higher risk-taking behavior in response to their reliance on the vaccine. Furthermore we assume that infected individuals who are diagnosed will quarantine themselves and thereby voluntarily lower their contact rate.

The main question addressed in (4) is to ask under what conditions one might obtain a higher epidemic reproductive number with vaccination than without it (which is the opposite wanted effect). The relevant result is that if certain conditions on risk-taking and vaccination efficacy hold, the unwanted effect of vaccination on disease transmission will happen if the contact rate of diagnosed individuals is too low. In other words, the presence of diagnosed individuals in the infection process becomes necessary in order to avoid this unwanted effect (all else being equal). Indeed, and counter to intuitive thinking, mathematical modeling shows that a perfect quarantine of diagnosed individuals guarantees an increase in the epidemic reproductive number.

In order to understand the reason for this unusual (and unwanted) effect we need to understand and interpret the epidemic reproductive number ($R_0$) in this situation. Recall that there are five classes of individuals: susceptible with low and high risk behavior (due to their vaccination status), their infected undiagnosed counterparts, and the infected diagnosed group. The transmission process, viewed as "reproduction" of infected, can happen in two ways: a low risk infected individual will create another

similar infected counterpart by either directly infecting a low risk susceptible individual or by going to the diagnosed class first and then transmitting the disease. Or, a high risk infected individual replicates itself by a direct transmission to a high risk susceptible or after being diagnosed. In this situation, the epidemic reproductive number can be broken in two parts:

$R_0 = R_1 + R_2$

The first term $R_1$ captures the low-risk replication and the second term $R_2$ is the high risk replication described above. The anomaly results (4) when $R_1 < 1$ and $R_2 > 1$. The interpretation is as follows: if the cumulative risk-taking behavior (due to the direct infection between two high risk individuals) is strong enough, the epidemic is driven only by high risk contacts. In this scenario, the diagnosed group acts not only as a low risk class but also as a group that, by its presence in the transmission process, dilutes the number of high risk contacts. To phrase it differently, the presence of the diagnosed class acts as a drain on the groups that sustain the epidemic. However, this means that the presence of the diagnosed class in the infection process is needed to avoid a failure of the vaccination.

It is important to note that this outcome occurs only in the context of a cumulative effect of risk compensation, i.e. the transmission risk is higher when two high risk individuals (infected and susceptible) come in contact. It is not clear whether two high risk individuals do indeed have a cumulative transmission risk strong enough to produce these results. For example, in the context of sexually transmitted infections in heterosexual populations, we have greater confidence that this outcome is plausible when the vaccination and the risk-taking factor apply to one gender only. To illustrate, consider a vaccination that is available to females only (such as the first vaccination available against HPV). Then a high risk contact happens indirectly: a high risk infected female infects a susceptible male who, in turn, infects a high risk susceptible female. The corresponding mathematical model shows that the cumulative risk taking effect is strong enough to produce such results.

**Conclusions**

Behavioral attitudes toward infectious diseases are already recognized as important for the accuracy of mathematical modeling of epidemics. Risk-avoidance can act as a significant control measure of the disease while risk-taking may partially or totally cancel the effect of treatment or vaccination. In most existing modeling studies, treatment and/or vaccination were still desirable measures to be taken, since an increase in their value would decrease the epidemic reproductive number.

In this article we have discussed recent results that suggest the effect of risk-taking may actually go beyond just increasing the contact rate. They may actually reverse the desirable effect obtained from increasing treatment, vaccination, or diagnosing rates. Nevertheless, we emphasize that the important message from this exercise is not to avoid providing treatment or encouraging disease testing. First, any individual in the population benefits from these measures regardless of the outcome of the epidemic. Furthermore, the situations described in (3, 4) may happen for narrow ranges of the predictive parameters that can be difficult or impossible to measure. Rather, we believe it is important to appreciate the full implications of risk-taking behavior whenever present and to actively work against it through public education measures which include, but are not limited to, realistic assessment of the efficacy of treatment or vaccines, presentation of possible unwanted outcome from over-reliance on drugs, education against misuse of medications, etc.

Besides better mathematical models incorporating behavioral attitudes, a needed avenue of further research lies in sociological/cultural studies that better assess and measure risk-taking behavior. The results presented herein have relied on theoretical functions under very general assumptions for risk-taking terms. A correlation of these functions with real data is the next step of this research.

**Appendix A.** An example for the epidemic reproductive number

Suppose that in a population of N individuals S are susceptible and I are infectious. If the population is well mixed we can assume that this infected individual comes in contact with a fraction fN of the total population per unit of time. Each of this contact is with a susceptible with probability S/N . Since, in total, there are I infectious individuals we arrive at the total number of new infections per unit of time:

$fN \times S/ N \times I = fSI.$

This form of the contact rate is also known as the mass action law. On the other hand, if the population is not well-mixed and one person can have contact with an average fixed number of individuals per unit of time, denoted by $\lambda$, then the number of new infections is represented by the following expression called the *standard incidence*:

$\lambda \times S/N \times I = \lambda SI/ N$

Many other more realistic forms of the contact rate are possible but these two remain widely used due to their mathematical simplicity.

To arrive the epidemic reproductive number, suppose initially the entire population is healthy (S = N) and that one infectious individual appears (I = 1). Suppose also that an individual leaves the infectious status with the rate $\mu$ (due to recovery, treatment or disease induced mortality). The reciprocal of this rate $1/\mu$ represents the estimated duration of infection. Using the standard incidence above we have that the number of new infections per unit of time (when the disease is rare) is:

$\lambda \times N/ N \times 1 = \lambda$

Finally,

$\lambda \times 1/ \mu = \lambda/ \mu$

represents the new infections created by an infectious individual during his entire infectious period while the disease is rare.

Under normal conditions, an increase in either treatment of vaccination will reduce the size of $R_0$. This is because vaccination reduces the number of available susceptible to infect and treatment reduces the duration of infection. An example of an epidemic reproductive number with vaccination and treatment could be:

$R_0 = \lambda/ (\mu + v)(\mu + r)$,   where v and r are the vaccination and treatment rates respectively.

In most epidemic models the contact rate $\lambda$ is a fixed term though it incorporates multiple assumptions such as: average number of individuals met per unit of time (a), probability of contact sufficient to transmit the disease ($p_1$), probability of actual transmission ($p_2$), probability of becoming infectious ($p_3$), etc. Then all these assumptions lead to:

$\lambda = a \times p_1 \times p_2 \times p_3$

As long as the features involving the transmission of the pathogen are taken to be constant, the contact rate can be described by a single variable. However, there are situations, as seen in (3), in which the contact rate itself may vary. In particular, it may vary with respect to the size of various types of individuals separated by their disease status: infected, recovered, vaccinated, etc.

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