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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.

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 un-diagnosed 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).

Table 1
Variables and parameters of model (1).

Symbol	Meaning
S_1	Susceptible individuals without preventive measures and with normal risk behavior and infection susceptibility
S_2	Susceptible individuals with preventive measures that have higher risk behavior and lower infection susceptibility
I_1	Infectious individuals without preventive measures and not yet diagnosed
I_2	Infectious individuals with preventive measures that are not yet diagnosed
J	Diagnosed infectious individuals with low risk behavior
β	The per capita birth rate
δ	The rate at which prevention is implemented
$\bar{\mu} = \mu + bP$	The logistic mortality rate
λ	The infection rate
$0 < \xi \leq 1$	The reduction factor of transmission for protected individuals
$\epsilon \geq 1$	The augmenting factor of transmission rate corresponding to risk compensation of protected individuals
$0 < \eta \leq 1$	The reduction factor of the transmission rate corresponding to low risk behavior of diagnosed infectious individuals
d	The rate at which infectious individuals are diagnosed
α	The disease induced mortality rate of infectious individuals not yet diagnosed
ν	The disease induced mortality of infectious diagnosed individuals.

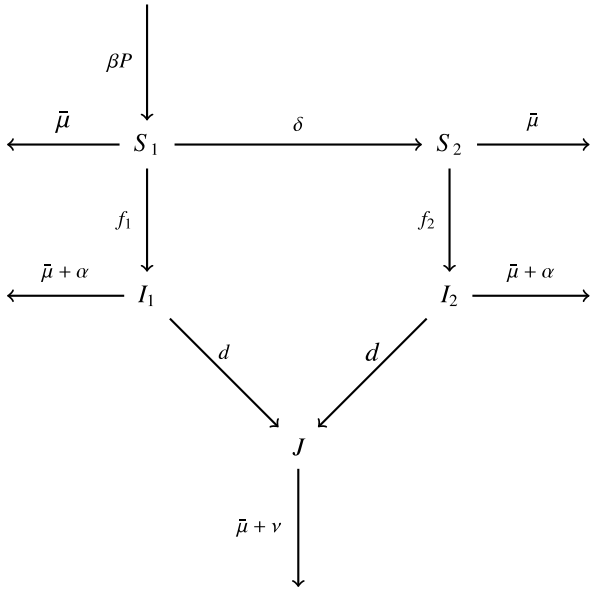

Fig. 1. Flow diagram of the one-sex model. The transmission rates are denoted by $f_1 := \frac{1}{P}(I_1 + \epsilon I_2 + \eta J)$ and $f_2 := \frac{1}{P}\lambda\xi\epsilon(I_1 + \epsilon I_2 + \eta J)$.

The epidemic reproductive number of this model is given below (see Appendix A for its derivation):

$$\mathcal{R}_0^{vd} := \lambda \frac{(\beta + \nu)(\beta + \xi\epsilon^2\delta) + d\eta(\beta + \xi\epsilon\delta)}{(\beta + \delta)(\beta + d + \alpha)(\beta + \nu)}.$$

In order to assess the effect of preventive measures in the context of risk-compensation, we provide below the epidemic reproductive number under several modified assumptions. First, if there is no prevention then it becomes

$$\mathcal{R}_0^{d} := \lambda \frac{\beta + \nu + \eta d}{(\beta + d + \alpha)(\beta + \nu)}.$$

With prevention but without diagnosis is

$$\mathcal{R}_0^{v} := \lambda \frac{\beta + \xi\epsilon^2\delta}{(\beta + \delta)(\beta + \alpha)}.$$

Finally, the reproductive number with neither prevention nor diagnosis is

$$\mathcal{R}_0 := \frac{\lambda}{\beta + \alpha}.$$

One measure of identifying whether risk-compensation renders the prevention measures ineffective as an intervention measure is to ask whether the epidemic reproductive number becomes actually greater with prevention than without it.

Thus, we wish to know under what conditions we have

$$\mathcal{R}_0^{vd} > \mathcal{R}_0^{d}.$$

This inequality is equivalent to

$$(\beta + \nu)(\xi\epsilon^2 - 1) > \eta d(1 - \xi\epsilon).$$

Notice that, if $\xi\epsilon > 1$, this inequality is trivially satisfied. This is expected since the risk-taking behavior factor $\epsilon > 1$ acts as a cancellation effect of the protection represented by ξ. On the other hand if the risk-compensation falls in a range given by

$$\xi\epsilon^2 > 1 > \xi\epsilon, \tag{2}$$

then $\mathcal{R}_0^{vd} > \mathcal{R}_0^{d}$ is satisfied provided we have the following upper bound on the diagnosis related terms:

$$\eta d < \frac{(\beta + \nu)(\xi\epsilon^2 - 1)}{1 - \xi\epsilon}.$$

An upper bound on the diagnosing rate d is to be expected since, under normal circumstances, diagnosed infectious individuals have a much lower risk behavior in order to avoid spreading the infection. On the other hand, the upper bound on the reduction of transmission of diagnosed individuals is more interesting. Assuming that all terms are fixed except η, this upper bound essentially says that prevention fails to reduce the reproductive number if transmission from the diagnosed individuals is too low.

Indeed, assuming that diagnosed individuals exhibit perfect quarantine (i.e. $\eta = 0$) then the implementation of preventive measures always fail to reduce the reproductive number if the risk-compensation falls within the range given by (2).

The key reason for this situation is the cumulative effect of risk-compensation driven by S_2–I_2 transmission. (2) describes a situation in which the risk-compensation is not strong enough to render ineffective the protection of normal risk contacts (i.e. $\xi\epsilon < 1$) but strong enough to revert the protection for high-risk contacts ($\xi\epsilon^2 > 1$). Hence, any possible epidemic is sustained primarily by S_2 and I_2 contacts and the presence of J class acts not only as a lower infectious class but also as a class that dilutes the number of high-risk contacts.

This creates a situation in which the low risk behavior practiced by diagnosed individuals facilitates an increase in the reproductive number after the introduction of prevention. This is not possible without partially effective protection and risk compensation as we can see in the following situation:

$$\mathcal{R}_0^{d} < \mathcal{R}_0 \quad \text{if and only if } \eta < \frac{\beta + \nu}{\beta + \alpha}.$$

If the mortality of the diagnosed and un-diagnosed is the same ($\nu = \alpha$) then the above inequality is equivalent to $\eta < 1$. In

general, if treatment is available, one can assume $\nu < \alpha$ and then the condition becomes:

$$\eta < \frac{\beta+\nu}{\beta+\alpha} < 1.$$

In all cases, as expected, a higher diagnosing rate paired with a reduced infectivity of the diagnosed individuals will lower the reproductive number. On the other hand, partially effective prevention combined with risk compensation may increase the reproductive number and, more importantly, this can happen whenever the risk behavior of diagnosed individuals is too low. In this sense, a low value of η becomes detrimental.

In order to get a better understanding of why this phenomenon occurs, we provide below a different format for $\mathcal{R}_0^{vd}$ that can be interpreted biologically:

$$\mathcal{R}_0^{vd} = \left(\frac{\beta}{\beta+\delta}\right)\left[\frac{\lambda}{\beta+d+\alpha} + \frac{d}{\beta+d+\alpha}\frac{\lambda\eta}{\beta+\nu}\right] + \left(\frac{\delta}{\beta+\delta}\right)\left[\frac{\lambda\xi\epsilon^2}{\beta+d+\alpha} + \frac{d}{\beta+d+\alpha}\frac{\lambda\xi\epsilon\eta}{\beta+\nu}\right].$$

For the purpose of this interpretation, we view β as the logistic natural mortality when the infection is absent (since, at the disease free state, $\beta = \mu + bP$). $\frac{1}{\beta+\delta}$ is the average time spent by a susceptible in the S_1 class. Hence $\frac{\beta}{\beta+\delta}$ represents the fraction of susceptible that remain un-protected and $\frac{\delta}{\beta+\delta}$ is the protected fraction that moves from S_1 to S_2. $\frac{1}{\beta+\delta+\alpha}$ is the average time spent by an infectious individual in either I_1 and I_2 classes while $\frac{1}{\beta+\nu}$ is the average time spent by a diagnosed individual in the J class. Finally, $\frac{d}{\beta+d+\alpha}$ represents the probability of diagnosing one infectious individual of either type I_1 or I_2.

The first term of $\mathcal{R}_0^{vd}$ describes the effect of an infectious agent introduced in a completely susceptible population without preventive measures while the second term has a similar meaning for a susceptible population that benefits of preventive measures in its entirety. Thus, the first term of $\mathcal{R}_0^{vd}$ represents the number of secondary cases of infections of type I_1 caused either directly or through transfer to the diagnosed class. Similarly, the second part represents the number of secondary cases of infections of type I_2 caused, again, either directly or through transfer to J first. The condition (2) that ensures a failure of prevention by an increase in the reproductive number can be written as:

$$\frac{\lambda\xi\epsilon^2}{\beta+d+\alpha} + \frac{d}{\beta+d+\alpha}\frac{\lambda\xi\epsilon\eta}{\beta+\nu} > \frac{\lambda}{\beta+d+\alpha} + \frac{d}{\beta+d+\alpha}\frac{\lambda\eta}{\beta+\nu}.$$

In other words, this inequality, essentially says that the unwanted effect of increasing $\mathcal{R}_0^{vd}$ happens if I_2 replacements created by the very process of implementation of preventive measures in the presence of risk-taking, exceeds the number of I_1 replacements. If we are in the case $\xi\epsilon^2 > 1 > \xi\epsilon$, we observe that this is in fact guaranteed if $\eta = 0$ or, in other words, if diagnosed individuals avoid all contacts. However, since I_2 cannot replace itself via a transfer through J (since $\xi\epsilon < 1$), the presence of diagnosed individuals in the transmission process can dilute the effect of direct I_2 replacements and, ultimately, avoid an increase in the reproductive number.

In this context, diagnosed individuals, by their mere presence in the infection process, dilute the cumulative risk compensation effect which explains the upper bound on η. We observe also that the condition $\xi\epsilon^2 > 1 > \xi\epsilon$ is more likely to happen whenever there is a discrepancy between the perceived and the true effectiveness of protection (i.e. high values of ξ require comparatively small risk compensation effects for this inequality to be true). For example, if $\xi = 0.75$, which would correspond to a prevention measure that is only 25% effective, then the risk compensation needs to be between $1.15 < \epsilon < 1.33$.

It is difficult to establish whether a certain numerical value of ϵ matches the real data on risk-taking. However, some evidence exists from several studies on behavioral attitudes toward infection risks. For example, in Chesney et al. (1997), Chesney et al. measured the risk compensation effect during an HIV vaccine trial in San Francisco and found an increase in unprotected sex from a base-line percentage of 9%–13% after 6 months (which is an increase of a factor of 1.4).

Below we provide several numerical examples to illustrate the effect of risk compensation and low risk behavior on the change in the epidemic reproductive number before and after prevention is implemented. Using demographic data from USA in 2013 provided by the Center of Disease Control and Prevention (CDC, http://www.cdc.gov/), we approximate the birth and the death rates: $\beta = 0.01244$ and $\mu = 0.00822$. From the same source we approximate the HIV additional mortality rate as being $\nu = 0.000025$. For undiagnosed individuals we use the disease mortality $\alpha = 0.08$ computed as the reciprocal of the average duration of the last two stages of HIV infection: latency (which is typically 10 years) and AIDS (approximately 3 years). CDC provides data for the new HIV infections in the form of total incidence (50,000 per year in USA) from which we can estimate $\lambda = 0.0418$. To estimate the diagnosing rate we use the CDC data from 2013 which mentions that 47,352 HIV diagnoses have been established. At the same time about 232,700 individuals are infected but unaware of their HIV positive status (approximately 21% of the infected population as provided in Gardner et al., 2011 and references therein). This gives us an estimated value of $d = 0.203$. While MC is not universally advertised as a partially effective vaccine, for the purpose of this example, we are going to use again CDC data regarding male circumcision in USA to estimate $\delta = 0.0036$ (here we assumed an even sex ratio at birth and the prevalence of circumcision of 58.3%).

In Fig. 2 we plot the threshold corresponding to $\mathcal{R}_0^{vd} = \mathcal{R}_0^{d}$ as an implicit curve in the (ϵ, η) region for three values of ξ and using the parameters mentioned above. The region where preventive measures lower the reproductive number is above each curve. Notice that, in all cases, low enough values of the risk-taking factor ϵ will ensure the outcome is positive while high enough values of ϵ lead to a negative outcome regardless of the diagnosed group risk level. The part that illustrates our results is observed for intermediate values of ϵ right under each curve. In that range, for any given value of ξ, the positive outcome appears only if η is high enough.

For example, if $\xi = 0.6$ and $\epsilon = 1.5$ the threshold level of η is $\eta^* = 0.215$. This means any value of η below this threshold will lead to an increase in the reproductive number while any value above it will decrease it. The epidemic may also change from clearance to persistence or the other way around as seen in the following samples:

$$\eta = 0.1, \quad \mathcal{R}_0^{d} = 0.372, \quad \mathcal{R}_0^{vd} = 0.378,$$
$$\eta = 0.375, \quad \mathcal{R}_0^{d} = 1.006, \quad \mathcal{R}_0^{vd} = 0.997.$$

Notice that in the first case, while the reproductive number increases, the disease free state is still locally stable since both values are less than 1. But this need not be the case. If λ is larger (all other parameters being the same) then we can have an increase to an endemic level:

$$\lambda = 0.088, \quad \eta = 0.144, \quad \mathcal{R}_0^{d} = 0.996, \quad \mathcal{R}_0^{vd} = 1.004.$$

Remark 2.1. It is important to point out that the effect described above only requires that an S_2–I_2 encounter comes with a higher

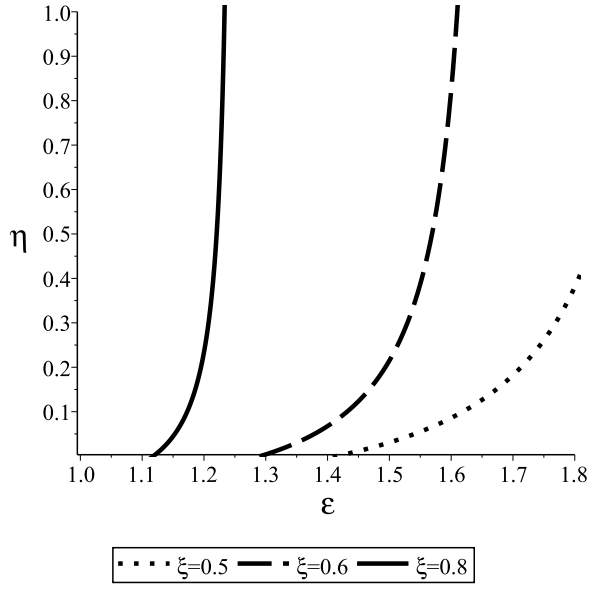

Fig. 2. The level curve $\mathcal{R}_0^{vd} = \mathcal{R}_0^{d}$ in the (ϵ, η) plane for three different levels of preventive measures efficacy ξ. The region above and to the left of each curve corresponds to the success of introduction of preventive measures (i.e. $\mathcal{R}_0^{vd} < \mathcal{R}_0^{v}$).

risk-compensation effect than in the case of S_1–I_2 or S_2–I_1 contacts but not necessarily of the multiplicative order (i.e. ϵ^2 vs ϵ). In other words ϵ^2 can be replaced by a different variable assumed greater than ϵ without significant differences in the analysis above. On the other hand, in the next section, we show that this multiplicative order of the cumulative risk compensation appears naturally in the case of sexually transmitted diseases where only one gender benefits from preventive measures and exhibits risk-compensation.

3. The two-sex model

As we mentioned in the Introduction, in the context of sexually transmitted diseases (STD), prevention may be available only for one gender only. Even if transmission is primarily through a heterosexual route, a cumulative risk compensation effect is still possible via a female intermediate: a susceptible female infected by a high risk male who later infects a high risk susceptible male. The purpose of this section is to show that the phenomenon described in the previous section appears in this framework as well. Below we propose a two-sex epidemic model with a partially effective protection available for males only:

$$\begin{cases} S_f' &= \beta\gamma_f \mathcal{M}(F,M) - \lambda \mathcal{M}(F,M)\dfrac{S_f}{F}\dfrac{I_m + \epsilon I_{mv} + \eta_m J_m}{M} - \bar{\mu}_f S_f, \\ S_m' &= \beta\gamma_m \mathcal{M}(F,M) - \lambda \mathcal{M}(F,M)\dfrac{S_m}{M}\dfrac{I_f + \eta_f J_f}{F} - \bar{\mu}_m S_m - \delta S_m, \\ S_{mv}' &= \delta S_m - \lambda\xi\epsilon \mathcal{M}(F,M)\dfrac{S_{mv}}{M}\dfrac{I_f + \eta_f J_f}{F} - \bar{\mu}_m S_{mv}, \\ I_f' &= \lambda \mathcal{M}(F,M)\dfrac{S_f}{F}\dfrac{I_m + \epsilon I_{mv} + \eta_m J_m}{M} \\ &\quad - \bar{\mu}_f I_f - \alpha I_f - d_f I_f, \\ I_m' &= \lambda \mathcal{M}(F,M)\dfrac{S_m}{M}\dfrac{I_f + \eta_f J_f}{F} - \bar{\mu}_m I_m - \alpha I_m - d_m I_m, \\ I_{mv}' &= \lambda\xi\epsilon \mathcal{M}(F,M)\dfrac{S_{mv}}{M}\dfrac{I_f + \eta_f J_f}{F} - \bar{\mu}_m I_{mv} - \alpha I_{mv} - d_m I_{mv}, \\ J_f' &= d_f I_f - \bar{\mu}_f J_f - \nu J_f, \\ J_m' &= d_m(I_m + I_{mv}) - \bar{\mu}_m J_m - \nu J_m, \end{cases} \tag{3}$$

where $F = S_f + I_f + J_f$ and $M = S_m + S_{mv} + I_m + I_m v + J_m$ represent the total female and male populations.

$\mathcal{M}(F, M)$ stands for the pair-formation function which models both the birth and the heterosexual transmission of the disease. For details about its properties and typical examples see Iannelli et al. (2005), Maxin and Sega (2013). The meaning of the other parameters is given in Table 2.

The derivation of the epidemic reproductive number is presented in Appendix B. Here we actually provide the square of $\mathcal{R}_0^{vd}$ in order to simplify the exposition when we discuss the case in which the risk compensation has a detrimental effect on the introduction of the preventive measures.

$$\left(\mathcal{R}_0^{vd}\right)^2 := (\lambda T^*)^2 F^* M^* \times \frac{(\mu_f^* + d_f\eta_f + \nu)[(\mu_m^* + \nu)(\mu_m^* + \delta\xi\epsilon^2) + d_m\eta_m(\mu_m^* + \delta\xi\epsilon)]}{(\mu_m^* + \delta)(\mu_f^* + \nu)(\mu_m^* + \nu)(\mu_f^* + \alpha + d_f)(\mu_m^* + \alpha + d_m)},$$

where F^* and M^* are the female and male equilibrium populations in the absence of the disease, μ_f^* and μ_m^* are the respective mortality rates when the population is at the disease free steady state and T^* denotes $\frac{\mathcal{M}(F^*,M^*)}{F^*M^*}$. In the absence of prevention ($\delta = 0$) the epidemic reproductive number becomes

$$\left(\mathcal{R}_0^{d}\right)^2 = (\lambda T^*)^2 F^* M^* \times \frac{(\mu_f^* + d_f\eta_f + \nu)(\mu_m^* + d_m\eta_m + \nu)}{(\mu_f^* + \nu)(\mu_m^* + \nu)(\mu_f^* + \alpha + d_f)(\mu_m^* + \alpha + d_m)}.$$

After some computations we see that $\mathcal{R}_0^{vd} > \mathcal{R}_0^{d}$ if and only if

$$(\xi\epsilon^2 - 1)(\mu_m^* + \nu) > d_m\eta_m(1 - \epsilon\xi).$$

This is identical to the condition obtained in the one-sex model except, here, it relates strictly to the parameters corresponding to the male population. Just as in the one-sex case, whenever the risk compensation term satisfies

$$\xi\epsilon^2 > 1 > \xi\epsilon, \tag{4}$$

the preventive measure has a detrimental effect and increases the reproductive number provided the following holds:

$$d_m\eta_m < \frac{(\mu_m^* + \nu)(\xi\epsilon^2 - 1)}{1 - \xi\epsilon}.$$

This, again, represents an upper bound on the reduction of infection risk corresponding to the diagnosed infected males. Notice that, in this case, the behavior of the diagnosed infected females does not play any role in the outcome of the introduction of preventive measures. This emphasizes the fact that the phenomenon is due to a cumulative effect of high-risk indirect contacts (here represented by un-diagnosed infected males via a female intermediate). The regions where $\mathcal{R}_0^{vd} < \mathcal{R}_0^{v}$ are very similar for the values used in the previous section and they are not reproduced again here.

Remark 3.1. We emphasize that, for both the one-sex and two-sex models, $\mathcal{R}_0^{vd}$ is an increasing function of η, η_f or η_m. The undesired outcome we pointed out in this analysis refers only when comparing the epidemic reproductive numbers before and after implementing preventive measures.

4. Discussion

In this article we studied the effect of risk compensation in an epidemic in which infectious individuals are separated between those who are aware of their infection (diagnosed) and those who do not know this information yet (un-diagnosed). This difference can be significant in infectious diseases for which symptoms appear much later or are milder in their manifestation and easily ignored. HIV is a prime example in the case of STD's.

Table 2
Variables and parameters for model (3).

Symbol	Meaning
S_f	Susceptible females
S_m	Susceptible males without preventive measures
S_{mv}	Susceptible males with preventive measures
I_f	Infected females not diagnosed
I_m	Infected males without protection and not diagnosed
I_{mv}	Infected males with protection and not diagnosed
J_f	Infected females diagnosed
J_m	Infected males diagnosed
β	The per couple birth rate
γ_f, γ_m with $\gamma_f + \gamma_m = 1$	The female and male probability at birth
δ	The rate at which preventive measures are implemented for males
$\bar{\mu}_f = \mu_f + bP$ and $\bar{\mu}_m = \mu_m + bP$	The logistic female and male mortality rates
λ	The infection rate
$0 < \xi \leq 1$	The reduction factor of transmission for protected male individuals
$\epsilon \geq 1$	The risk compensation factor for protected male individuals
$0 < \eta_f \leq 1$ and $0 < \eta_m < 1$	The reduction factor of the transmission rate corresponding to diagnosed female and male infectious individuals
d_f and d_m	The rate at which infectious females and males are diagnosed
α	The disease induced mortality rate of infectious individuals not diagnosed
ν	The disease induced mortality of infectious diagnosed individuals

Thus the risk behavior of undiagnosed individuals is identical to the corresponding susceptible ones. We showed that, if the risk compensation manifests itself in response to a partially effective preventive measure, the cumulative effect from a high risk contact can play an important role: when preventive measures are introduced, the epidemic reproductive number may increase if the infection risk from diagnosed individuals is too low.

Nevertheless, this phenomenon should be interpreted carefully. As mentioned in previous remarks, a low risk behavior does reduce the epidemic reproductive number, all else being equal. The question we focused on here is whether a reduction is observed when we compare $\mathcal{R}_0$ before and after the introduction of preventive measures. It is in this context in which a low risk behavior may cause an increase in $\mathcal{R}_0$. In other words, a low η may cause an increase of $\mathcal{R}_0$ between two relatively *low* values and a high η may cause a decrease of $\mathcal{R}_0$ between two relatively *high* values. Therefore, the main message is to still promote high rates of testing and to encourage diagnosed individuals to avoid infecting others, but to be aware that risk-compensation may act against the benefits derived from these positive behavior traits.

Furthermore, high rates of diagnostic are always beneficial (since (2) is also an upper bound on d). So, if η is too low to avoid an increase in $\mathcal{R}_0$ then a high enough d will counteract the effect and cause the desired reduction in the reproductive number after the implementation of prevention. However, if diagnosed individuals completely avoid infecting others (i.e. $\eta = 0$), then no practical level of the diagnosing rate d can avoid the situation described in this paper.

The promotion for regular testing and treatment of possible infections has been and continues to be a hallmark in any effort of disease prevention. In addition to the individual benefits (early detection means better chances for successful treatment) there is also a moral aspect that plays an important role: diagnosed individuals are expected to take all measures possible in avoiding the spread of the disease. We still believe that encouraging early diagnosis should continue. On the other hand, our results show that the danger of risk compensation goes beyond the direct reduction in the effectiveness of prevention at an individual level. Due to the population dynamics and the subgroups that drive the epidemic, failing to improve the outcome through preventive measures may be more likely to happen at low level of risk behavior in the diagnosed group.

Our result also shows the importance of how and when a certain vaccine or prophylactic measure is advertised. We suggest that extra care should be exercised when the following factors are likely: questionable effectiveness of preventive measures, high likelihood of risk compensation and an already established dependence on testing for disease prevention.

We believe the next step in this research is to expand the efforts already done in measuring and quantifying risk compensation in order to have a sense as to whether situations described in this paper are likely. This remains a difficult process and relies on sociological and psychological research among other things. Even more important, from our perspective, is to measure the discrepancy between perceived and real risk assessments since this discrepancy causes even lower levels of risk compensation to have undesired effects.

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Appendix A. Derivation of the reproductive number for the one-sex model

We use the next generation matrix approach in van den Driessche and Watmough (2002) to compute the epidemic reproductive number. The disease free equilibrium (DFE) of the model (1) is

$$S_1^* = \frac{\beta(\beta-\mu)}{b(\beta+\delta)}, \qquad S_2^* = \frac{\delta(\beta-\mu)}{b(\beta+\delta)},$$
$$I_1^* = 0, \qquad I_2^* = 0, \qquad J^* = 0,$$

which is feasible provided that $\beta > \mu$. The entrance and removal rates into the infectious classes I_1, I_2 and J are

$$\mathcal{F} = \begin{pmatrix} \lambda \dfrac{S_1}{P}(I_1 + \epsilon I_2 + \eta J) \\ \lambda\xi\epsilon \dfrac{S_2}{P}(I_1 + \epsilon I_2 + \eta J) \\ 0 \end{pmatrix},$$

$$\mathcal{V} = \begin{pmatrix} \bar{\mu} I_1 + d I_1 + \alpha I_1 \\ \bar{\mu} I_2 + d I_2 + \alpha I_2 \\ -d(I_1 + I_2) + \bar{\mu} J + \nu J. \end{pmatrix}.$$

The Jacobians evaluated at DFE are

$$F = \begin{pmatrix} \lambda\frac{S_1^*}{P^*} & \lambda\epsilon\frac{S_1^*}{P^*} & \lambda\eta\frac{S_1^*}{P^*} \\ \lambda\xi\epsilon\frac{S_2^*}{P^*} & \lambda\xi\epsilon^2\frac{S_2^*}{P^*} & \lambda\xi\epsilon\eta\frac{S_2^*}{P^*} \\ 0 & 0 & 0 \end{pmatrix},$$

$$V = \begin{pmatrix} \beta+d+\alpha & 0 & 0 \\ 0 & \beta+d+\alpha & 0 \\ -d & -d & \beta+\nu \end{pmatrix}.$$

The epidemic reproductive number is the spectral radius of FV^{-1} which is

$$\mathcal{R}_0^{vd} = \lambda\frac{(\beta+\nu)(\beta+\xi\epsilon^2\delta)+d\eta(\beta+\xi\epsilon\delta)}{(\beta+\delta)(\beta+d+\alpha)(\beta+\nu)}.$$

Appendix B. The derivation of the reproductive number for the two-sex model

In the absence of the disease we can reduce the original model (3) to the following planar system in $F := S_f$ and $M = S_m + S_{mv}$:

$$\begin{cases} F' &= \beta\gamma_f\mathcal{M}(F,M) - \bar{\mu}_f F, \\ M' &= \beta\gamma_m\mathcal{M}(F,M) - \bar{\mu}_m M. \end{cases} \tag{B.1}$$

This model was fully analyzed in a more general form in Maxin and Sega (2013) and we only provide here the main result which relates to the existence and the stability condition of the disease free equilibrium of (3). From Maxin and Sega (2013), (B.1) admits a unique positive interior steady state (F^*, M^*) that is globally stable if and only if

$$\mathcal{M}\left(\frac{\beta\gamma_f}{\mu_f}, \frac{\beta\gamma_m}{\mu_m}\right) > 1.$$

Assuming the condition above, the disease free equilibrium is

$$S_f^* := F^*, \qquad S_m^* = \frac{\mu_m^* M^*}{\delta+\mu_m^*}, \qquad S_{mv}^* = \frac{\delta M^*}{\delta+\mu_m^*},$$
$$I_f^* = 0, \qquad I_m^* = 0, \qquad I_{mv}^* = 0, \qquad J_f^* = 0, \qquad J_m^* = 0.$$

We now proceed to compute the epidemic reproductive number using the next-generation matrix method described in van den Driessche and Watmough (2002). The entrance and removal rates into the infectious classes are:

$$\mathcal{F} = \begin{pmatrix} \lambda\mathcal{M}(F,M)\frac{S_f}{F}\frac{I_m+\epsilon I_{mv}+\eta_m J_m}{M} \\ \lambda\mathcal{M}(F,M)\frac{S_m}{M}\frac{I_f+\eta_f J_f}{F} \\ \lambda\xi\epsilon\mathcal{M}(F,M)\frac{S_{mv}}{M}\frac{I_f+\eta_f J_f}{F} \\ 0 \\ 0 \end{pmatrix},$$

$$\mathcal{V} = \begin{pmatrix} \bar{\mu}_f I_f + \alpha I_f + d_f I_f \\ \bar{\mu}_m I_m + \alpha I_m + d_m I_m \\ \bar{\mu}_m I_{mv} + \alpha I_{mv} + d_m I_{mv} \\ -d_f I_f + \bar{\mu}_f J_f + \nu J_f \\ -d_m(I_m + I_{mv}) + \bar{\mu}_m J_m + \nu J_m. \end{pmatrix}.$$

The Jacobians evaluated at DFE are

$$F = \begin{pmatrix} 0 & \lambda T^* S_f^* & \lambda\epsilon T^* S_f^* & 0 & \lambda\eta_m T^* S_f^* \\ \lambda T^* S_m^* & 0 & 0 & \lambda\eta_f T^* S_m^* & 0 \\ \lambda\xi\epsilon T^* S_{mv}^* & 0 & 0 & \lambda\xi\epsilon T^* S_{mv}^* & 0 \\ 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \end{pmatrix},$$

$$V = \begin{pmatrix} \mu_f^*+\alpha+d_f & 0 & 0 & 0 & 0 \\ 0 & \mu_m^*+\alpha+d_m & 0 & 0 & 0 \\ 0 & 0 & \mu_m^*+\alpha+d_m & 0 & 0 \\ -d_f & 0 & 0 & \mu_f^*+\nu & 0 \\ 0 & -d_m & -d_m & 0 & \mu_m^*+\nu \end{pmatrix},$$

where $T^* := \frac{1}{F^*M^*}\mathcal{M}(F^*, M^*)$. The epidemic reproductive number is the spectral radius of FV^{-1} which, in this case, is

$$\mathcal{R}_0^{vd} = \lambda T^* \times \sqrt{F^*M^*\frac{(\mu_f^*+d_f\eta_f+\nu)[(\mu_m^*+\nu)(\mu_m^*+\delta\xi\epsilon^2)+d_m\eta_m(\mu_m^*+\delta\xi\epsilon)]}{(\mu_m^*+\delta)(\mu_f^*+\nu)(\mu_m^*+\nu)(\mu_f^*+\alpha+d_f)(\mu_m^*+\alpha+d_m)}}.$$

Appendix C. The derivation of the reproductive number for a one-sex model with constant recruitment and mortality rates

The model shown below is similar to (1). The state variables and the parameters have the same meaning while K denotes the constant recruitment rate in the population of susceptibles. Furthermore, unlike (1), the mortality rates are constants.

$$\begin{cases} S_1' &= K - \lambda S_1(I_1+\epsilon I_2+\eta J) - \mu S_1 - \delta S_1, \\ S_2' &= \delta S_1 - \lambda\xi\epsilon S_2(I_1+\epsilon I_2+\eta J) - \mu S_2, \\ I_1' &= \lambda S_1(I_1+\epsilon I_2+\eta J) - \mu I_1 - dI_1 - \alpha I_1, \\ I_2' &= \lambda\xi\epsilon S_2(I_1+\epsilon I_2+\eta J) - \mu I_2 - dI_2 - \alpha I_2, \\ J' &= d(I_1+I_2) - \mu J - \nu J, \end{cases} \tag{C.1}$$

where $P = S_1 + S_2 + I_1 + I_2 + J$ is the total population size.

The disease free equilibrium is

$$S_1^* := \frac{K}{\mu+\delta}, \qquad S_2^* := \frac{\delta K}{\mu(\mu+\delta)}, \qquad I_1^* = I_2^* = J^* := 0.$$

Using the same next generation matrix approach as in the previous appendixes we obtain the following epidemic reproductive number

$$\mathcal{R}_0^{vd} = \frac{\lambda K[(\mu+\nu)(\mu+\xi\epsilon^2\delta)+d\eta(\mu+\xi\epsilon\delta)]}{\mu(\mu+\delta)(\mu+d+\alpha)(\mu+\nu)}.$$

In the absence of preventive measures ($\delta = 0$) this becomes

$$R_0^d = \frac{\lambda K(\mu+\nu+d\eta)}{\mu(\mu+\nu)(\mu+d+\alpha)}.$$

After a straightforward computation we see that the same thresholds are obtained whenever we verify under what conditions the introduction of preventive measures increases the reproductive number, i.e.

$\mathcal{R}_0^{vd} > \mathcal{R}_0^d$ is equivalent to $(\xi\epsilon^2 - 1)(\mu+\nu) > d\eta(1-\xi\epsilon)$.

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