# Introduction

Papua New Guinea (PNG) is a small developing island nation just north of Australia, with a population of 7.3 million people. It has an HIV prevalence of 1 in 2000, the highest in the Pacific and five times the prevalence in Australia. HIV is an uncurable sexually transmitted virus, which attacks and eventually destroys T cells, a key part of a person’s immune system. AIDS, the consequent lack of T cells, is the main cause of death at PNG’s main hospital. Reducing the number of people who acquire HIV is thus a priority for some policymakers.

There are certain sexually transmitted infections (STIs) which make a person more likely to transmit or acquire HIV. The STIs for which this effect is strongest are called *ulcerating STIs*, because they produce ulcers or sores. These ulcers are weak points in the skin, which is body’s main barrier against pathogens including HIV. Ulcerating STIs increase a person’s likelihood of acquiring or receiving HIV by 2 to 5 times, where this number is called the *HIV cofactor*. The main ulcerating STIs in Papua New Guinea are herpes simplex virus 2, syphilis or chancroid. These STIs are very common in PNG. For example, syphilis occurs in one in 20 men, one in 12 women and 1 in 3 female sex workers (FSW). By way of comparison, syphilis occurs in 1 in 14,000 people in Australia, predominantly among MSM. Syphilis, like many STIs, is curable if treated early enough. Unlike many STIs, syphilis is eventually fatal in 40% of cases if left untreated, but in common with many STIs syphilis has no symptoms other than its effect on HIV in a third of cases. Thus, lowering levels of syphilis is of direct benefit to the population, but is also a possible method of lowering the incidence, or number of new cases, of HIV.

For some STIs, such as chlamydia, there is no quick and cheap test that can be administered in the field. Testing requires laboratory equipment which is not available at all clinics in PNG. In Australia, this delay in receiving results would not matter, since people would simply make another appointment and receive treatment shortly after they were notified of a positive diagnosis.

In PNG this would be less practical. Only 50% of the population has a mobile SIM, and many people might be unwilling to receive notification about STI results using a shared phone. Large numbers of people diagnosed may not receive their diagnosis, or not receive it for a long time. Moreover, many people have to travel long distances to reach a clinic. Some people also find the clinic environment hostile, and feel judged by the people there, and so want to minimise their visits to clinics. Thus, people who have received a positive diagnosis, may also never return for treatment, or may only return later, after they have had a chance to infect others.

An alternative treatment program for chlamydia involves treating people immediately when they come into a clinic, without waiting for test results. This is called *periodic presumptive treatment* (PPT). PPT is typically provided to high-risk sub-populations, especially FSW. If enough people are reached, significant feedback will develop between the prevalence among people receiving treatment and the prevalence among their partners, and will also filter out into the wider community. This intervention has been used against chlamydia in several large-scale trials, and has proven effective.

A chlamydia PPT program could be easily and cheaply combined with a PPT program for STIs such as syphilis which increase the risk of HIV transmission. PPT would then become a combined intervention targeting both curable STIs, and HIV. The effect on HIV is the focus of this paper.

In this paper, we develop a dynamical deterministic compartmental homogenous mixing model for a curable STI with a high HIV cofactor. We calibrate the steady state of our model to the current prevalence of syphilis in PNG. Although there is enough data to model specific diseases differently, we assume that there is only one STI with a relevant HIV cofactor which our intervention will affect. We assume that all other STIs with a non-trivial HIV cofactor have the same cofactor as syphilis, and that their prevalence will remain constant during our intervention. We add these prevalences together, and input them into an existing HIV model to forecast the impact on HIV of a PPT intervention into an STI with a high HIV cofactor.

Our STI model uses two non-interacting regions and four sub-populations. The HIV model divides into rural and urban regions, so our STI model does the same. Baseline STI prevalences are all higher in the rural region than in the urban region. Also following the HIV model, our STI model divides the population into female sex workers, general females, general males, and men who have sex with men and women (MSMW). Following our HIV model, we merge men who have sex exclusively with men into this category. PNG also has low levels of injecting drug use, so we do not model this population. There is little research targeting MSMW in PNG, making it hard to model them as a sub-population, so we assume their STI prevalence is halfway between those of general males and general females. Our baseline STI prevalences were as shown in Table 1.

Table : Prevalences used as steady state for STI model

|  |  |  |
| --- | --- | --- |
|  |  |  |
|  |  |  |
|  |  |  |
|  |  |  |
|  |  |  |

Our STI model was a SIPS model, where people could be susceptible, infected, or protected by PPT. We designed it such that that when PPT coverage was 0, the model collapsed to a SIS model. We let and denote the proportions of sub-population who are susceptible and infected, respectively, where the subscript is one of for FSW, for general females, for general males, or for MSMW. The equations are identical in structure for FSW, general females and general males, and slightly different for MSMW. Note that since and are proportions, we have . Our model equations for FSW are presented in Equation 1.

Equation : Main equations for STI prevalence for FSW when PPT coverage is 0

Infected FSW stop being infected at the constant rate . This accounts for existing treatment for the STI. A proportion of mothers will seek prevention of mother-to-child transmission of their STIs, which reduces the proportion of infected newborns below the proportion of mothers infected. There is also a chance that an infected and untreated mother will not pass on her STI to an unborn child. Moreover, an uninfected child is more likely to survive birth than an infected child. Thus, new entrants to the population are less infected than existing members of the population. Thus, loss among the population reduces the proportion of the population infected. This effect is also included in .

Susceptible FSW became infected at a variable rate , which depended on the infection rate among males. is the maximum rate at which FSW would be infected if all of their partners were infected. The infection rate is simply this infection rate times the probability that a randomly selected partner of an FSW is infected. We assume that general males and MSMW have the same levels of sexual partnerships with general females and FSW, so is just the proportion of males who are MSMW.

These equations are the same for general males and general females. For general females, we again use the partner infection probability . For general males, we replace and with and , and we replace with , the probability that a random sex act by a general male will be with a FSW, which must be adjusted for FSW performing more sex acts with general males per person than general females perform. For MSMW, we took a slightly different approach. We added the probability that an MSMW will acquire an STI from a female, which we assumed is the same as the probability that a general male will acquire an STI, to the probability that an MSMW will acquire an STI from an MSMW. Thus, took the form shown in Equation 2, below.

Equation : Infection rate equation for MSMW

We generalised from these equations when we added PPT.

We modelled our PPT intervention keeping in mind two main policy decisions. Firstly, we allowed the coverage of FSW to vary. We considered coverage two scenarios, so that the intervention reached either 50% or 75% of FSW. Secondly, we allowed the average frequency of visits to vary. We again considered two scenarios, so that FSW received treatment either once per month, or once per two months. We assumed that people receiving PPT would receive it at random times, whenever they visited a hospital or travelled to a town which had one. We assumed that these people’s risk behaviour such as condom use would neither decrease due to any associated education program, or decrease due to people feeling safe, and that people would not enter or exit the program apart from at the beginning, where a random sample of people would enter.

To model PPT being provided to FSW, we split the FSW sub-populations into FSW who would receive PPT, and FSW who would not. For the FSW who would never receive PPT, we used the same SIS dynamics as Equation 1. For the FSW who did receive treatment, we modified these dynamics to an SIPS model, adding a “protected by PPT” state . Our new model is described by

Equation : prevalence equations for STI for FSW receiving PPT

Susceptible and infected people now receive treatment at a constant rate . In reality, people are likely to seek treatment more when they know they have just engaged in risky behaviour, or when it is a longer times since their last visit. People in PNG have quite high levels of knowledge about risky behaviour. Thus, the rate of PPT should be higher among infected people and lower among susceptible people. However, we could not observe this effect size, so we disregarded it. Protected people lose their protection at a constant rate . We calculated the infection rate for males using the weighted average of the infection levels among FSW receiving treatment and FSW not receiving treatment.

We ignore protection provided by any treatment other than our PPT intervention.

|  |  |
| --- | --- |
|  |  |
| Proportion of people in urban areas | 14% |
| Proportion of women who are FSW |  |
| * In urban areas | 5% |
| * In rural areas | 1% |
| Proportion of men who are MSMW |  |
| * In urban areas | 6% |
| * In rural areas | 4% |
|  |  |