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The cumulative effect of risk compensation on infection preventive measures

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ABSTRACT

We study several epidemic models (with and without gender structure) that incorporate risk compensation behavior in response to a lower chance of acquiring the infection as a result of preventive measures that are only partially effective. We show that the cumulative risk compensation that occurs between a high risk susceptible and infectious individual may play an important role in whether the implementation of these measures is successful in lowering the epidemic reproductive number. In addition, we show that certain levels of risk compensation may cancel the benefit of the low infection risk practiced by diagnosed infectious individuals when the goal is a reduction of the epidemic reproductive number.

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1. Introduction

Risk compensation represents the behavioral adjustments that individuals undertake in response to the perceived levels of risk: people become more careful if the level of risk is high and less careful if it is low. In the context of epidemiology, risk compensation translates to whether individuals take precautionary measures to avoid infection. Depending on the nature of the disease, these measures can include: washing hands, avoiding crowds, prophylactic treatments, using condoms in the case of sexually transmitted infections, etc. Risk compensation in response to low risk levels may not constitute a major problem if the risk is indeed low or insignificant. On the other hand, there might be a major discrepancy between the *perceived* and the *real* level of risk. For example, a vaccine that offers partial (but not total) protection might have a much lower protective effect than what people believe. New and effective treatments may also constitute additional factors for increasing the risk behavior.

Most of the existing studies on risk compensation focus on how sensitive the epidemic is with respect to behavioral changes (see Poletti et al., 2009, Poletti et al., 2012). Many disease specific articles on risk behavior are done in the context of HIV pandemic. Specifically, some researchers have documented associations between improvements in HIV treatment outcomes and greater sexual risk taking; i.e. more cases of condomless sex and/or increased number of sex partners in response to information that HIV treatments have improved (see Chen, 2013, Crepaz et al., 2004, Ostrow et al., 2002, Stolte et al., 2004, Ostrow et al., 2000). While no vaccine in the classical sense exists for HIV, recent research focused on measures that act as partially effective preventive measures. One of them is “Treatment as Prevention” or “TasP” which refers to HIV prevention by using certain antiretroviral drug regimens (ART). This reduces the risk of acquiring the infection.

Other preventive measures may be gender specific. One example is given by the *Human Papillomavirus* (HPV) vaccine which was originally available for girls and young women (though now it is recommended for both genders). Another gender specific example is given by male circumcision (MC) which is thought to offer significant protection against woman-to-man HIV transmission. While these measures are advertised, the issue of risk compensation has been recognized as a potential danger in the fight against infectious diseases (in the case of HIV see Cassell et al., 2006, Blower and McLean, 1994).

One additional aspect related to risk behavior (which is the main motivation for our paper) is the potential of *compounding of risk compensation* when the contact happens between a susceptible and an infected person who is not aware of his/her infection status. This delay between the moment of infection and the realization

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that one is infected is caused by multiple disease specific factors. In the case of HIV infection, it is common for individuals to go years without diagnosis even if they are experiencing symptoms caused by HIV infection. The reasons for this outcome are attributable to the fact that symptoms of HIV infection (at least in the initial years of infection) can be easily attributed to flu/cold related infections (and, therefore, do not cause alarm), and/or the stigma associated with testing HIV positive deters individuals from getting HIV tested and providers from asking about risk taking related to HIV transmission which delays testing/treatment even if alarming symptoms are present. Therefore it is likely that the increased risk behavior due to preventive measures remains more or less the same during this period between infection and diagnosis. Finally, when individuals are diagnosed with HIV, a reduction in their sexual risk taking is reported: individuals diagnosed with HIV report fewer acts of condomless sex than individuals who are HIV infected/unaware (see Eaton and Kalichman, 2009, Marks et al., 2005).

This aspect is crucial for HIV where this delay is measured in years. Once an infected person is diagnosed, the risk behavior should decrease dramatically, to a level below normal risk, since, presumably, the person will lower his/her risk behavior in order to protect others from infection (such as strict adherence with safe sex practices in the case of HIV). Low risk behavior of diagnosed individuals remains a crucial weapon against any epidemic which is the reason why individuals are encouraged both to undergo disease testing and, if results are positive, to safeguard others against transmission risk.

Although it is not obvious how one can measure the compounding of risk behavior, a study that suggests how two high risk individuals may collude and accumulate their risk can be inferred from an economic model about the market for unprotected commercial sex (Gertler et al., 2005). Using the notations in this paper, the bargaining model proposed therein, assumes that there is a maximum amount a client is willing to pay (β) and a minimum amount a sex worker will accept (γ) in exchange for the riskier condom-less sex. It follows that the condom will not be used if $\beta > \gamma$. While this model does not categorize individuals by risk-taking levels, one can assume that, in real life, these monetary thresholds are not fixed and they are subject to change on the spot depending on how negotiation unfolds. In other words, there should be a lower probability of not using a condom if β is only slightly larger than γ and a much higher one if $\beta \gg \gamma$. The latter essentially indicates an encounter between two high risk individuals (a client willing to pay a lot and a sex-worker willing to accept very little).

In our article we investigate the effect of risk compensation in response to the presence of a partially effective preventive measure for a disease without recovery. We analyze how this affects the implementation of this measure if the infectious class is split into diagnosed and un-diagnosed individuals. If risk compensation is present, we assume that its effect is even more pronounced if a higher risk susceptible comes in contact with a higher risk infectious individual who is not aware of his/her infectious status (i.e. un-diagnosed). The focus of our assessment is on how the epidemic reproductive number changes once the prevention is implemented and the risk compensation is taken into account.

The epidemic reproductive number (traditionally denoted by $\mathcal{R}_0$) usually represents the secondary number of infections caused by an infectious individual in a healthy population. It is also related to the stability condition of the disease free equilibrium: if $\mathcal{R}_0 < 1$ the disease free state is locally stable and the disease may clear while, if $\mathcal{R}_0 > 1$, then the epidemic will persist. Therefore, a measure of success of any intervention is whether $\mathcal{R}_0$ decreases with such intervention. This is true even if the reduction is not below the 1 threshold since a lower $\mathcal{R}_0$ suggests fewer new infections per unit of time. Our results are two-fold:

- We show that the compounding effect of risk compensation increases the chance of reverting the protective effect of prevention.
- More interestingly, the expected and promoted low risk behavior of diagnosed individuals may have the effect of undermining the positive outcome of introducing a partially effective prevention measure: low infection rates from diagnosed individuals may cause the epidemic reproductive number to actually *increase* if prevention is introduced. This correlation is primarily due to the demographic interplay between the high risk groups.

In order to confer some disease generality, we show these results using two models: with and without gender structure. Another motivation for showing our result in these two frameworks is due to the possibility that a partially effective prevention measure may be available for one gender only. In the next section we introduce the one-sex model and compute the epidemic reproductive number with and without prevention. We analyze under what conditions the reproductive number actually increases with the level of prevention and show that this may happen if the risk behavior of the diagnosed individuals is too low. In Section 3 we show that a similar situation happens in a two-sex model in which the prevention measure is available for males only (to resemble a potential benefit from MC as a working example). We show that the cumulative risk compensation effect happens indirectly, between two males that pass the infection to one-another, via infecting a susceptible female. We retrieve the same upper bound on the reduction of infection risk parameter on males as a condition for worsening the epidemic in the presence of prevention (similar to the one found in the one-sex model). In the last section we provide our interpretation of these results and thoughts on expanding this research further.

2. The one-sex model

The model below describes a generic disease without recovery for which a partially effective preventive measure is available. The susceptible class without protection is denoted by S_1 and their infected counterparts by I_1. We also assume that individuals, in response to the knowledge of this protection, exhibit a certain degree of risk compensation described by a constant parameter $\epsilon > 1$. S_1 denotes the susceptible population without preventive measures and S_2 are the susceptible individuals that benefit from prevention. I_1 and I_2 denote their infected counterparts before diagnosing is made. Furthermore, we assume that this risk compensation is stronger whenever the contact happens between two protected individuals: a susceptible and an infected undiagnosed one. In addition to distinguishing between protected and non-protected individuals, we also assume that, initially, all infectious individuals are not-diagnosed. This means that, for a certain amount of time after infection, the risk-behavior is identical to that of the susceptible individuals and only upon learning of the infectious status (i.e. diagnosis) individuals will lower their risk behavior to avoid spreading of the disease. Diagnosed individuals are denoted by J. The resulting five equations model is given below together with its flow diagram. The description of the parameters is provided in Table 1.

$$\begin{cases} S_1' = \beta P - \lambda \dfrac{S_1}{P}(I_1 + \epsilon I_2 + \eta J) - \bar{\mu} S_1 - \delta S_1, \\ S_2' = \delta S_1 - \lambda \xi \epsilon \dfrac{S_2}{P}(I_1 + \epsilon I_2 + \eta J) - \bar{\mu} S_2, \\ I_1' = \lambda \dfrac{S_1}{P}(I_1 + \epsilon I_2 + \eta J) - \bar{\mu} I_1 - d I_1 - \alpha I_1, \\ I_2' = \lambda \xi \epsilon \dfrac{S_2}{P}(I_1 + \epsilon I_2 + \eta J) - \bar{\mu} I_2 - d I_2 - \alpha I_2, \\ J' = d(I_1 + I_2) - \bar{\mu} J - \nu J, \end{cases} \tag{1}$$

where $P = S_1 + S_2 + I_1 + I_2 + J$ is the total population size (see Fig. 1).

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