

# The Role of Disease-Specific Infectivity and Number of Disease Exposures on Long-Term Effectiveness of the Latex Condom

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**Background:** Condom use is a primary prevention strategy for sexually transmitted infections (STIs). Consistent condom use substantially reduces the risk of HIV infection. Similar levels of effectiveness for reducing the risk of other STIs have not been established. Differences in disease-specific infectivity and the number of exposures to an infected partner may explain differences in condom effectiveness.

**Goal:** The goal was to examine the impact of differing infectivities and increasing numbers of exposures on theoretical condom effectiveness.

**Study Design:** Mathematical modeling using estimated disease-specific infectivities and condom failure rates.

**Results:** Condom effectiveness decreases as disease-specific infectivity and the number of exposures to infection risk increase.

**Conclusions:** Condom effectiveness for decreasing STI risk is influenced by disease infectivity and the number of exposures. Generalizations from studies of relatively uninfected STIs to highly infectious STIs or from short-term studies to longer-term situations will overestimate condom effectiveness.

THE ORIGIN OF THE MALE CONDOM is unknown, but its use for preventing pregnancy and reducing the risk of sexually transmitted infection (STI) goes back for centuries.<sup>1</sup> Since the introduction of hormonal contraception in the mid 20th century, the condom's role as a primary method of birth control has declined. Multiple factors have precipitated this decline, including the superiority of hormonal contraceptives for pregnancy prevention,<sup>2</sup> the ease of use, and the greater degree of female control afforded by these newer methods.

Hormonal contraceptives, however, offer no appreciable protection from STIs, and with the appearance of HIV as a major health concern in the 1980s, prevention of HIV infection became a personal and public health priority. Currently, use of the male latex condom is the primary strategy

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for HIV and STI prevention in sexually active individuals worldwide. Condoms are promoted as a highly effective means of preventing HIV and other STIs. With the exception of HIV,<sup>3</sup> however, research demonstrating the effectiveness of condoms for STI prevention is lacking. Most research indicates that condoms are less effective for preventing transmission of non-HIV STIs than for preventing HIV infection.<sup>4-7</sup> A few studies have revealed high levels of condom effectiveness for STI prevention.<sup>8,9</sup> Such studies, however, typically involve a short follow-up period and a small number of potential exposures.

Condoms are likely to be less effective in preventing infections that are spread by direct skin contact than infections spread by the exchange of body fluids, since infection can occur on areas of the skin not covered by the condom.<sup>10,11</sup> An acceptable explanation for observed differences in condom effectiveness for prevention of infections that are transmitted by body fluids (such as HIV, gonorrhea, and chlamydia), however, has not been offered. Such findings are often dismissed as being due to inconsistent/incorrect condom use<sup>7</sup> or suboptimal study designs.

But if condoms can fail even with consistent use, differences in disease-specific infectivity may explain the noted variation in disease-specific condom effectiveness. For vaginal intercourse, estimates of per-episode infectivity range from a low of 0.001 for HIV<sup>12,13</sup> (because actual HIV infectivity has been shown to vary with viral load, 0.001 represents an estimated average) to approximately 0.20 for gonorrhea in men,<sup>8</sup> 0.30 for syphilis,<sup>14,15</sup> 0.50 for gonorrhea in women and for human papillomavirus,<sup>16,17</sup> and 0.70 for chancroid.<sup>18</sup> Intuitively, differences in STI infectivity should be mitigated by the use of relative risk measurements to quantify condom effectiveness; greater infectivity would convey higher risk to both condom users and condom non-users. However, as the number of exposures (episodes of intercourse with an infected partner) increases, an interac-

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tion between disease-specific infectivity and number of episodes of intercourse may explain many of the reported differences in the condom's ability to reduce the risk of infection. This article examines the impact of varying disease infectivity and increasing number of exposures on condom effectiveness.

## Methods

Infection risks and relative risks were calculated for increasing numbers of coital exposures to an infected partner, on the basis of hypothetical per-episode infectivities and a consistent condom breakage and slippage rate. Infectivity estimates were chosen to approximate the infectivities of HIV (0.001), gonorrhea (0.20 for men and 0.50 for women), syphilis (0.30), and chancroid (0.70).

We chose a condom breakage and complete slippage rate of 0.03 for our calculations. This estimate of condom breakage and slippage risk is based on numerous studies showing that combined clinical breakage and complete slippage rates usually range between two and four percent in the general population.<sup>19</sup> In fact, rates of condom breakage and combined (partial and complete) slippage are higher, but only complete slippage is included in our calculations because it is more likely to result in significant infection risk.

### Calculating Infection Risk

The probability ( $q$ ) that a nonuser of condoms will remain uninfected after  $n$  episodes of sexual intercourse with an infected partner can be calculated as

$$q = (1 - inf)^n.$$

where  $inf$  represents the per-episode infectivity of the disease in question (that is, the likelihood of becoming infected during a single act of sexual intercourse with an infected person).<sup>8</sup> This is the familiar Bernoulli process that has been used to model HIV infection risk and condom effectiveness for HIV prevention.<sup>20,21</sup>

The probability ( $p_{n \text{ nonuser}}$ ) that a condom nonuser will become infected after  $n$  episodes of intercourse with an infected partner is calculated as

$$p_{n \text{ nonuser}} = 1 - q \text{ or } 1 - (1 - inf)^n.$$

If one assumes that disease transmission occurs only when the condom breaks or slips, the probability of infection for a condom user depends on (1) the *number of episodes* of intercourse with an infected partner; (2) the *infectivity* of the STI; and (3) the *per-episode probability (f) of condom failure (breakage or slippage)*. If the risk of becoming infected from coital exposure during which the condom breaks or slips off is equivalent to the infection risk if condoms are not used, the probability ( $p_{n \text{ condom user}}$ ) that

a condom user will become infected after  $n$  episodes of intercourse with an infected partner is:

$$p_{n \text{ condom user}} = 1 - [1 - (f)(inf)]^n.$$

This approach assumes that each coital act carries with it the same risk of infection: the risk of condom breakage or slippage times the risk of infection when condom breakage or slippage occurs. Given our assumption that new infections occur only when condoms break or slip off, it would perhaps be more appropriate to think of infection risk as being zero when condom breakage or slippage does not occur and being equal to the infectivity ( $inf$ ) when breakage or slippage does occur. Calculations based on these assumptions can be made using a binomial distribution to quantify the probability of achieving any number ( $a$ ) of condom breakages/slippages, given a number ( $n$ ) of episodes of sexual intercourse. The Bernoulli process can then be used to calculate infection risk, given each number ( $a$ ) of condom breakages/slippages. The weighted average of the risk for each potential number of breakages/slippages represents the total infection risk.

The results of the two approaches are identical for every exposure scenario. Because the calculations can be carried out much more efficiently with the strictly logarithmic approach, it is used exclusively throughout the remainder of this article.

### Calculating Relative Risk

Condom effectiveness is normally expressed as relative risk—that is, the ratio of infection risk in condom users to infection risk in nonusers. It is calculated as

$$RR = (\text{infection risk in condom user}) / (\text{infection risk in nonuser})$$

A relative risk of 1.0 indicates that an intervention has no protective effect on infection risk. A relative risk below 1.0 demonstrates some protective effect. This protective effect becomes stronger as the relative risk approaches zero. A relative risk  $>1.0$  would indicate an increased risk due to condom use (not a medically plausible outcome).

## Results

### Infection Risk With Condom Nonuse

As illustrated in Figure 1, the calculated risk of infection when condoms are not used increases as the number of coital exposures to an infected individual increases. The rate of increase is very rapid for infections with high infectivities. Infection risk approaches 100% after 10 unprotected exposures for diseases with infectivities of 0.50 or 0.70. The probability of infection when a disease's infectivity is 0.2 or

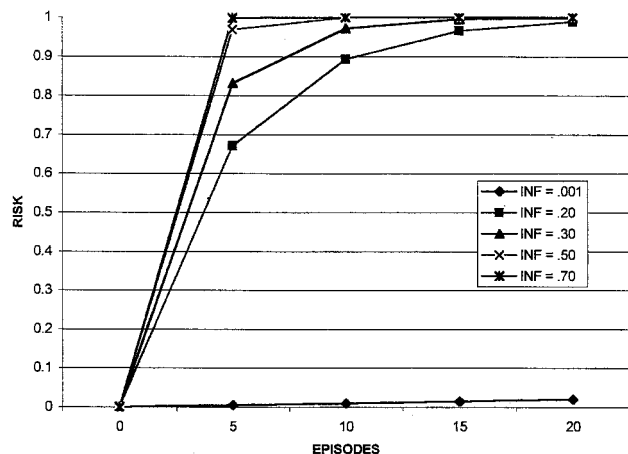


Fig. 1. Infection risk for nonusers of condoms, given differing levels of infectivity and increasing exposures to an infected partner.

0.3 approaches 100% with approximately 30 unprotected exposures.

For a disease with an infectivity of 0.001, infection risk rises much less rapidly. After 30 unprotected exposures, the probability of infection is 3%. After 100 exposures, the probability of infection is 9.5%.

#### Infection Risk with Condom Use

Calculated infection risk when condoms are used is illustrated in Figure 2. As with condom nonuse, infection risk increases as the number of exposures increases. Again, this increase occurs more rapidly when the infectivity of the STI is greater. Initially, the risk of infection when condoms are used is low. However, as the number of exposures increases,

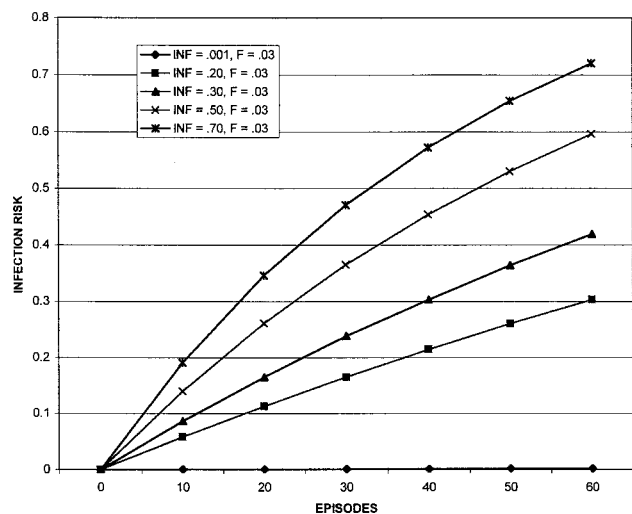


Fig. 2. Infection risk for condom users, given differing levels of infectivity and increasing exposures to an infected partner.

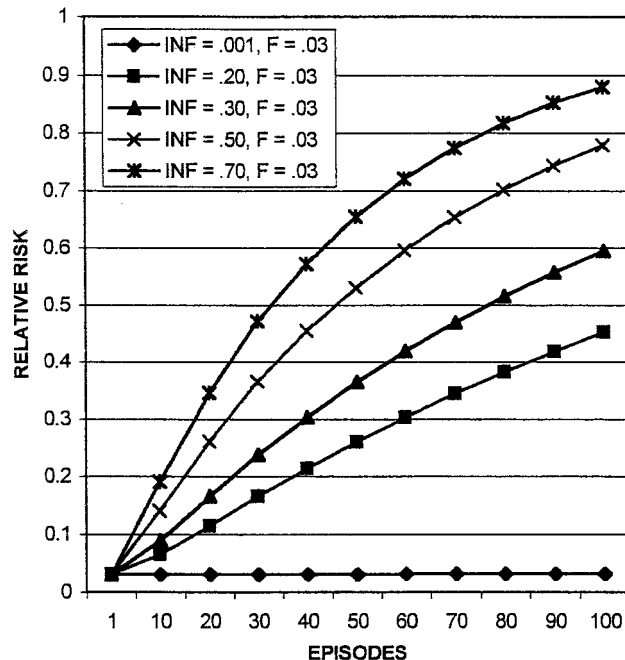


Fig. 3. Relative risk of infection (condom users versus nonusers), by infectivity and number of exposures.

the probability of infection can become quite high, especially for highly infective STIs.

When infectivity is 0.70, 10 exposures with a condom confer a 19% risk of infection. With 50 exposures, the risk is 65%, and with 100 exposures the risk is 88%. When infectivity is 0.50, 10 exposures confer a 14% risk of infection and 50 exposures confer a 53% risk. After 10 exposures, when infectivity equals 0.30, the probability of infection is 8.6%. After 50 exposures, infection risk is 36%, and after 100 exposures, it is 59.5%. After 10 exposures using a condom when infectivity is 0.20, the probability of infection is 5.9%. With 100 exposures, the risk is 45.2%. When infectivity is very low (0.001), the risk after 10 exposures using a condom is only 0.03%, and after 100 exposures it is just 0.3%.

#### Relative Risk of Infection With Condom Use (Condom Effectiveness)

Although absolute infection risk is important and may be especially useful for counseling patients, condom effectiveness is typically expressed as a relative risk. The calculated impact of different levels of disease infectivity and increasing numbers of exposures on the relative risk of infection with condom use is depicted in Figure 3. Relative risk (and therefore Figure 3) is a function of the risk of infection in condom users (Figure 2) and the risk of infection in nonusers (Figure 1).

When infectivity is 0.70, the calculated relative risk is

low for the first few episodes of intercourse, but then it increases to 0.19 with 10 exposures, 0.65 for 50 exposures, and 0.88 for 100 exposures. This means that when infectivity is 0.70, an individual who always uses a condom and who has 100 exposures to an infected sexual partner is 88% as likely to have been infected as someone with the same number of exposures to the same infection who never uses a condom.

When infectivity is 0.50, the calculated relative risk is 0.14 after 10 exposures, 0.53 after 50 acts, and 0.78 after 100. For an infectivity of 0.30, relative risk is calculated as 0.09 with 10 exposures but increases to 0.36 after 50 exposures and 0.60 after 100 exposures. An infectivity of 0.20 results in a calculated relative risk of 0.066 after 10 exposures, 0.26 after 50 acts, and 0.45 after 100.

When infectivity is very low, relative risk remains low despite large numbers of exposures. When infectivity equals 0.001, the calculated relative risk after 10 and 100 exposures is 0.030 and 0.032, respectively.

### Discussion

One's risk of infection increases with increasing numbers of *unprotected* sexual acts with an infected partner. Similarly, our calculations indicate that one's risk of infection increases with increasing numbers of *condom-protected* sexual exposures. Based on our models, the *relative risk* of infection in condom users also increases with increasing numbers of exposures. Our models also indicate that relative risk is highly associated with STI-specific infectivity. Relative risk increases (less risk reduction) more rapidly with increasing exposure in more infectious diseases. These findings are in agreement with previously published models of HIV risk using a similar approach.<sup>20,21</sup>

Because of the impact of increasing infectivity on relative risk, it is inappropriate to generalize condom effectiveness from infections with low infectivity (such as HIV) to infections with high infectivity (such as gonorrhea) or vice versa. Clinical research is needed to determine whether differences in disease-specific infectivity will actually result in different relative risks.

Our findings also call into question the validity of studies that attempt to measure the effectiveness of condoms in preventing STI transmission but fail to measure the number of sexual acts (exposures) of participants. Future studies of condom effectiveness must measure the number of sexual acts in which study subjects participate. Follow-up periods should be of adequate duration to allow a substantial number of exposures to occur. Similarly, because of the impact of multiple episodes of intercourse on condom effectiveness, results from studies that follow participants for a short period of time (a few episodes of intercourse, or even a few weeks of follow-up) cannot be used to estimate long-term condom effectiveness.

In addition, general statements about condoms being “80%, 90%, or 99% effective” for preventing STIs must be avoided. For such statements to be accurate, the number of exposures to a particular STI must be specified. Generic estimates of effectiveness fail to account for differences in relative risk based on different numbers of exposures and differing disease-specific infectivities.

In this article, we use the term *effectiveness* to describe the ability of condoms, when used consistently, to reduce STI risk. *Efficacy* may be a preferable term to describe this measure, because our models predict condom performance under “ideal” conditions, i.e., consistent use. However, efficacy in the context of condom research typically refers to *both* consistent and correct use. Some portion of condom breakage and slippage is likely due to user error and not mechanical failure. Since the breakage and slippage rates used in our models probably include some user error, we have chosen *effectiveness*. However, we acknowledge that our estimates may more closely approximate condom *efficacy* than *effectiveness* in actual use.

Our calculations rest on a number of assumptions. First, we assume that all episodes of intercourse occur with an infected partner. This is an appropriate assumption because, although most bacterial STIs will eventually resolve even without treatment, most STIs persist (and continue to be infectious) for several months to a year or longer if not successfully treated. Some viral STIs persist for the remainder of the infected person's life. The upper range of sexual encounters with an infected individual used in our calculations (100) is well within the realm of possibilities for the number of episodes of vaginal intercourse over a 1-year period. Lower levels of sexual exposure (1–50 acts of intercourse) are reasonable for a sexual relationship that is measured in days, weeks, or (possibly) months. Assuming that all episodes of sex occur with an infected partner allows us to isolate the effectiveness of the latex condom itself, which is the real purpose of this article.

Second, we assume that disease transmission with condom users occurs only if the condom breaks or slips off. This assumption is based on the body of research that shows condoms, in the absence of manufacturing defects, to be effective barriers for infectious agents. The assumption should hold true for STIs spread by the exchange of body fluids, such as HIV and gonorrhea. For those infections spread by skin-to-skin contact, such as chancroid, syphilis, genital herpes, and infection due to human papillomavirus, infection that occurs outside the area covered by an intact condom can be transmitted even if a condom is used and does not break or slip. Therefore, condom failure rates for these infections are likely higher than would be predicted by our models.

We also assume a consistent rate of condom breakage and complete slippage during intercourse. The rate we have used in our calculations (0.03) falls within the usual range of

reported breakage and complete slippage rates. It is likely that breakage and slippage rates are not randomly distributed throughout the population of condom users, and previous research shows that breakage and slippage rates may decrease with increasing condom use experience.<sup>22</sup> For the most experienced condom users (prostitutes, for example), our breakage and slippage assumptions are likely to overestimate condom failure. However, for the least experienced condom users (adolescents, for example), the risk of breakage and slippage is likely to be underestimated. To compensate for overestimation or underestimation, we performed calculations using three different and plausible rates for condom breakage and slippage: 0.01, 0.03, and 0.05. The results of each calculation (available from the authors upon request) were similar to those described in this article, although actual calculated infection risks and relative risks were different for each probability of breakage/slippage.

Fourth, we assume that STI infectivity is constant on a per-act basis. In actuality, most estimates of infectivity are averages. In the case of HIV, evidence suggests that infectivity varies substantially over the course of the disease, as the viral load rises and falls.<sup>13</sup> However, calculated infectivities of HIV are consistently an order of magnitude or more below those of most other STIs. Our assumption of constant infectivity should not detract from the purpose of this paper: to examine the impact of differences in infectivity and number of exposures on condom effectiveness.

Last, we assume that each event of breakage or complete slippage exposes one to the same risk of infection. Furthermore, we assume that this risk is the same as the risk with condom nonuse. These assumptions are likely not entirely correct. Whereas some breakages/slippages are likely to expose the individual to an infection risk identical to the risk of unprotected intercourse, others probably involve less risk. However, limiting our considerations to clinical breakage (breakage during intercourse) and complete slippage (slippage in which the condom completely slips off the penis) rather than all breakage and slippage will minimize this problem. Adjusting for remaining differences in the amount of infection risk due to breakage and slippage would have the same effect as using different estimates for breakage and slippage rates. The slopes of the curves would change, but the underlying phenomenon (increasing relative risk due to increasing infectivity and number of acts of intercourse) would not be changed.

If every individual within an entire population of sexually active individuals used latex condoms consistently and correctly, the incidence and eventually the prevalence of many STDs would decline. This is the perspective of those interested in population health: find a prevention strategy that reduces the risk of an adverse health consequence and implement that strategy throughout the population. Universal implementation of even a marginally effective prevention strategy should ultimately reduce disease prevalence.

But the goals of the clinician often differ from the goals of the population health advocate. Clinicians advocate for individuals and are primarily interested in the personal health of individual patients. Nonetheless, counseling individual patients about prevention strategies requires knowledge of the principles and measures of population health. Which STIs are most prevalent in the patient's community of sexual partners? Are the prevalent diseases transmitted by infected body fluids or by contact with infected skin? How contagious are the prevalent diseases? How experienced in using condoms is this patient? All of these factors will impact the patient's per-episode risk of contracting a new STI.

Finally, the clinician must keep in mind the concept of cumulative risk. As the number of uses of any imperfect prevention intervention increases, the cumulative potential for intervention failure also increases. If the intervention fails, disease may occur. Therefore, disease screening and other early recognition and treatment programs are important components of clinical practice. The findings of this report, particularly when coupled with the knowledge that many STIs are clinically asymptomatic, should encourage the clinician to screen appropriately for STIs with high prevalence in the community—even in patients who report they consistently and correctly use condoms.

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