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Prevalence and control of sexually transmitted infections in sub-Saharan Africa

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Submitted in part fulfilment of the requirements for the degree of
Doctor of Philosophy of Imperial College London, August 2025

Statement of Originality

I declare that the work presented in this thesis is my own, completed under the supervision of Dr Jeffrey Imai-Eaton, Dr Anne Cori, and Prof Marie-Claude Boily. Several chapters are adapted from published manuscripts, to which collaborators contributed as acknowledged below. All methods, analyses, figures, and text included in this thesis are entirely my own. All secondary sources have been cited.

Chapter 2 has been published as: **Michalow J**, Hall L, Rowley J, Anderson RL, Hayre Q, Chico RM, Edun O, Knight J, Kuchukhidze S, Reed DM, Stevens O, Walters MW, Peters RPH, Cori A, Boily MC, Imai-Eaton JW. *Prevalence of chlamydia, gonorrhoea, and trichomoniasis among male and female general populations in sub-Saharan Africa from 2000 to 2024: a systematic review and meta-regression analysis.* eClinicalMedicine. 2025 5;83:103210. Co-authors supported the review and selection of studies for inclusion in the systematic review, and provided feedback on the analysis and manuscript.

Chapter 3 has been published as: **Michalow J**, Walters MK, Edun O, Wybrant M, Davies B, Kufa T, Mathega T, Chabata ST, Cowan FM, Cori A, Boily MC, Imai-Eaton JW. *Aetiology of vaginal discharge, urethral discharge, and genital ulcer in sub-Saharan Africa: A systematic review and meta-regression.* PLoS Medicine. 2024 5;21(5):e1004385. Access to unpublished data used in this chapter was facilitated by Dr Tendesayi Kufa and Thabitha Mathega at the National Institute for Communicable Diseases in South Africa, and by Prof Frances M. Cowan and Dr Sungai Chabata at the Centre for Sexual Health and HIV/AIDS Research in Zimbabwe. Co-authors supported the review and selection of published studies for inclusion in the systematic review, contributed to interpreting results, and provided feedback on the manuscript.

Chapter 5 has been published as: **Michalow J**, Jahn A, Cori A, Boily MC, Chimpandule T, Mbiriyawanda S, Ozituosauka W, Nyirenda R, Imai-Eaton JW. *Burden and Trends of Symptomatic Sexually Transmitted Infections in Malawi From 2000 to 2021: Comparative Analysis of Survey and Case Report Data.* Sexually Transmitted Diseases. 2024 3;51(3):206-13. The syndromic surveillance data analysed in this chapter were collected and managed by the Malawi Ministry of Health. Co-authors contributed to interpreting findings and provided feedback on the manuscript.

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Acknowledgements

I am deeply grateful for the opportunity to undertake this PhD, a journey enriched by the mentorship provided by my supervisors: Dr Jeffrey Imai-Eaton, Dr Anne Cori, and Professor Marie-Claude Boily. Thank you for your gentle guidance, patience, and good humour. It has truly been a privilege to learn from each of you.

Thank you to my fellow researchers at DIDE for creating a vibrant and collaborative intellectual environment that has fostered so much growth. Special thanks to the colleagues and now friends who graciously tolerated repeated enlistment in my systematic reviews. Thanks also to Jane Rowley at the WHO for so generously sharing her extensive knowledge of the field.

I was fortunate to receive the Imperial College President's PhD Scholarship, which made this work possible.

Thank you to my mother, the Ehrlich clan, and my dear friends for your boundless support and encouragement. I appreciate your continued efforts to understand what I do, even if I haven't made it very easy to follow.

And to Roi, my person, thank you for walking (and laughing) with me every step of the way.

Abstract

Sexually transmitted infections (STIs) are a major yet often overlooked public health challenge in sub-Saharan Africa (SSA), associated with severe reproductive and neonatal sequelae, chronic complications, and an increased risk of HIV acquisition in the presence of co-infection. The WHO has set ambitious global incidence reduction targets for 2030, but progress in SSA is hindered by high rates of asymptomatic infection, limited diagnostic capacity, weak surveillance infrastructure, and fragmented health systems. This thesis combines systematic reviews, data synthesis, and mathematical modelling to generate evidence that strengthens STI surveillance and control in SSA. It estimates sub-regional trends in the prevalence of curable STIs among general populations, and characterises the evolving aetiologies of vaginal discharge, urethral discharge, and genital ulcer syndromes. It then investigates the utility of self-reported symptoms and treatment-seeking behaviours from population-representative surveys for quantifying the relative STI burden and trends, and monitoring treatment coverage. This includes assessing the consistency of these survey measures with national aetiologic prevalence estimates across SSA and with sub-national syndromic case diagnoses from health facility surveillance in Malawi. Finally, it evaluates how a limited supply of gonorrhoea point-of-care tests could be allocated across different testing strategies and target populations to maximise reductions in population-level incidence and morbidity in Kenya. Taken together, the findings highlight persistent surveillance gaps that undermine burden estimation and progress monitoring against global targets, identify considerable epidemiologic heterogeneity requiring more targeted programme strategies, and demonstrate that novel diagnostic technologies have potential to mitigate the high burden despite resource constraints.

Abbreviations

AGYW	Adolescent girls and young women
AIS	AIDS Indicator Survey
AMR	Antimicrobial resistance
ANC	Antenatal care
BV	Bacterial vaginosis
CA	*Candida albicans*
CFSW	Clients of female sex workers
CPP	Chronic pelvic pain
CS	*Candida* species
CT	*Chlamydia trachomatis*
DFA	Direct fluorescent antibody
DHS	Demographic and Health Surveys
EA	Eastern Africa
EDS	Epididymitis
ELISA	Enzyme-linked immunosorbent assay
EP	Ectopic pregnancy
FGM	Female genital mutilation
FP	Family planning
FSW	Female sex workers
GBD	Global Burden of Disease
GU	Genital ulcer
GUD	Genital ulcer disease
HD	*Haemophilus ducreyi*
HIV	Human immunodeficiency virus
HPV	Human papillomavirus
HSV	Herpes simplex virus
HSV-1	Herpes simplex virus type 1
HSV-2	Herpes simplex virus type 2
LB	Live birth
LBW	Low birth weight
LGV	Lymphogranuloma venereum
MG	*Mycoplasma genitalium*
MSM	Men who have sex with men
NAAT	Nucleic acid amplification test
NG	*Neisseria gonorrhoeae*
NP	Neonatal pneumonia
ON	Ophthalmia neonatorum
OPD	Outpatient department
PID	Pelvic inflammatory disease
PHC	Primary healthcare
POC	Point-of-care
POCT	Point-of-care test
QALY	Quality-adjusted life year
ROB	Risk of bias
RTI	Reproductive tract infection
SA	Southern Africa
SSA	Sub-Saharan Africa
STI	Sexually transmitted infection
TFI	Tubal factor infertility
TP	*Treponema pallidum*
TPP	Target product profile
TV	*Trichomonas vaginalis*
UD	Urethral discharge
UDS	Urethral discharge syndrome
VD	Vaginal discharge
VDS	Vaginal discharge syndrome
WCA	Western and Central Africa
WHO	World Health Organization
WPP	World Population Prospects

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Chapter 1

Background

1.1 Introduction

Sexually transmitted infections (STIs) are a major public health challenge affecting sexual and reproductive health worldwide, with more than one million new infections acquired daily.[1] Of the many pathogens transmitted sexually, several have garnered particular global attention due to their health burden. These include the bacterial infections *Chlamydia trachomatis* (*C. trachomatis*, chlamydia), *Neisseria gonorrhoeae* (*N. gonorrhoeae*, gonorrhoea), and *Treponema pallidum* (*T. pallidum*, syphilis); the parasitic infection *Trichomonas vaginalis* (*T. vaginalis*, trichomoniasis); and the viral infections human immunodeficiency virus (HIV), herpes simplex virus type 2 (HSV-2), human papillomavirus (HPV), hepatitis B virus (a major cause of viral hepatitis), and mpox (formerly known as monkeypox). If left untreated, these infections can lead to severe long-term outcomes, including infertility, chronic pelvic pain, pregnancy complications, and congenital infections, regardless of whether symptoms are present.[1] Psychological, social, and economic consequences of STIs can substantially affect quality of life. A disproportionate share of this global burden is in sub-Saharan Africa (SSA).[2]

Public health responses have remained inadequate in many parts of the world, despite the severity of these infections. Although HIV has been the focus of unprecedented global investment and surveillance, other STIs have received comparatively limited attention in global health policy. In 2016, the World Health Organization (WHO) set ambitious global targets to be achieved by 2030: reduce the incidence of gonorrhoea and syphilis by 90%, reduce the incidence of chlamydia and trichomoniasis by 50%, lower congenital syphilis cases to fewer than 50 per 100 000 live births, and achieve 90% HPV vaccination coverage among girls under 15 years of age.[2] However, progress has been slow, and these targets are

unlikely to be met without substantial acceleration.[3] The WHO Global Health Sector Strategies on HIV, viral hepatitis, and STIs for 2022–2030 calls for integrated, cross-cutting action to strengthen prevention, screening, case management, and treatment.[1]

This thesis predominantly focuses on five STIs, which collectively account for a substantial burden of disease and the majority of symptomatic cases managed in SSA: chlamydia, gonorrhoea, trichomoniasis, syphilis, and HSV-2. While HIV remains a significant public health concern, it is not a primary focus, given extensive research already dedicated to its prevention and control. Associations between HIV and other STIs are however considered throughout.

This chapter introduces the epidemiology of the five focal STIs, the key interventions currently available for their control, and the programmatic and surveillance responses implemented to date in SSA.

1.2 Epidemiology

1.2.1 Regional burden

According to WHO estimates in 2016, SSA accounts for approximately 40% of the global STI burden.[2] In a WHO meta-analysis, estimated adult prevalence (proportion of individuals with active symptomatic or asymptomatic infection) in 2020 for the African region was 4.7% (3.4–6.5%) for chlamydia, 1.4% (0.9–2.0%) for gonorrhoea, 6.6% (4.8–9.0%) for trichomoniasis, and 1.7% (1.4–2.0%) for syphilis.[4] HSV-2, an incurable infection, affects over one-third of adults in the region, with prevalence estimated at 37.3% (34.9–39.7%) in a 2021 meta-analysis.[5] These infections are sustained within the general population, with higher prevalence among women than men, and a particularly high burden among adolescent girls and young women (AGYW) and key populations such as female sex workers (FSW) and men who have sex with men (MSM).[1] Prevalence is also higher among people living with HIV, reflecting shared behavioural and structural vulnerabilities, as well as biological interactions through which some STIs increase the risk of both acquiring and transmitting HIV.[1]

National prevalence estimates from the Global Burden of Disease (GBD) study suggest considerable geographic heterogeneity across SSA (Figure 1.1). Prevalence of curable STIs tends to be higher in Southern and Eastern Africa than in Western and Central Africa. HSV-2 follows a similar distribution, though prevalence is also high in several Central African countries.[5,6] These sub-regional estimates are however limited by sparse case reporting and a lack of population-representative prevalence studies.

Additionally, there are weaknesses in the methods and assumptions underpinning both WHO and GBD estimates, which use statistical modelling techniques to synthesise prevalence data (Section 1.5).

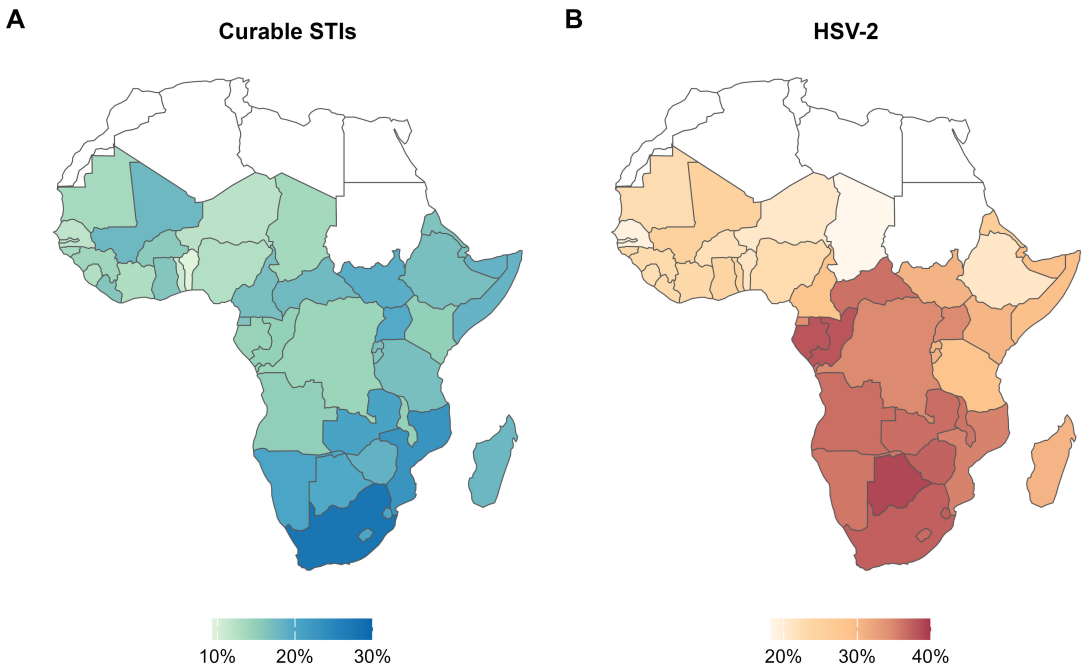


Figure 1.1: Estimated national prevalence of sexually transmitted infections in SSA, 2020
National estimates from the Global Burden of Disease study for (A) the combined prevalence of chlamydia, gonorrhoea, trichomoniasis, and syphilis; and (B) the prevalence of HSV-2, among adults 15-49 years in SSA.[6]

1.2.2 Natural history

Natural history describes the course of infection from acquisition to spontaneous clearance, and is shaped by pathogen characteristics, host biology, and behavioural factors. These features influence transmission patterns and the likelihood of infections being diagnosed, affecting the interpretation of surveillance data and the design of control strategies. Much of the current understanding of natural history is drawn from empirical studies and mathematical models used to infer individual-level parameters, though both approaches are limited by study design, diagnostic test performance, and methodological assumptions.

Transmission mechanisms

STIs are primarily transmitted through sexual contact, including vaginal, anal, and oral sex. Transmission occurs through genital secretions, skin-to-skin contact, or blood, depending on the pathogen, with efficiency varying by organism, anatomical site, and biological sex.

Chlamydia, gonorrhoea, and trichomoniasis are transmitted via direct exposure to infected genital secretions. Gonorrhoea is the most efficiently transmitted of the three, with estimated per-act probabilities of 20–50% for male-to-female and 10–30% for female-to-male intercourse (Table 1.1).[7–13] Transmission probabilities are lower for chlamydia, estimated at 15–25% per act for male-to-female and 10–20% for female-to-male intercourse,[7,12–14] while transmission of trichomoniasis is markedly asymmetrical, ranging between 14–25% per-act for male-to-female and 2-5% for female-to-male.[7,15] Anatomical site plays an important role in susceptibility. The rectal mucosa is more vulnerable to chlamydia and gonorrhoea than vaginal or oral tissue, due to its thinner epithelium and limited local immune response.[16,17] This increases the probability of transmission during receptive anal intercourse and, alongside behavioural and structural factors, contributes to the high prevalence of chlamydia and gonorrhoea among MSM. In contrast, trichomoniasis is largely confined to the urogenital tract.[18]

Ulcerative infections, such as syphilis and HSV-2, are transmitted through skin-to-skin contact. Syphilis is typically acquired via direct contact with infectious lesions and has a transmission efficiency comparable to that of chlamydia.[7,19] HSV-2 is acquired via contact with infected genital or peri-anal areas, even in the absence of visible lesions. Although its per-act transmission probability is relatively low (<3% when averaged across symptomatic and asymptomatic episodes), lifelong infection, frequent reactivation, and asymptomatic viral shedding sustain its widespread transmission.[20,21]

Host factors influence susceptibility to infection. Anatomical and physiological differences contribute to greater male-to-female transmission efficiency for most STIs, primarily due to the larger mucosal surface area and prolonged exposure to infectious secretions in the female genital tract.[22] For trichomoniasis, this asymmetry is especially pronounced due to enhanced colonisation in the vagina and faster clearance from the male urethra.[23] Cervical ectopy, a benign condition in which glandular cells extend onto the outer cervix, is more common among adolescents and younger women and increases susceptibility to chlamydia,[24] although its association with gonorrhoea remains inconclusive.[24] Other host factors, including co-infections, vaginal microbiota composition, hormonal contraception, and circumcision status, can further modulate transmission.

Progression and symptomatology

Chlamydia, gonorrhoea, and trichomoniasis are typically acute, single-phase infections. Following acquisition, each progresses through incubation, active infection, and eventual resolution through treatment or natural clearance (Table 1.1).[25] Incubation periods range from several days to one month, depending

Table 1.1: Natural history and characteristics of common sexually transmitted infections

Category	Chlamydia	Gonorrhoea	Trichomoniasis	Syphilis	HSV-2
Pathogen					
Name	*Chlamydia trachomatis*	*Neisseria gonorrhoeae*	*Trichomonas vaginalis*	*Treponema pallidum*	*Herpes simplex virus 2*
Type	Bacteria	Bacteria	Parasite	Bacteria	Virus
Transmission probability per sex act*					
Male-to-Female	0.15 - 0.25 [7,12–14]	0.20 - 0.50 [7,10–13]	0.14 - 0.25 [7,15]	0.15 - 0.30 [7,56–58]	0.0009 - 0.03 [7,21,59]
Female-to-Male	0.10 - 0.20 [7,12–14]	0.10 - 0.30 [7–9,11–13]	0.02 - 0.05 [7,15]	0.10 - 0.20 [7,56–58]	0.00015 - 0.006 [7,21,59]
Duration per stage of infection (weeks)‡					
Incubation§	1.5 - 4 [7,12,53,60]	1 - 1.5 [10,26,27,32,61]	0.5 - 2 [28,29]	3 - 4.5 [7,62]	0.5 - 1.5 [63]
Single-phase					
• Active infection	60 - 90 [7,12,14,42,43]	20 - 24 [7,12,14,32,40,41]	M: 10 - 15 [7,15,34,44,64] F: 150 - 250 [7,15,34,64]	-	-
• Immunity	8 - 12 [49–53]	Limited evidence [45,52,54]	Limited evidence [28,52,55]	-	-
Multi-phase					
• Primary	-	-	-	5 - 8 [7,62]	2 - 3 [7,21,65,66]
• Secondary	-	-	-	12 - 18 [7,62]	-
• Reactivation	-	-	-	-	1 - 2 [21,66]
• Latent	-	-	-	500 - 700 [7,57,62]	Lifelong [65,66]
Symptomatic proportion					
Male	0.20 - 0.50 [7,12,14,31]	0.45 - 0.90 [7,11,12,30–32]	0.25 - 0.50 [7,15,34,44]	> 0.90 [67†]	0.10 - 0.25 [7,68]
Female	0.15 - 0.30 [7,12,14,31,33]	0.20 - 0.60 [7,11,12,30–33]	0.15 - 0.40 [7,15,33,34]	> 0.90 [67†]	0.10 - 0.25 [7,68]
Symptoms					
Male	Mucoid or watery urethral discharge, dysuria, epididymitis	Purulent urethral discharge, dysuria, epididymo-orchitis	Scant frothy or clear urethral discharge, dysuria, irritation	Chancre with rash, lymphadenopathy, condylomata lata	Anogenital vesicular lesions and ulcerations
Female	Mucopurulent cervical discharge, dysuria, intermenstrual bleeding, pelvic pain	Mucopurulent cervical discharge, dysuria, intermenstrual bleeding, pelvic pain	Frothy malodorous vaginal discharge, vulvovaginal irritation, dysuria	Chancre with rash, lymphadenopathy, condylomata lata	Anogenital vesicular lesions and ulcerations

*Transmission probability applies to infectious stages only. †Most syphilis infections develop a chancre during the primary stage of infection, although it may go unnoticed. ‡Durations reflect average infection length assuming natural progression without treatment. For most STIs, these durations approximate the course of asymptomatic infection in the absence of care-seeking or diagnosis. §Time from initial infection to exhibiting symptoms. F: female, M: male. Table compiled based on a narrative review of published epidemiologic evidence and transmission modelling studies.

on the pathogen and site of infection.[7,10,26–29] A large proportion of infections remain asymptomatic, particularly among females. Symptomatic presentation occurs in approximately 45-90% of males and 20-60% of females for gonorrhoea,[7,11,12,30–33] 20-50% and 15-30% for chlamydia,[7,12,14,31,33] 25-40% and 15-40% for trichomoniasis,[7,15,33,34] respectively. In males, common symptoms include urethral discharge and dysuria (painful urination) following urethral infection.[35–37] In females, chlamydia and gonorrhoea infect the cervix and can cause vaginal discharge, dysuria, intermenstrual bleeding, and lower abdominal pain due to ascending infection,[35,36] while trichomoniasis, which primarily affects the vagina, is more often associated with malodorous discharge and vulvovaginal irritation.[37] Symptoms may not persist for the full duration of infection and can become asymptomatic over time due to spontaneous immune modulation or partially effective treatment, though empirical evidence for such transitions is limited.[38,39] The duration of untreated infection varies by pathogen. Gonorrhoea typically resolves within 4–6 months,[7,32,40,41] chlamydia can persist for over a year,[7,42,43] and trichomoniasis typically clears within 2–4 months in men but can last up to 3–5 years in women due to sustained vaginal colonisation.[7,15,34,44] Immunological protection following natural resolution appears limited, with high reinfection rates for all three pathogens suggesting that any immunity is partial or short-lived.[45–48] Evidence of partial immunity is best supported for chlamydia, where previous infection may confer protection against subsequent infections by reducing bacterial load, shortening infection duration, or partially preventing reinfection.[49–53] Evidence of partial immunity is more limited for gonorrhoea and trichomoniasis.[28,45,52,54,55] All three infections respond rapidly to effective treatment (Section 1.3.2).[25]

Syphilis and HSV-2 have more complex, multi-stage natural histories. Syphilis progresses through primary, secondary, latent, and tertiary stages of infection (Table 1.1).[7,62,67,69] The primary stage, marked by a painless chancre at the site of infection, develops approximately one month post-exposure and resolves spontaneously 1-2 months thereafter. Secondary syphilis is characterised by systemic symptoms, such as rash, mucocutaneous lesions, and lymphadenopathy. These too resolve spontaneously within 3-5 months, though recurrences occur in roughly 25% of cases.[62] The infection then enters a latency, during which individuals are asymptomatic but remain infected. Early latency (within one year of infection) is more infectious than late latency, due to the more frequent recurrence of secondary symptoms.[62] HSV-2 establishes lifelong infection with intermittent reactivation.[7,21,70] After a short incubation period,[63] primary infection can cause painful genital or peri-anal ulcers that last 2-3 weeks,[7,21,65,66] often accompanied by systemic symptoms such as fever, malaise, and inguinal lymphadenopathy. However, 75–90% of cases are asymptomatic or unrecognised.[7] The virus remains latent in sacral nerve ganglia and periodically reactivates, causing symptomatic or asymptomatic

viral shedding. Shedding episodes occur frequently in the first year post-infection, up to 4-8 times per year,[21,70] and decline over time, but infectiousness persists for life. Antiviral therapy can reduce shedding frequency and lower transmission risk, but cannot eliminate viral latency.[21,70]

Complications and sequelae

Untreated STIs are associated with adverse reproductive, perinatal, systemic, and psychological consequences, many of which disproportionately affect women. In females, chlamydia and gonorrhoea are the leading infectious causes of pelvic inflammatory disease (PID), a syndrome that can result in chronic pelvic pain (CPP), ectopic pregnancy (EP), and tubal factor infertility (TFI), though non-infectious causes also contribute to these outcomes.[35,36] In males, both infections can cause epididymitis, prostatitis, and, less commonly, urethral stricture, all of which may contribute to infertility or persistent urogenital symptoms.[35,36]

Several infections have adverse pregnancy and neonatal outcomes. Chlamydia, gonorrhoea, and trichomoniasis are associated with preterm birth, premature rupture of membranes, and low birth weight, while chlamydia and gonorrhoea are also implicated in stillbirth.[71–73] During delivery, neonates exposed to chlamydia or gonorrhoea may develop conjunctivitis or pneumonia.[71,74] HSV-2 exposure during delivery can result in neonatal herpes, which has a case fatality rate exceeding 50% without treatment and leads to long-term neurological impairment among survivors.[75] Syphilis can be transmitted transplacentally at any stage of pregnancy and may cause miscarriage, stillbirth, preterm birth, or congenital infection with severe multisystem complications and developmental consequences.[76]

Syphilis and HSV-2 are also associated with longer-term systemic outcomes. If untreated, syphilis may progress to tertiary disease involving neurological, cardiovascular, or gummatous (granulomatous inflammation causing local tissue necrosis) manifestations.[67] HSV-2 can cause chronic neuropathic pain in affected dermatomes and, in rare cases, neurological complications such as aseptic meningitis or sacral radiculopathy.[70] Ulcerative infections also increase the risk of HIV acquisition by approximately three-fold,[77,78] and increase HIV transmissibility via increased genital shedding.[79] Non-ulcerative STIs, such as including chlamydia and gonorrhoea, may also enhance HIV susceptibility via mucosal inflammation,[80,81] while their role in HIV transmission is less well established.[79]

Beyond physical outcomes, STIs can profoundly affect mental wellbeing. Individuals commonly experience stigma, anxiety, and relationship difficulties that diminish quality of life, particularly in the context of chronic or recurrent infections, such as syphilis and HSV-2.[82]

1.2.3 Transmission dynamics

The sustained transmission of STIs within a population is governed by both biological and social processes. A widely used measure of transmission potential is the basic reproductive number (R_0), defined as the average number of secondary infections caused by one infected individual in a fully susceptible population. For STIs, it can be expressed as $R_0 = \beta cD$, where β is the probability of transmission per sex act, c is the rate of sexual contact, and D is the average duration of infectiousness.[83] The value of R_0 determines whether an epidemic will occur, the rate of spread of the epidemic, the endemic equilibrium level, and the intensity of intervention required to bring the infection under control.[25] While R_0 describes transmission potential in a fully susceptible population, the effective reproductive number (R_t) reflects transmission at a given point in time.

Transmission probability (β) varies by pathogen, anatomical site, and host susceptibility (Section 1.2.2). Biological factors such as the presence of ulcers, mucosal vulnerability, and co-infections can increase transmission efficiency.

Sexual contact rate (c) reflects individual behaviour within broader sexual network structures and the social contexts in which they exist. These networks are shaped by patterns of partnership formation and dissolution. In most populations, the majority of individuals have few sexual partners, while a small proportion account for a disproportionate share of total sex acts and associated transmission.[84,85] Risk is influenced not only by the number of partners, but also by an individual's position within the broader network. Individuals with high centrality within sexual networks are more likely to acquire and transmit infection.[86,87] Those occupying bridging positions, such as clients of sex workers, may also play a key role in transmission by linking otherwise disconnected groups,[88,89] although their influence is context-dependent and often overlaps with other network features such as partner number or centrality.[90] Transmission risk also accumulates through repeated exposures. Long-term partnerships with frequent sex acts may contribute equally or more to transmission than several casual partnerships with fewer sex acts, particularly for asymptomatic infections of longer duration.[91]

Duration of infectiousness (D) is influenced by both pathogen biology and treatment coverage. Many STIs are asymptomatic or mildly symptomatic, leading to undiagnosed and untreated infections that extend the infectious period and allow sustained transmission within sexual networks.[25]

Together, these interacting factors determine the potential for sustained transmission within a population ($R_t > 1$).[83] Acute infections with short durations (e.g. gonorrhoea and chlamydia) typically require

high rates of partner change and concentrated sexual networks to maintain transmission.[25,92] In contrast, viral infections with latency and reactivation cycles (e.g. HSV-2) can persist even in sparse networks, due to lifelong duration with asymptomatic shedding.[21] As a result, bacterial and protozoal STIs tend to cluster in higher-risk groups, while viral STIs can achieve high prevalence in the general population, particularly in settings with limited treatment coverage.[93]

1.3 Strategies for control

Effective STI control requires reducing the effective reproductive number (R_t) below one, such that each infection generates fewer than one secondary infection on average. This can be achieved by lowering the transmission probability (β) through biomedical or behavioural prevention interventions (e.g. condom use, male circumcision), reducing the sexual contact rate (c) through behaviour change, or shortening the duration of infectiousness (D) through timely diagnosis and treatment.

In SSA, STI control efforts have primarily focused on preventive measures and the treatment of symptomatic infections. Diagnostic and screening interventions remain limited, predominantly due to resource constraints, but also due to implementation challenges and mixed evidence of effectiveness. Nonetheless, there is growing recognition that expanded diagnostic capacity is essential to improving case detection and strengthening control efforts across the region.

1.3.1 Prevention

STI prevention has historically received limited dedicated investment in SSA, with most interventions delivered through HIV programmes rather than STI-specific initiatives.[2] Early optimism that STI control could reduce HIV incidence waned after trials showed limited impact in generalised epidemics,[94–96] contributing to declining donor interest. As a result, and amidst competing health system priorities, comprehensive STI prevention strategies have not consistently been prioritised.[2]

Available prevention approaches span biomedical, behavioural, and structural interventions. Biomedical interventions form the foundation of STI prevention. Condom promotion, the most widely implemented strategy, substantially reduces the transmission of most STIs but offers only partial protection against ulcerative infections spread through skin-to-skin contact.[97,98] Voluntary medical male circumcision, introduced primarily for HIV prevention, reduces male acquisition of HSV-2 by 25-35% and syphilis by 30-40%, while indirectly protecting female partners against HSV-2, trichomoniasis, and bacterial vaginosis.[99] Periodic presumptive treatment among FSW has achieved short-term reductions in

bacterial STI incidence,[100,101] but is no longer widely recommended due to concerns about antimicrobial resistance (AMR). Partner notification and treatment can reduce reinfection rates,[102] but is generally underutilised due to legal, social, and logistical barriers.[103–105] Behavioural interventions, including sexual health education, counselling, and mass communication campaigns, have improved knowledge of STI transmission and prevention, increased condom use, and enhanced engagement with health services.[106–109] Structural approaches have sought to expand access to prevention and care, such as through integrating STI services into HIV and primary healthcare platforms, decentralising delivery through mobile clinics and community health workers, and establishing school-based or youth-friendly sexual health services.[110–112] However, evidence that behavioural and structural interventions reduce the incidence of STIs other than HIV remains limited, with effectiveness varying across interventions, populations, and settings.[113]

1.3.2 Syndromic management

Syndromic case management was introduced by the WHO in 1984 to address limitations in access to laboratory infrastructure and point-of-care (POC) diagnostics for STIs in low-resource settings.[114] It has since remained the primary strategy for STI management in most of SSA. The approach uses simplified clinical algorithms to identify consistent groups of signs and symptoms and to treat the most probable causative infections, sometimes incorporating risk assessment to guide treatment when symptoms are non-specific. Benefits include timely treatment of symptomatic individuals, particularly in settings where follow-up is limited.[115] It requires minimal infrastructure, basic clinical training, and is feasible across all levels of the health system.[116] Implementation has contributed to declines in acutely symptomatic infections.[116,117] For example, modelling in South Africa and Uganda suggests that syndromic management was associated with the near-elimination of chancroid and substantial reductions in syphilis prevalence, both of which are predominantly symptomatic.[118,119] Despite these operational advantages, syndromic management has several critical limitations. Most notably, it fails to detect and treat asymptomatic infections, particularly impacting cervical infections in women and extragenital infections in MSM.[120,121] The diagnostic accuracy of syndromic management algorithms is generally suboptimal, despite efforts to account for evolving STI epidemiology.[116] This leads to unnecessary, incorrect, or missed treatment, with repeated use of broad-spectrum antibiotics potentially contributing to AMR.[122]

In 2021, the WHO released new guidelines for symptomatic STI management, the first revision since 2003.[117] Global evidence on aetiology, diagnostic performance, and treatment efficacy was utilised to

revise the algorithms for five key syndromes: urethral discharge, vaginal discharge, lower abdominal pain, genital ulcer disease, and anorectal discharge. The guidelines recommend integrating diagnostic testing into syndromic algorithms where feasible to improve accuracy, and adapting global algorithms to reflect local epidemiology and optimise appropriate treatment coverage.[117] The three most common syndromes are outlined below, as they account for the majority of symptomatic cases in SSA.

Urethral discharge syndrome (UDS) typically presents with discharge, dysuria, and itching, and is most commonly caused by gonorrhoea, followed by chlamydia, trichomoniasis, and increasingly *Mycoplasma genitalium* (*M. genitalium*).[117,123] Gonococcal urethritis typically produces a more profuse and purulent discharge, while non-gonococcal infections produce a scant or clear discharge.[124] These clinical characteristics are considered alongside history and risk assessment, but are generally non-specific and inconsistently assessed.[124] Syndromic algorithms perform relatively well, with estimated sensitivity and specificity of 85.2% (95% CI: 80.5-89%) and 66.5% (62.4-70.5%) for detecting gonorrhoea or chlamydia, and 69.6% (CI: 55.2-80.9%) and 96.0% (93.1-97.8%) for gonorrhoea alone.[125] Empirical treatment generally targets gonorrhoea and chlamydia with ceftriaxone and either azithromycin or doxycycline. *M. genitalium* is addressed in guidance for treatment failure, though standard regimens may be ineffective due to rising resistance.[117,126,127]

Vaginal discharge syndrome (VDS) presents with abnormal discharge, often accompanied by odour, vulvar pruritus, or dysuria. The most common cause is vaginitis due to bacterial vaginosis or *Candida albicans* (*C. albicans*), which are reproductive tract infections not classified as sexually transmitted, as well as trichomoniasis. Cervical infections with chlamydia, gonorrhoea, and *M. genitalium* are less frequently implicated due to their asymptomatic nature. Discharge characteristics vary by aetiology: bacterial vaginosis typically causes a thin, grey-white discharge with a fishy odour; candidiasis presents with thick, curdy white discharge and itching; trichomoniasis can cause a frothy, yellow-green discharge, and cervical infections may result in mucopurulent discharge.[128] These clinical features are of limited diagnostic utility.[128] The VDS algorithm has performed poorly since its introduction.[116,120,129–132] The algorithm based on clinical history and risk assessment has estimated sensitivity and specificity of 56.2% (95% CI: 54.5-57.9%) and 71.0% (69.4-72.6%) for bacterial vaginosis or trichomoniasis, and 27.9% (24.7-31.1%) and 57.0% (56.1-58.0%) for gonorrhoea or chlamydia.[116] Including speculum examination modestly improves performance, but is often infeasible in primary care.[116] Diagnostic accuracy is further reduced among symptomatic pregnant women, in whom candidiasis may be a more frequent cause of discharge.[133] Attempts to improve diagnostic performance using risk scores that

incorporate demographic and behavioural factors to estimate the likelihood of cervical infection have shown limited benefit, as these tools have often been applied without local validation.[116,134] Empirical treatment for VDS typically includes antifungals for candidiasis, metronidazole for bacterial vaginosis and trichomoniasis, and broad-spectrum antibiotics for cervical infections.[117]

Genital ulcer disease (GUD) presents with genital or perianal ulcers that vary in size, number, and depth, and may be accompanied by pain, itching, or inguinal lymphadenopathy. HSV-2 is the predominant aetiology, having replaced *Haemophilus ducreyi* (*H. ducreyi*; chancroid) and syphilis as the leading cause of GUD in the early 2000s.[5,117,135] Other causes include HSV type 1 (HSV-1), and lymphogranuloma venereum (LGV) due to *C. trachomatis* serovars L1-L3.[117] Herpes and chancroid typically cause multiple painful ulcers, whereas syphilis and LGV more often cause solitary, painless lesions.[136] However, these distinctions are unreliable, as ulcer appearance varies with co-infection, secondary bacterial colonisation, or partial treatment.[136] Syndromic algorithms perform poorly, with estimated sensitivity and specificity of 43.5% (95% CI: 26.2-62.4%) and 88.0% (67.0-96.3%) for herpes, 52.8% (23.0-80.7%) and 72.1% (28.0-94.5%) for syphilis, and 71.9% (45.9-88.5%) and 53.1% (36.6-68.9%) for chancroid.[137,138] Performance is further undermined by slow adoption of updated guidance for HSV-2 in national protocols.[139] Empirical treatment includes benzathine penicillin for syphilis, ceftriaxone for chancroid, and acyclovir for herpes,[117] though acyclovir is often omitted from national guidelines and unavailable in practice.

1.3.3 Diagnostic testing

Given the limitations of syndromic management, there is increasing emphasis on expanding access to aetiologic testing to improve diagnosis and consequent treatment coverage, antimicrobial stewardship, and surveillance. A range of diagnostic technologies exists for most pathogens, but their use in SSA remains largely restricted to research, sentinel surveillance, or periodic prevalence studies due to cost, infrastructure, and operational barriers (Table 1.2).

Microscopy is a low-cost, rapid diagnostic method that is useful in settings with basic laboratory equipment. Gram-stained smears have high sensitivity (80-90%) for detecting *N. gonorrhoeae* in men with urethral discharge, but substantially lower performance in women, asymptomatic individuals, and at extragenital sites.[140] Wet-mount microscopy is frequently used for *T. vaginalis* and *C. albicans*, though sensitivity is low in women (50-60%) and even lower in men (40-50%) due to rapid loss of organism motility, low pathogen load, and visual confusion with host cells.[140–143] Darkfield microscopy

enables direct visualisation of *T. pallidum* in ulcer exudates and was historically used to diagnose primary syphilis. It offers high sensitivity and specificity, but requires immediate specimen processing, specialised equipment, and is not recommended for oral or rectal lesions due to the presence of non-pathogenic treponemes.[140,144] Interpretation of microscopy is often subjective and depends on adequate training, particularly for more specialised techniques such as darkfield. While microscopy can support presumptive diagnosis and same-day treatment in specific contexts, its diagnostic performance is generally suboptimal and has largely been superseded by more accurate methods.[140,144]

Culture was historically the gold standard for diagnosing several pathogens, including *N. gonorrhoeae*, *C. trachomatis*, *T. vaginalis*, and HSV.[140,144,145] Culture for *N. gonorrhoeae* remains essential for AMR surveillance and offers high sensitivity (80-90%) and specificity (∼100%) for genital specimens, although performance is reduced for extragenital sites.[140,146] It requires selective media, controlled incubation, timely specimen handling, and trained personnel, which together limit accessibility and ease of use, despite relatively low cost.[140,144] Culture for *T. vaginalis* is substantially more sensitive (70-80%) than wet-mount microscopy and can be performed using simplified systems such as InPouch TV (BioMed Diagnostics), which allow direct visualisation of motile organisms without slide preparation.[143,147] However, these still require incubation and are rarely used outside research or specialised diagnostic settings. *C. trachomatis* and HSV can also be cultured, but only in specialised laboratories due to requiring advanced infrastructure and technical expertise.[144] Culture has largely been replaced by molecular methods for routine diagnosis.[146]

Rapid antigen tests detect pathogen-specific proteins, most commonly using lateral flow immunochromatographic assays. These tests are designed for POC use, offering fast results (typically within 30 minutes), minimal infrastructure requirements, and straightforward visual interpretation. The most widely used antigen-based test is for *T. vaginalis*,[148] which has moderate sensitivity (70-80%) compared to molecular methods.[140,141,144] Antigen-based rapid tests for *C. trachomatis* and *N. gonorrhoeae* perform poorly, particularly for asymptomatic infections, and are not recommended by WHO for clinical diagnosis.[140,141]

Serological tests detect pathogen-specific antibodies and are primarily used to diagnose syphilis and HSV. For syphilis, diagnosis typically involves a non-treponemal assay to detect active infection and monitor treatment response, followed by a treponemal assay to confirm exposure. Treponemal antibodies persist for life and cannot distinguish past from current infection, while non-treponemal titres decline with effective treatment.[140,149,150] Both tests may be negative in early primary syphilis,

Table 1.2: Diagnostic methods for common pathogens

Method	Specimen type	Sensitivity	Specificity	Affordability	Speed	Accessibility	Ease of use
N. gonorrhoeae							
Microscopy (gram stain)	Urethral/cervical swab	High (male), Low (female)	Very high	High	Rapid	High	High
Culture	Urethral/cervical/vaginal swab	High	Very high	Moderate	Next day	Moderate	Moderate
Rapid antigen tests	Urethral/cervical/vaginal swab	Low	High	Moderate-High	Rapid	Very high	Very high
NAAT	Urine, urethral/cervical/vaginal swab	Very high	Very high	Low	Next day	Low	Moderate
GeneXpert	Urine, urethral/cervical/vaginal swab	Very high	Very high	Low	Same day	Moderate	High
C. trachomatis							
Culture	Urethral/cervical swab	Moderate	Very high	Moderate	Multiple days	Low	Low
Rapid antigen tests	Urethral/cervical/vaginal swab	Low	High	Moderate-High	Rapid	Very high	Very high
NAAT	Urine, urethral/cervical/vaginal swab	Very high	Very high	Low	Next day	Low	Moderate
GeneXpert	Urine, urethral/cervical/vaginal swab	Very high	Very high	Low	Same day	Moderate	High
T. vaginalis							
Wet-mount microscopy	Urethral/vaginal swab	Low	Very high	Very high	Rapid	High	High
Culture	Urethral/vaginal swab	Moderate	Very high	Moderate	Multiple days	Moderate	Moderate
Rapid antigen tests	Urethral/vaginal swab	Moderate	High	Moderate-High	Rapid	Very high	Very high
NAAT	Urine, urethral/vaginal swab	Very high	Very high	Low	Next day	Low	Moderate
GeneXpert	Urine, urethral/vaginal swab	Very high	Very high	Low	Same day	Moderate	High
T. pallidum							
Darkfield microscopy	Lesion exudate	High	High	High	Rapid	Moderate	Low
Treponemal tests*	Serum/plasma	Very high	Very high	Moderate	Same day	Moderate	Moderate
Non-treponemal tests	Serum/plasma	Very high	High	High	Same day	Moderate	Moderate
Rapid dual tests†	Whole blood/serum/plasma	High	High	Moderate	Rapid	Very high	Very high
NAAT	Lesion swab/blood	Very high	Very high	Low	Next day	Low	Moderate
GeneXpert	Lesion swab/blood	Very high	Very high	Low	Same day	Moderate	High
HSV types 1 or 2							
Culture	Lesion swab	Moderate	Very high	Low	Multiple days	Low	Low
Type-specific serology*	Serum/plasma	High	High	Moderate	Same day	Moderate	Low
NAAT	Lesion swab	Very high	Very high	Low	Next day	Low	Moderate
GeneXpert	Lesion swab	Very high	Very high	Low	Same day	Moderate	Moderate

Sensitivity and specificity: Low (≤70%), Moderate (70-85%), High (85-95%), Very high (≥95%). Affordability: Low (expensive reagents and/or sophisticated equipment), Moderate (consumables and reagents), High (basic consumables). Speed: Rapid (<30 min), Same day (few hours), Next day (1-2 days), Multiple days (>2 days). Accessibility: Low (specialised infrastructure), Moderate (standard laboratory equipment), High (basic tools or bench-top instruments), Very high (no equipment required). Ease of use: Low (specialised expertise), Moderate (laboratory or technical training), High (basic training), Very high (minimal or no training). *Confirms exposure, but cannot distinguish active from past infection. †Combines treponemal and non-treponemal detection. NAAT: Nucleic acid amplification test. Content collated and adapted from WHO guidelines.[140,141,144]

limiting their utility at initial presentation of genital ulceration.[149] These laboratory-based assays are relatively affordable, require standard equipment, and rely on trained personnel. Rapid treponemal and dual treponemal/non-treponemal tests require no equipment and minimal training.[140,151] While diagnostic performance is lower than laboratory-based assays, their simplicity and accessibility make them well suited to resource-limited settings.[151,152] Most HSV serological tests are laboratory-based and require trained personnel. They can differentiate between HSV-1 and HSV-2, but do not reliably detect recent infection.[144,153] Serology for *C. trachomatis* was used historically, but is no longer recommended due to poor diagnostic performance, and the inability to distinguish past from current infection.[140,145,150]

Nucleic acid amplification tests (NAAT) are the gold standard for diagnosing *C. trachomatis*, *N. gonorrhoeae*, *T. vaginalis*, *M. genitalium*, and HSV-1/2. These tests identify pathogen-specific DNA or RNA using amplification technologies such as polymerase chain reaction, transcription-mediated amplification, and strand displacement amplification.[140] NAAT offer very high sensitivity ($\geq$95%) and specificity ($\sim$100%) and can be performed on non-invasive specimens, such as urine and self-collected vaginal swabs.[140,141,144] Multiplex assays capable of detecting multiple pathogens simultaneously are increasingly available.[154] Commercial NAAT platforms have evolved substantially over the past two decades but remain costly, require trained personnel, and need continuous power, clean running water, and climate control to operate.[140,141] Cartridge-based platforms, such as GeneXpert, offer simplified workflows, near POC functionality, and results within 90 minutes, but are similarly constrained by infrastructure and cost.[140,141] More recently, regulatory approval has been granted for several NAAT that are capable of delivering results in under 30 minutes with minimal equipment or training.[140,155] NAAT is not routinely used for *T. pallidum*, given low organism load outside of primary lesions, but can aid early syphilis diagnosis.[144] For HSV, NAAT is the most accurate test for symptomatic infection, though not recommended for asymptomatic screening due to intermittent viral shedding.[140,150] It is essential for detecting *M. genitalium*, which cannot be detected by other routine methods.[140]

1.3.4 Screening

Syndromic management and diagnostic testing primarily assess presenting symptomatic clients. However, the high proportion of asymptomatic cases, particularly for cervical infections in women and extragenital infections in key populations, has driven efforts to introduce systematic screening that can interrupt transmission and prevent sequelae among higher-risk populations. Many diagnostic tools, especially NAAT, can accurately detect asymptomatic infections and support such interventions.[140]

In high-income countries, routine or risk-based screening for chlamydia, gonorrhoea, and syphilis is recommended for young women, pregnant women, people living with HIV, and key populations.[156–160] However, in SSA, only antenatal syphilis screening is routinely recommended, aligned with WHO guidance for universal syphilis testing at the first antenatal care (ANC) visit.[151,161] Screening coverage varies widely across countries, with major gaps in early ANC attendance and health facility capacity for testing and treatment.[139,162] WHO also recommends at least annual syphilis screening among key populations,[163] though coverage remains highly limited in SSA.[164]

1.3.5 Point-of-care platforms

Point-of-care tests (POCTs) offer a practical alternative to conventional laboratory diagnostics by enabling same-day testing and treatment in decentralised settings.[116,165,166] These tests can help overcome the limitations of syndromic management and enable detection of asymptomatic infections through screening. POCTs also support timely counselling, reduce loss to follow-up, and have demonstrated cost-effectiveness compared to both syndromic algorithms and laboratory-based diagnostics.[167–170] Despite this potential, no currently available tests meet usability and accessibility standards defined by the WHO REASSURED criteria (**R**eal-time connectivity, **E**ase of specimen collection, **A**ffordable, **S**ensitive, **S**pecific, **U**ser-friendly, **R**apid and robust, **E**quipment-free, and **D**eliverable to end-users).[171] While progress has been made in developing tests for syphilis, chlamydia, gonorrhoea, and trichomoniasis, existing diagnostics fall short in at least one domain (Table 1.2).[140,141]

Gonorrhoea poses the most urgent diagnostic gap, with missed cases and unnecessary antibiotic use from syndromic management being of particular concern amidst rising global AMR (Section 1.4.1).[172–174] To guide diagnostic development, WHO has published target product profiles (TPPs) for gonorrhoea POCTs that should be able to detect infection and identify genetic markers of resistance.[175,176] These profiles outline minimum and optimal thresholds for sensitivity (80% and 95%), specificity (95% and 98%), time to result ($\leq$30 and $\leq$10 minutes), cost (<US$3 for low complexity rapid test, and <US$12 for moderate/high complexity molecular test), and operational characteristics suitable for integration into primary care.[175] Several promising POCTs for gonorrhoea are in development, including both antigen-based and molecular assays, which aim to meet these benchmarks.[140,141,177] A novel lateral flow assay, recently evaluated in South Africa, demonstrated high sensitivity (>90%) and specificity (>95%) in symptomatic men and women.[178] However, most candidate POCTs remain in late-stage development or early implementation trials, with persistent challenges around affordability, supply chain integration, and primary care deployment.[174,175,179]

1.4 Barriers to effective control

Despite the availability of effective prevention, diagnostic, and treatment interventions, STI epidemics remain largely uncontrolled in SSA. This persistent burden reflects a combination of biological and programmatic challenges that limit the effectiveness of current strategies.

1.4.1 Antimicrobial resistance

AMR poses a growing threat to the treatment and control of STIs, particularly gonorrhoea, which has repeatedly acquired resistance to successive classes of antibiotics. The genetic adaptability of *N. gonorrhoeae*, combined with widespread antimicrobial misuse, has progressively narrowed effective treatment options globally.[180] In SSA, gonorrhoea has near-universal resistance to older first-line therapies, including penicillin, tetracyclines, and fluoroquinolones such as ciprofloxacin.[122,181] Ciprofloxacin resistance exceeds 90% in several settings,[122,181] and although no longer recommended for treating gonorrhoea, it continues to be used empirically for urethritis and other infections, contributing to sustained selective pressure.[182] Current treatment for gonorrhoea relies on extended-spectrum cephalosporins, primarily ceftriaxone, often administered alongside azithromycin.[117] In most SSA settings, susceptibility to these agents remains high, but sporadic cases of resistance and early warning signs, such as rising minimum inhibitory concentrations, are beginning to emerge.[122,182,183] In contrast, global resistance is increasing more rapidly; decreased susceptibility or resistance to ceftriaxone has been reported in 21% of countries, and resistance to azithromycin in 84% of countries globally.[181] However, AMR surveillance remains limited in SSA, and resistance may therefore be under-detected (Section 1.5).

1.4.2 Programmatic and structural challenges

Programmatic and structural challenges span the full continuum of STI care and include interrelated constraints on care-seeking, service delivery, and health system infrastructure.

Demand-side barriers include low and delayed treatment-seeking for symptomatic infections. Population-based surveys indicate that 50-65% of symptomatic individuals seek treatment in SSA.[184–187] Men are more likely to report seeking care than women, and to do so more promptly,[188–191] while adolescents seek care less than adults.[190,192,193] Urban residence, employment, higher education, and socioeconomic status are also associated with increased care-seeking,[184–186,189,190,194–196] particularly when symptoms are severe or persistent.[187,188,197,198] Nearly half of symptomatic individuals delay care for over a week,[199] often due to poor symptom recognition, stigma, or financial barriers.[192,197,200–202]

Service delivery barriers include fragmented care pathways, inconsistent quality, and poor implementation of syndromic management protocols. In SSA, fragmentation stems from complex health systems and weak coordination between sectors, with individuals seeking care from diverse providers. Public health facilities are most commonly used, especially by women, while others turn to private clinics, pharmacies, or informal providers such as traditional healers.[187,203] Non-governmental providers are often perceived to be more confidential and convenient, and less stigmatising.[200,203,204] Utilisation patterns vary by gender, age, and socioeconomic status,[187,192,196,200,203,205] but remain characterised, complicating programme delivery, surveillance, and resource allocation. Among those accessing care, syndromic management is often inadequately implemented. Only 50-60% of symptomatic individuals were appropriately examined in studies from West Africa,[206] Kenya,[207] and South Africa,[208] while the correct treatment was identified or administered by 40-60% of providers in Kenya,[207] Ethiopia,[209] South Africa,[208,210,211] and Namibia.[212] Case management is particularly weak in pharmacies, where only 10-20% of symptomatic clients received appropriate treatment in studies from Ghana,[213] Kenya,[214] and South Africa.[215] Incomplete symptom assessment, limited physical examination, and poor adherence to guidelines all contribute to suboptimal care and undermine the effectiveness of syndromic management.

Structural barriers include limitations in health system infrastructure, governance, and financing. Health systems often face human resource shortages, particularly in rural areas, and geographic barriers that hinder access to services. Supply chain disruptions are common, with stock-outs of essential commodities (benzathine penicillin, azithromycin, ceftriaxone, acyclovir, and rapid syphilis tests) reported by over one-third of African countries in recent WHO assessments.[139,216] These shortages are driven by disjointed procurement systems, weak forecasting, and the exclusion of STI commodities from many national essential medicine lists.[1,217,218] Surveillance systems are limited by poor integration, lack of disaggregated data, and delays in reporting, which undermine timely programmatic responses (Section 1.5). Governance challenges include national strategies that often lag behind global guidance and lack coordination and accountability mechanisms.[1,2,219] Many countries also depend heavily on external donors, with limited domestic funding. Abrupt cuts to foreign aid in 2025 are disrupting HIV prevention and treatment programmes,[220,221] with wider consequences for health system capacity, key population services, and surveillance that threaten the effectiveness of STI programmes globally.

1.5 Surveillance

STI prevention and control requires effective surveillance to monitor epidemic trends, evaluate and optimise interventions, and measure progress against national and global targets. Surveillance data underpin strategic planning, resource allocation, and programme advocacy.[1,222] In SSA, STI surveillance remains fragmented despite the availability of recommended tools and frameworks. This section describes its core components and the current status of prevalence estimates.

1.5.1 Core components

The WHO recommends four components of national STI surveillance: case reporting, prevalence assessments, aetiologic assessment of STI syndromes, and AMR monitoring. The first two quantify the magnitude of the STI burden to support programme management, while the latter two inform treatment recommendations to improve patient care.[223,224] To operationalise and monitor progress across these domains, WHO recently published a monitoring framework with 11 core indicators structured along a results chain that covers inputs and processes (e.g. policies, financing, surveillance systems, commodity security), outputs and outcomes (e.g. condom distribution, service coverage, case numbers), and impact (e.g. test positivity, congenital syphilis case rate).[222] These indicators are reported through global platforms such as the Global AIDS Monitoring system and the Gonococcal Antimicrobial Surveillance Programme. Each of the four surveillance components is described below, with a focus on its implementation and associated challenges in SSA.

Clinical case reporting is the primary mechanism to monitor the incidence and geographic distribution of symptomatic infections.[222] In SSA, case reporting is predominantly syndromic, which substantially under-represents infection incidence due to the high proportion of asymptomatic infections and the limited sensitivity of syndromic algorithms.[224] These data also exclude individuals who do not seek care, and public health reporting generally omits those treated in private or informal sectors.[224] Further challenges include the absence of standardised case definitions, incomplete and aggregate reporting, and coverage of only selected facilities or geographies.[223–225] For example, only 36% of countries in the WHO African region (17 of 47 included) reported urethral discharge case numbers in 2016–17, the most recent period reported at the regional level.[224] As a result, case reporting has had limited utility for surveillance or programmatic decision-making in SSA.

Infection prevalence surveys support estimation of the burden of infection among general and

key populations, and are recommended every 3-5 years.[222,223] In SSA, surveys are infrequent, seldom population-representative, and poorly integrated into national planning or guideline development processes.[224,225] Implementation is hindered by high costs, logistical complexity, and reliance on donor funding, with many surveys using diagnostic tests with poor performance.[223] Survey findings are generally synthesised using systematic reviews, meta-regressions, or modelling (Section 1.5.2).

Aetiologic assessments of STI syndromes support evaluating whether syndromic management protocols are clinically appropriate, and are recommended every 2-3 years.[222,224] They help identify pathogens not adequately treated by existing algorithms and detect shifts in predominant aetiologies or patterns of co-infection.[223] Given the continued reliance on syndromic management in SSA, despite its limited diagnostic accuracy, these routine aetiologic assessments are critical. However, only 40% of countries in the WHO African Region (11 of 26 surveyed) have reported including aetiologic assessments in their national STI surveillance plans.[139] The extent to which syndrome aetiologies vary across settings and sub-populations remains insufficiently documented, and no systematic synthesis has described the geographic scope or representativeness of existing studies.

AMR monitoring, currently focused on *N. gonorrhoeae*, is supported through the Enhanced Gonococcal Antimicrobial Surveillance Programme, which facilitates early detection of resistant strains to inform treatment guidelines at both national and global levels.[222] As of 2023, the programme had been fully implemented in only four countries in the WHO African Region (South Africa, Uganda, Malawi, and Zimbabwe).[180] Surveillance across the region remains constrained due to limited laboratory capacity and infrastructure.

While not considered core to surveillance, **nationally representative surveys**, such as Demographic and Health Surveys,[226] measure self-reported STI-related genital symptoms and associated treatment-seeking behaviours. The potential of these data to complement traditional surveillance has not been fully explored. This is largely due to concerns about data validity, since self-reported symptoms lack specificity and may reflect several infectious or non-infectious causes. Reporting may also be affected by poor symptom recognition, stigma, and social desirability bias, leading to misclassification. Despite these limitations, household surveys are more consistently available and population-representative than aetiologic prevalence surveys, and include individuals typically missed by facility-based case reporting. These surveys may therefore help characterise patterns in symptomatc STI burden and care-seeking, and could support programme planning where surveillance data are limited.

1.5.2 Prevalence estimates

Prevalence estimates quantify the burden of asymptomatic and symptomatic infections to inform programme priorities and monitor progress towards global targets. As mentioned, high-quality and population-representative prevalence studies remain sparse in SSA.[222] Statistical modelling can consolidate heterogeneous study findings and extrapolate them to broader populations, enabling comparisons across settings and over time.

Three global initiatives generate regular STI prevalence estimates. WHO publishes global and regional prevalence estimates approximately every four years for chlamydia, gonorrhoea, trichomoniasis, and syphilis,[4,227–230] and less frequently for HSV-2.[231–233] The GBD study generates global, regional, and national prevalence trends for several STIs.[234] Spectrum-STI produces national estimates for chlamydia, gonorrhoea, and syphilis,[235] although only syphilis estimates are routinely generated, owing to the availability of syphilis data from ANC screening and survey-based assessments.[236–239] All three estimation approaches aim to account for limitations in available data. Strategies include applying fixed assumptions, such as prevalence ratios between sexes or urban and rural settings, or using covariate-based predictions informed by socio-demographic or behavioural factors.[227,230,234,234] While these strategies can fill gaps when data are scarce, they may also obscure local epidemiologic patterns and introduce bias when assumptions are not well validated at regional or subregional levels.

Additional regional systematic reviews and meta-analyses have largely focused on specific subpopulations, such as ANC attendees[240,241] or women living with HIV,[242] and have rarely examined spatial and temporal trends in SSA. As a result, current prevalence estimates in the region, whether derived from global modelling initiatives or population-specific reviews, fall short of providing sex-stratified, population-representative trends across subregions and over time. This gap is particularly acute for the curable STIs chlamydia, gonorrhoea, and trichomoniasis, which are included in WHO incidence reduction targets but remain poorly characterised in SSA.

1.6 Modelling diagnostic and screening interventions

Improved case detection, whether through diagnostic testing of symptomatic individuals or systematic screening of asymptomatic populations, has potential to substantially strengthen STI control by increasing effective treatment and thereby reducing transmission. Improved case detection could be particularly impactful for chlamydia and gonorrhoea, which are highly prevalent, frequently asymp-

tomatic, and associated with adverse reproductive health outcomes. Mathematical modelling provides a framework to evaluate the potential population-level impact, efficiency, and cost-effectiveness of such interventions, particularly where empirical evidence is limited or infeasible to generate.

Modelling approaches

Modelling approaches used to assess diagnostic and screening interventions for STIs span a continuum of complexity. Transmission dynamic models simulate STI transmission over time and evaluate how interventions influence epidemic trajectories. Most dynamic models use compartmental frameworks that stratify the population into homogeneous groups by characteristics such as age, sex, and sexual activity class.[243,244] These models typically assume that transmission rates are proportional to the prevalence of infection among potential partners. They are flexible, adaptable to limited data, and computationally efficient. However, they tend to oversimplify sexual behaviour.[245] By aggregating individuals into compartments and not explicitly accounting for partnership concurrency, turnover, or duration, these models may understate the role of higher-risk groups in transmission, while overstating the contribution of lower-risk individuals in long-term partnerships.[7] Pair-formation models extend compartmental approaches by explicitly representing sexual partnerships as repeated contacts with the same individual over time, thereby capturing both the structure and duration of relationships. This allows for more realistic modelling of reinfection dynamics and the timing of intervention effects.[246,247] Individual-based models offer the most detailed framework to represent dynamic sexual networks, allowing for explicit modelling of partnership formation, dissolution, concurrency, and duration, as influenced by individual-level attributes.[245] By capturing the structure and timing of partnerships more explicitly, they are particularly valuable for exploring interventions that rely on individual contact histories, such as partner notification or contact tracing.[245] However, these models typically require more extensive parametrisation and computational resources than compartmental approaches. Static models, including cohort and other decision-analytic approaches, estimate outcomes based on a fixed set of assumptions without incorporating changes in transmission over time. These models are computationally simpler but tend to underestimate population-level impact by not accounting for secondary infections averted through intervention (indirect effects) and often yield more conservative estimates of effectiveness and cost-effectiveness.[248]

Evidence from high-income countries

Most mathematical modelling of diagnostic and screening interventions for STIs has been conducted in high-income countries, often to inform national chlamydia screening policies. These studies consistently identify routine screening of young women (typically aged 15–24 years) as the most impactful and cost-effective strategy.[14,246,248–252] This reflects the high infection prevalence among young women, partly due to their biological susceptibility (e.g. cervical ectopy); the large proportion of asymptomatic infections; and behavioural dynamics through which screening women confers indirect protection to male partners. Screening men has generally shown limited additional benefit at the population level, unless targeted toward groups with high prevalence or limited indirect protection, such as MSM.[14,249,252–255] Modelled scenarios suggest that even moderate annual screening coverage of 20–40% among young adults can reduce chlamydia prevalence by more than 50% over 5-10 years,[14,249,251,256] with further gains achieved through timely treatment and partner notification,[248,249,253,257] although estimates vary across models. However, divergences between modelled predictions and observed trends, such as rising case notifications during the UK screening programme, highlight key uncertainties in natural history, sexual behaviour, and the challenges of real-world implementation.[252,258–261]

Gonorrhoea screening has been modelled less extensively in high-income countries. Broad age-based screening strategies designed for chlamydia are less applicable to gonorrhoea, due to its concentration in higher-risk sexual networks, shorter duration of infection, and more frequent symptomatic presentation among men.[14,246,262] As a result, models have generally supported more targeted strategies, particularly among MSM,[254,262,263] FSW and their partners,[246,264] and other higher-risk groups.[14,246,262,265] Among MSM, modelling studies have shown that screening extragenital sites (rectal and pharyngeal) enhances the population-level impact of screening programmes compared to urogenital-only strategies.[255,263,266–268] At the same time, intensified screening among low-risk MSM has been shown to have minimal impact on population-level prevalence but may increase the risk of AMR by increasing overall antibiotic consumption.[269]

More recent models have evaluated the use of POCTs for chlamydia and gonorrhoea to reduce treatment delays and loss to follow-up. These studies suggest that POCTs can enhance the impact of screening programmes, even when sensitivity is lower than that of conventional molecular tests, provided they are implemented with sufficiently high coverage among targeted populations.[14,168,169,270]

Evidence from and implications for SSA

In contrast to extensive modelling literature from high-income countries, few studies have evaluated diagnostic and screening strategies in SSA. One individual-based model (MicroCOSM) assessed opportunistic screening for chlamydia and gonorrhoea among different populations in South Africa. It found that population-level prevalence reductions were greatest when targeting youth or individuals in HIV care, more modest for pregnant women, and smallest for FSW, largely due to differences in the number of individuals screened.[271] However, screening FSW was the most efficient strategy per test used, particularly for gonorrhoea, due to their high infection burden and transmission potential.[271]

Other modelling studies in SSA have assessed the impact of POCTs for screening and diagnosis. In South Africa, POCT-based screening among ANC attendees was estimated to substantially reduce adverse pregnancy outcomes.[272] In Benin, using POCTs to replace syndromic management among FSW reduced overtreatment, decreased population-level incidence, and was cost-effective.[273] In Benin, South Africa, and Tanzania, modelling of sensitivity and coverage scenarios among FSW and women in long-term partnerships found that even moderate-sensitivity POCTs for diagnosing symptomatic infections would avert more cases than laboratory-based diagnostics.[274]

Collectively, findings from SSA reinforce several mechanisms observed in high-income settings, such as the efficiency of targeted screening in high prevalence groups and the value of timely linkage to treatment. However, there are also contextual differences: high prevalence, limited diagnostic infrastructure, and low levels of treatment-seeking can potentially increase the relative advantages of POCTs in SSA. Broader or less targeted strategies may therefore be justified in this context, despite lower positivity yield per test, due to potentially greater population-level impact. Despite these insights, key questions remain unanswered. These include how to allocate limited resources between diagnostic and screening strategies, which population groups to prioritise, and how these decisions might vary depending on whether the primary goal is to reduce transmission or prevent long-term sequelae.

1.7 Thesis aims

The overarching aim of this thesis is to generate evidence that strengthens the surveillance and control of STIs in SSA, where high infection burdens, widespread asymptomatic transmission, and limited diagnostic capacity hinder progress towards global incidence reduction targets. To achieve this, I characterise the epidemiology of STIs among both the general population and individuals with

genital symptoms, assess the utility of self-reported data from population-representative surveys to monitor symptomatic infection burden and treatment coverage, and evaluate strategies to optimise the deployment of future POCTs in contexts reliant on syndromic management. Countries for in-depth analysis were selected based on the availability of relevant data, existing research collaborations, and the potential to inform national and regional STI policy. The following chapters address these aims:

Chapter 2 estimates the prevalence of chlamydia, gonorrhoea, and trichomoniasis among general populations in SSA. I conduct a systematic review and meta-regression analyses to generate sub-regional and sex-stratified prevalence estimates over time and derive male-to-female prevalence ratios for each infection. My analysis addresses limitations in existing prevalence estimates for the region and supports future global burden estimation efforts.

Chapter 3 characterises the aetiologies of vaginal discharge, urethral discharge, and genital ulcer disease in SSA. I conduct a systematic review and meta-regression analyses to estimate the distribution of reproductive tract infections causing each symptom and examine variations over time, by sub-region, and for specific subpopulations. My findings provide an empirical basis for evaluating and refining syndromic management guidelines in the region.

Chapter 4 investigates whether self-reported genital symptoms from nationally representative surveys can support quantification of the prevalence and trends of STIs in SSA. I use regression analyses to estimate national prevalence of reported vaginal discharge, urethral discharge, and genital ulcer over time, and evaluate the correlation between these symptom estimates and the aetiologic prevalence observations from Chapter 2. I also analyse the prevalence of reported treatment for these symptoms and patterns of healthcare utilisation across different sectors over time.

Chapter 5 further evaluates surveillance of symptomatic STIs by comparing self-reported genital symptoms and treatment-seeking from nationally representative surveys with syndromic case diagnoses from health facility surveillance in Malawi. I use descriptive and regression analyses to examine temporal, spatial, and socio-demographic patterns in vaginal discharge, urethral discharge, and genital ulcer across the two data sources. My analysis assesses whether self-reported data can complement routine case reporting in settings with limited surveillance.

Chapter 6 evaluates how future gonorrhoea POCTs could be most effectively deployed in Kenya. I use a compartmental transmission model to compare the population-level impact of diagnostic testing and screening strategies among five target populations. My analysis informs how limited POCT supplies

could be allocated to maximise reductions in incidence and morbidity in resource-constrained settings.

Chapter 7 concludes the thesis by synthesising my key findings, outlining their implications for STI surveillance and control, and identifying priorities for future research in SSA.

Chapter 2

Prevalence of curable sexually transmitted infections among male and female general populations in sub-Saharan Africa

2.1 Introduction

Reaching WHO 2030 STI incidence reduction targets requires ambitious scale-up of national prevention and control programmes in SSA.[1] However, monitoring and optimising these programmes is challenged by high rates of asymptomatic infection, variable health-seeking behaviour, limited diagnostic capacity, and weak surveillance.[222] STI prevalence estimates are crucial for evaluating progress toward targets but are hindered by infrequent population-representative studies, especially among men.[224]

Estimates of curable STI prevalence are limited in SSA. WHO publishes global and regional prevalence and incidence estimates for curable STIs, including chlamydia, gonorrhoea, and trichomoniasis, approximately every four years.[4,227–230] These focus on global geographic regions and do not report sub-regional variation or temporal trends. The GBD study generates global, regional, and national prevalence and incidence trends for several STIs.[234] However, the limited quantity and representativeness of available prevalence data necessitate model assumptions that may obscure local epidemiologic variation. Due to limited data among men, WHO and GBD have derived male-to-female prevalence ratios from global

data and applied these in most regions to support male prevalence estimates.[227,230,234] Although WHO reviews its global ratios prior to each estimation round, values have not been updated since 1999 for trichomoniasis and 2005 for chlamydia and gonorrhoea.[227,230] Additionally, systematic reviews have assessed the prevalence of curable STIs in SSA among pregnant women,[240,241] women participating in HIV prevention trials,[275] or women living with HIV,[242] but their generalisability beyond the specified populations is limited and these reviews have not examined changes in STI prevalence over time.

In this chapter, I estimate prevalence patterns for chlamydia, gonorrhoea, and trichomoniasis among adults considered representative of the general population (i.e. not conducted among groups identified as key populations, as defined in WHO guidelines on HIV, STIs, and viral hepatitis[1]) in SSA from 2000 to 2024. I perform a systematic review and meta-analyses to assess variations in STI prevalence by sex, sub-region, and over time. To inform future regional and global STI burden estimation efforts, I derive updated male-to-female prevalence ratios specific to SSA.

2.2 Methods

2.2.1 Search strategy

I systematically searched Embase (Ovid), MEDLINE (Ovid), Global Health (Ovid), PubMed, and African Index Medicus from 1 January 2000 to 17 September 2024 for studies assessing the prevalence of chlamydia, gonorrhoea, and trichomoniasis in SSA. Search term domains included relevant terms and synonyms for "sexually transmitted infections" and "sub-Saharan Africa" (Table A1). I also searched citations from relevant published systematic reviews.[4,227,229,240–242,275–281]

2.2.2 Eligibility criteria and study selection

I included studies reporting sex-stratified empirical data on the prevalence of chlamydia, gonorrhoea, or trichomoniasis among adults from the general population in SSA between 2000 and 2024. I considered studies to represent the general population if they enrolled participants from ANC, family planning, or gynaecology clinics; primary healthcare or other outpatient services; educational institutions; or community venues (e.g., social spaces or places of worship); or recruited participants for HIV or STI prevention trials (baseline data only) or population-representative surveys. Studies were eligible regardless of participants' symptom status, though I excluded those that exclusively enrolled symptomatic individuals or individuals seeking treatment for STIs or infertility. I excluded studies that exclusively enrolled participants from populations living with HIV or key populations.

I included studies with participants aged 12 years or older who were tested using internationally recognised aetiologic diagnostic tests with urogenital specimens (urine, urethral, or cervicovaginal samples), and excluded studies that only collected pharyngeal or rectal samples. Case reports, commentaries, longitudinal and randomised controlled studies reporting only post-baseline outcomes, and studies with fewer than 15 participants were also excluded. I used the UN M49 standard to define SSA and its sub-regions (Table A2).[282]

Search results were managed and de-duplicated using Covidence systematic review software (Veritas Health Innovation, Melbourne, Australia).[283] I worked with co-authors (DMR, EM, JWI-E, JK, LH, MKW, OE, OS, QH, RLA, SK) to independently double-screen title and abstract records for eligibility and assess full text articles for inclusion. We resolved any discrepancies with a third co-author.

2.2.3 Data extraction

I worked with co-authors (LH, RLA, QH) to independently double-extract prevalence observations (number of individuals with a positive result of the total number tested) for each study with stratification by country, sex, and population group, as available. Information on study characteristics, participant characteristics, and diagnostic methodology was also recorded (Table A3). We resolved discrepancies through consensus. If outcomes from the same study were reported in multiple articles, we preferentially extracted observations from the largest sample or, if samples were the same size, the first published article. For studies collecting samples from multiple anatomical sites, we extracted only urogenital results. If multiple diagnostic tests were conducted, we extracted outcomes from the test and sample type with highest sensitivity and specificity for pathogen identification (Table A4). We preferentially extracted non-stratified rather than HIV-stratified outcomes if both were reported.

2.2.4 Data analysis

To adjust study prevalence observations for imperfect diagnostic test performance, I collated sensitivity and specificity values for each test category from published literature (Table A4)[140,230] and used a Bayesian approach to estimate prevalence in each study population (Text A.1.2).[284,285]

For studies that did not report data collection dates (n=26), I imputed the midpoint year of data collection by subtracting the median publication lag from the publication year (three years, based on the difference between the data collection midpoint year and year of publication among studies with dates reported). For one study spanning multiple sub-regions without country- or region-specific data

reported,[286] I classified the study sub-region according to where most participants had been recruited.

To estimate sub-regional trends in sex-specific STI prevalence in SSA, I fit log-binomial mixed-effects meta-regressions for each pathogen. Fixed effects included year (midpoint of data collection), study population (ANC attendees, family planning clinic attendees, gynaecology clinic attendees, primary healthcare or other outpatient facility attendees, students, community members recruited from non-clinical venues, HIV/STI prevention trial participants, and population-representative survey participants), sex (female, male), age group (younger adults 12-25 years, all adults $\geq$ 12 years), HIV status (HIV negative, non-stratified HIV status), diagnostic test (nucleic acid amplification test [NAAT], culture, direct fluorescent antibody, enzyme immunosorbent assay, rapid antigen test, wet mount), sub-region (Western and Central Africa [WCA], Eastern Africa [EA], or Southern Africa [SA]), year and sub-region interaction term, and sex and sub-region interaction term. A year and sex interaction term was not included due to limited observations of male prevalence. I combined Western and Central Africa due to limited prevalence observations in Central Africa. Study-level random intercepts allowed for between-study heterogeneity. Fitted models were used to predict the prevalence of each infection among male and female adults by sub-region between 2000 and 2024, using reference categories for covariates in the model (study population: ANC attendees, HIV status: non-stratified, diagnostic test: NAAT). Estimates are reported for sub-regions and all of SSA for 2020. SSA estimates were obtained by weighting sub-regional predictions with sex-matched population size for adults aged 15-49 years in 2020, according to UN World Population Prospects 2024.[287] Standard errors were determined using the delta method.[288]

To assess sex differences in prevalence, I estimated male-to-female prevalence ratios using observations from all studies (between-study ratios) and from the subset of studies providing estimates for both sexes (within-study ratios). Between-study ratios were estimated using the previously described meta-regression models. I determined within-study ratios using the same meta-regression approach, but without fixed effects for population group and an interaction term for sex and sub-region, due to limited observations. Pooled within-study ratios were derived without adjustment for any covariates.

2.2.5 Sensitivity analyses and risk of bias

I assessed the sensitivity of prevalence estimates, trends, and male-to-female prevalence ratios to adjustments for diagnostic performance by comparing results from models using all available observations versus NAAT-diagnosed observations, with and without accounting for test sensitivity and specificity.

I adapted the Joanna Briggs Institute critical appraisal tool for prevalence studies to assess risk of bias. I classified studies as lower or higher risk of selection bias (sampling method, participant characterisation), measurement bias (diagnostic method consistency), and poor precision (sample size adequacy; Table A5).[289] I then extended my primary meta-regressions to include fixed effects for each criterion, to explore whether prevalence estimates differed between studies assessed as lower versus higher risk of bias.

Unless indicated otherwise, all main results reflect observations adjusted for diagnostic test performance. Results are presented as means with 95% confidence intervals (95% CIs). Model coefficients are presented as adjusted prevalence ratios (aPRs), with confidence intervals calculated on the log scale prior to exponentiation. Study observation heterogeneity was assessed per model as the percentage of total variance attributed to observation-level random effects.[290] Analyses were conducted in R version 4.2.3; Bayesian diagnostic test performance adjustments used `rstan` version 2.32.6[291] and meta-regression modelling used `glmmTMB` version 1.1.9.[292]

The study was pre-registered with PROSPERO (CRD 42023420384)[293] and reported according to the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) guideline (Table A6).[294] The Imperial College Research Ethics Committee approved analysis of secondary data for this study (ICREC 6365329).

2.3 Results

2.3.1 Search results and scope

A total of 11 136 records were identified through the database search, of which 5 934 were duplicates and 5 202 were screened (Figure 2.1). Of these, 783 full-text articles were assessed and 301 met the eligibility criteria. An additional 11 articles were identified through citation searching. After excluding 98 articles with duplicate study observations, 214 articles from 211 unique studies were included, from which 614 prevalence observations were extracted (NG=203, CT=203, TV=208; Table A7). Most studies were conducted in Eastern Africa (N=90/211, 42.7%), followed by Western and Central Africa (N=71/211, 33.6%) and Southern Africa (N=59/211, 28.0%; Table 2.1). Studies were conducted in 28 different SSA countries, with most in South Africa (N=53/211, 25.1%), Nigeria (N=38/211, 18.0%), Kenya (N=26/211, 12.3%), United Republic of Tanzania (N=20/211, 9.5%), and Uganda (N=19/211, 9.0%; Figure A1). Study populations were predominantly ANC attendees (N=85/211, 40.3%), primary

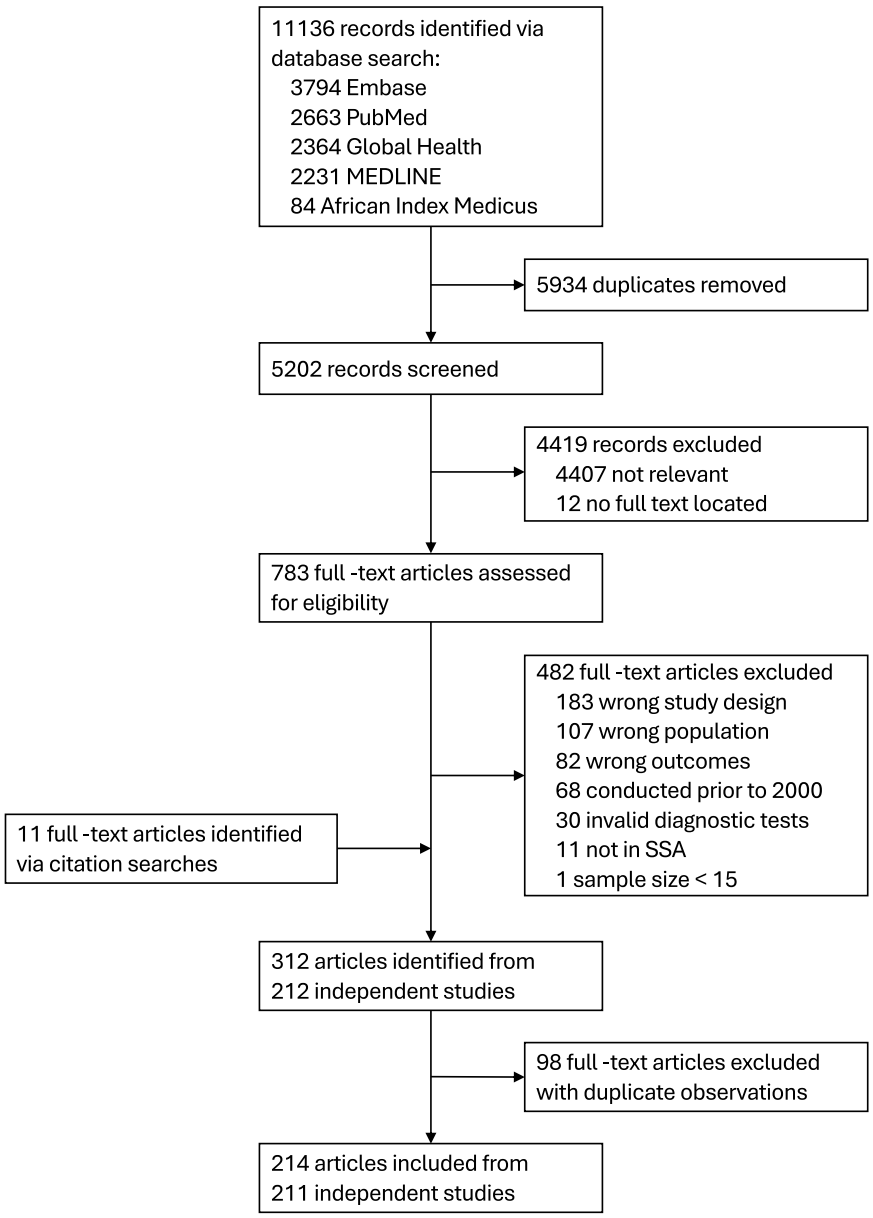


Figure 2.1: Study selection flowchart

Flowchart of study selection for systematic review

healthcare or outpatient department attendees (N=30/211, 14.2%), and HIV/STI prevention trial participants (N=24/211, 11.4%). Few studies were among population-representative survey samples (N=11/211, 5.2%). Most studies were solely among females (N=174/211, 82.5%). A limited number included both sexes (N=29/211, 13.7%) or males only (N=8/211, 3.8%). Studies were predominantly among all adults (N=180/211, 85.3%), rather than restricted to younger adults (N=31/211, 14.6%). Across all studies, most enrolled within the 15-49-year age range (N=109/211, 51.7%), while others included younger and/or older participants (N=45/211, 21.3%), reported only a minimum adult age (N=37/211, 17.5%), or did not report age ranges (N=20/211, 9.5%).

Table 2.1: Summary of characteristics for included studies

Variable	Chlamydia (N=139)	Gonorrhoea (N=140)	Trichomoniasis (N=162)	Total (N=211)
Sub-region[*]				
Western Africa	20 (14.4%)	20 (14.3%)	51 (31.5%)	59 (28.0%)
Central Africa[†]	10 (7.2%)	8 (5.7%)	8 (4.9%)	12 (5.7%)
Eastern Africa	69 (49.6%)	70 (50.0%)	63 (38.9%)	90 (42.7%)
Southern Africa	49 (35.3%)	51 (36.4%)	46 (28.4%)	59 (28.0%)
Multiple sub-regions[‡]	1 (0.7%)	1 (0.7%)	1 (0.6%)	1 (0.5%)
Study midpoint year[*§]				
2000-2004	27 (19.4%)	32 (22.9%)	34 (21.0%)	45 (21.3%)
2005-2009	18 (12.9%)	19 (13.6%)	21 (13.0%)	27 (12.8%)
2010-2014	35 (25.2%)	31 (22.1%)	38 (23.5%)	51 (24.2%)
2015-2019	42 (30.2%)	41 (29.3%)	54 (33.3%)	64 (30.3%)
2020-2024	18 (12.9%)	17 (12.1%)	15 (9.3%)	25 (11.8%)
Population group[*]				
ANC attendees	39 (28.1%)	42 (30.0%)	75 (46.3%)	85 (40.3%)
FP attendees	12 (8.6%)	12 (8.6%)	11 (6.8%)	17 (8.1%)
GYN attendees	6 (4.3%)	8 (5.7%)	11 (6.8%)	14 (6.6%)
PHC/OPD attendees	22 (15.8%)	22 (15.7%)	20 (12.3%)	30 (14.2%)
Students	15 (10.8%)	11 (7.9%)	15 (9.3%)	19 (9.0%)
Community members[¶]	15 (10.8%)	16 (11.4%)	6 (3.7%)	17 (8.1%)
HIV/STI prevention trial participants[‖]	22 (15.8%)	22 (15.7%)	22 (13.6%)	24 (11.4%)
Population-representative survey participants	10 (7.2%)	10 (7.1%)	8 (4.9%)	11 (5.2%)
Sex				
Female only	106 (76.3%)	109 (77.9%)	143 (88.3%)	174 (82.5%)
Male only	7 (5.0%)	5 (3.6%)	7 (4.3%)	8 (3.8%)
Both sexes (stratified)	26 (18.7%)	26 (18.6%)	12 (7.4%)	29 (13.7%)
Age group				
All adults (12+ years)	112 (80.6%)	116 (82.9%)	140 (86.4%)	180 (85.3%)
Younger adults (12-25 years)	27 (19.4%)	25 (17.9%)	22 (13.6%)	31 (14.7%)
HIV status[††]				
HIV-negative	37 (26.6%)	38 (27.1%)	38 (23.5%)	46 (21.8%)
Non-stratified	103 (74.1%)	103 (73.6%)	125 (77.2%)	166 (78.4%)

[*]The same study is included in more than one subcategory if it reports across different variable levels or when multiple levels are relevant. [†]Western and Central Africa combined for analysis. [‡]Study without country- or region-specific reporting was allocated to Eastern Africa for analysis, according to where most participants were recruited. [§]Midpoint year between start and end of data collection period. [¶]Community members recruited through non-clinical venues, such as social spaces or places of worship. [‖]Studies reporting baseline data among trial participants. [††]Studies exclusively among populations living with HIV were excluded. Data were preferentially extracted and analysed without stratifying by HIV status if both were reported. N: number of studies, ANC: antenatal care, FP: family planning, GYN: gynaecology clinic, PHC/OPD: Primary healthcare or outpatient department.

2.3.2 Prevalence estimates

In 2020, estimated prevalence of chlamydia in SSA was 6.9% (95%CI: 5.2-8.7%, number of observations (n)=169) among females and 4.2% (3.0-5.5%, n=33) among males, gonorrhoea was 1.8% (1.1-2.5%,

n=171) and 1.5% (0.8-2.2%, n=31), and trichomoniasis was 7.6% (5.1-10.2%, n=188) and 1.7% (1.1-2.4%, n=19), respectively (Table A8, Figure 2.2). From 2010 to 2020, chlamydia prevalence increased by 34.5% (11.1-62.9%) in SSA, while trends for gonorrhoea (3.3% increase [-20.2-33.9%]) and trichomoniasis (17.8% increase [-3.8-34.9%]) were not statistically significant (Table 2.2, Figure 2.3).

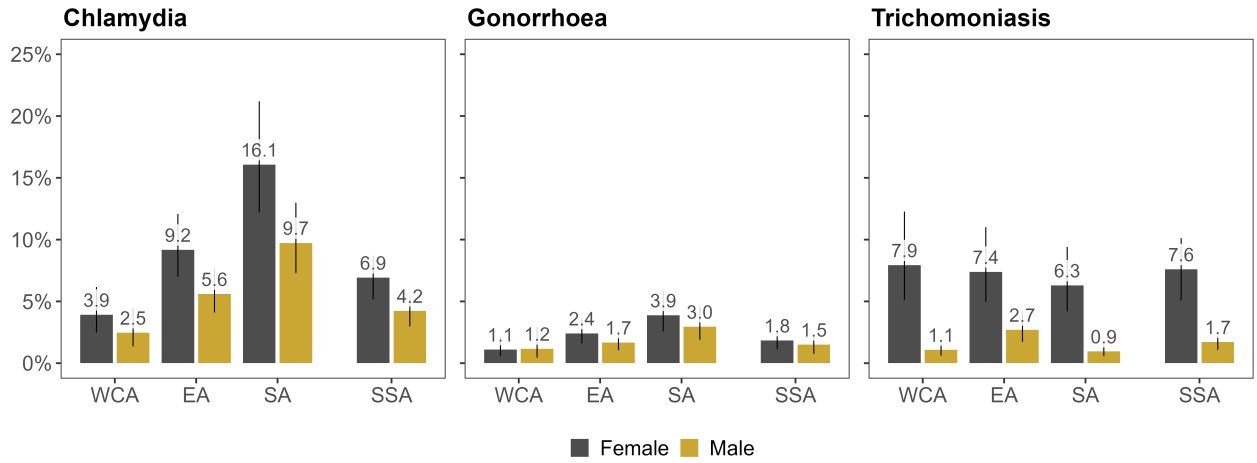


Figure 2.2: Estimated sexually transmitted infection prevalence in SSA, 2020

Estimates of chlamydia, gonorrhoea, and trichomoniasis prevalence by sex and sub-region in 2020. Bars and lines depict mean with 95% CIs. EA: Eastern Africa, SA: Southern Africa, WCA: Western and Central Africa.

Sub-regional variations in prevalence were similar for chlamydia and gonorrhoea (Table 2.2, Figure 2.2). Among females, relative to Southern Africa, prevalence of chlamydia and gonorrhoea were lowest in Western and Central Africa (aPR_{CT}: 0.31 [0.22-0.43], aPR_{NG}: 0.36 [0.23-0.57]), followed by Eastern Africa (aPR_{CT}: 0.38 [0.35-0.41], aPR_{NG}: 0.64 [0.56-0.74]). Similarly for males, prevalence was lower in Western and Central (aPR_{CT}: 0.38 [0.32-0.46], aPR_{NG}: 0.58 [0.44-0.78]) and Eastern Africa (aPR_{CT}: 0.32 [0.19-0.53], aPR_{NG}: 0.50 [0.23-1.10]) compared to Southern Africa. For trichomoniasis, female prevalence was similar across sub-regions (WCA aPR_{TV}: 0.94 [0.68-1.31], EA aPR_{TV}: 1.01 [0.85-1.21]), while male prevalence was higher in Eastern (aPR_{TV}: 2.45 [1.66-3.61]) than Southern Africa. Time trends for each infection varied across sub-regions. Chlamydia prevalence increased in Eastern (aPR_{CT} per year: 1.07 [1.05-1.09]) and Southern Africa (aPR_{CT}: 1.02 [1.00-1.04]), while trichomoniasis prevalence decreased in Southern Africa (aPR_{TV}: 0.96 [0.92-0.99]) only. Gonorrhoea prevalence was stable across all three sub-regions (Table 2.2, Figure 2.3).

Across study populations, there was no evidence that the prevalence of any infection differed compared to ANC attendees, except for lower trichomoniasis prevalence among family planning clinic attendees (aPR_{TV}: 0.54 [0.32-0.90]). Studies diagnosing chlamydia with a rapid antigen test (aPR_{CT}: 2.94 [1.76-4.92]), gonorrhoea with culture (aPR_{NG}: 1.60 [1.00-2.54]), and trichomoniasis with wet mount

(aPR$_{TV}$: 1.61 [1.14-2.29]) had higher prevalence than those that used NAAT, after accounting for diagnostic test performance (Table 2.2). Study observations were heterogenous for all three infections, with observation-level random effect variance comprising 36.4%, 60.8%, and 50.9% of total variance for chlamydia, gonorrhoea, and trichomoniasis, respectively (Table 2.2).

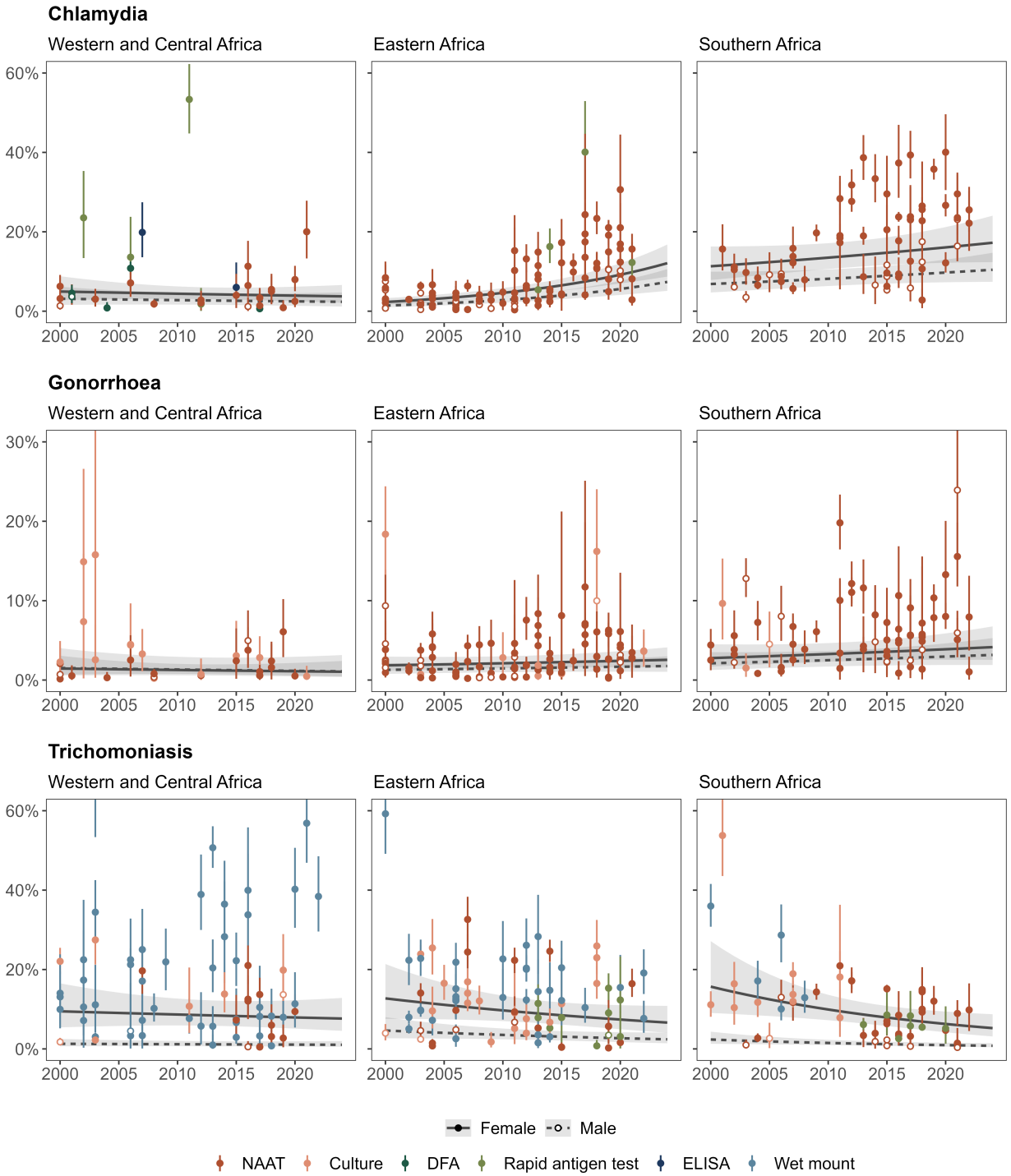


Figure 2.3: Estimated sexually transmitted infection prevalence in SSA, 2000-2024

Estimates of chlamydia, gonorrhoea, and trichomoniasis prevalence by sex and sub-region between 2000 and 2024. Lines and shading depict mean estimates with 95% CIs. Points and error lines represent study observations with 95% CIs. Y-axes truncated. DFA: direct fluorescent antibody, NAAT: nucleic acid amplification test.

Table 2.2: Adjusted prevalence ratios for chlamydia, gonorrhoea, and trichomoniasis in SSA, estimated via log-binomial generalised linear mixed-effects models

Variable	Chlamydia	Gonorrhoea	Trichomoniasis
	aPR (95% CI)	aPR (95% CI)	aPR (95% CI)
Intercept	0.14 (0.11-0.18)	0.03 (0.02-0.05)	0.09 (0.07-0.12)
Sub-region			
Western and Central	0.31 (0.22-0.43)	0.36 (0.23-0.57)	0.94 (0.68-1.31)
Eastern	0.38 (0.35-0.41)	0.64 (0.56-0.74)	1.01 (0.85-1.21)
Southern	Ref	Ref	Ref
Sub-region:Sex[*]			
Western and Central:Male	0.63 (0.43-0.93)	1.05 (0.54-2.04)	0.14 (0.09-0.20)
Eastern:Male	0.61 (0.53-0.70)	0.69 (0.56-0.85)	0.36 (0.30-0.44)
Southern:Male	0.61 (0.55-0.66)	0.76 (0.65-0.90)	0.15 (0.11-0.20)
Sub-region:Year[†]			
Western and Central:Year	0.95 (0.87-1.03)	0.99 (0.94-1.04)	0.99 (0.96-1.02)
Eastern:Year	1.07 (1.05-1.09)	1.01 (0.99-1.04)	0.99 (0.97-1.02)
Southern:Year	1.02 (1.00-1.04)	1.02 (0.99-1.05)	0.96 (0.92-0.99)
Population			
ANC attendees	Ref	Ref	Ref
FP attendees	0.95 (0.63-1.45)	0.90 (0.50-1.64)	0.54 (0.32-0.90)
GYN attendees	1.03 (0.57-1.88)	0.87 (0.42-1.82)	1.34 (0.80-2.25)
PHC/OPD attendees	0.81 (0.57-1.14)	1.48 (0.92-2.38)	0.60 (0.41-0.88)
Students	1.06 (0.69-1.63)	1.14 (0.56-2.31)	1.23 (0.76-1.99)
Community members	0.97 (0.66-1.44)	1.18 (0.65-1.87)	0.96 (0.50-1.85)
HIV/STI prevention trial participants	0.89 (0.61-1.28)	1.20 (0.72-1.99)	1.03 (0.65-1.63)
Population-representative survey participants	0.90 (0.58-1.42)	1.19 (0.64-2.20)	1.32 (0.71-2.44)
Age group			
All adults (12+ years)	Ref	Ref	Ref
Younger adults (12-25 years)	1.14 (0.85-1.52)	1.12 (0.75-1.68)	0.55 (0.35-0.88)
HIV status			
Non-stratified	Ref	Ref	Ref
HIV-negative	1.42 (1.06-1.90)	1.06 (0.71-1.59)	0.79 (0.56-1.14)
Diagnostic test			
NAAT	Ref	Ref	Ref
ELISA	2.26 (0.86-5.98)	-	1.36 (0.89-2.09)
DFA	0.70 (0.32-1.51)	-	-
ELISA	2.26 (0.86-5.98)	-	-
Rapid antigen test	2.94 (1.76-4.92)	0.52 (0.06-4.61)	1.03 (0.56-1.90)
Wet mount	-	-	1.61 (1.14-2.29)
Model variance			
τ^2 fixed	0.51 (54.5%)	0.16 (16.3%)	0.45 (38.0%)
τ^2 random	0.34 (36.4%)	0.60 (60.8%)	0.60 (50.9%)
τ^2 distribution	0.08 (8.7%)	0.23 (22.9%)	0.13 (11.0%)
τ^2 total	0.93 (100%)	0.98 (100%)	1.18 (100%)
Number groups			
Number studies	139	140	162
Number observations	202	202	207

[*]Reference sex is female. [†]Midpoint year between start and end of data collection period; centred at 2012. aPR: adjusted prevalence ratio, 95% CI: 95% confidence interval, ANC: antenatal care, DFA: direct fluorescent antibody, FP: family planning clinic, GYN: gynaecology clinic, NAAT: nucleic acid amplification test, PHC/OPD: primary health care or outpatient department, Ref: Reference group, τ^2: variance.

2.3.3 Male-to-female prevalence ratios

In SSA, male-to-female prevalence ratios for each infection were similar when estimated as adjusted between-study ratios (all available data), adjusted within-study ratios (paired data), or unadjusted pooled within-study ratios (Table 2.2, Table A9, Figure 2.4). Male-to-female prevalence ratios (adjusted between-study) for SSA in 2020 were 0.61 (0.53-0.71) for chlamydia, 0.81 (0.61-1.09) for gonorrhoea, and 0.23 (0.18-0.28) for trichomoniasis (Table 2.2, Figure 2.4). Between-study prevalence ratios were consistent across sub-regions for chlamydia (WCA:0.63 [0.43-0.93], EA:0.61 [0.53-0.70], SA:0.61 [0.55-0.66]), but varied for gonorrhoea (WCA:1.05 [0.54-2.04], EA:0.69 [0.56-0.85], SA:0.76 [0.65-0.90]) and trichomoniasis (WCA:0.14 [0.09-0.20], EA:0.36 [0.30-0.44], SA:0.15 [0.11-0.20]; Table 2.2).

2.3.4 Sensitivity analyses and risk of bias

Prevalence estimates in 2020 were consistent for all three infections when derived using all available or NAAT-only observations, with or without accounting for diagnostic test performance (Tables 2.2, A10 - A12, Figure A2). Temporal trends for chlamydia and gonorrhoea also remained consistent across sub-regions. However, for trichomoniasis, all-observation models showed a prevalence decline in Southern Africa (aPR_{TV}: 0.96 [0.92-0.99] with and without test performance), while NAAT-only models did not (with test performance: 0.96 [0.90-1.02]; without: 0.96 [0.92-1.01]; Tables 2.2, A10 - A12, Figure A3). Between-study and within-study male-to-female prevalence ratios were consistent for all three infections, whether derived using all available or NAAT-only observations. However, accounting for test performance led to higher ratios for gonorrhoea and lower ratios for chlamydia and trichomoniasis (Tables 2.2, A10 - A15, Figure A4).

The risk of selection bias was high, as most studies lacked probability-based sampling (N=174/211, 82.5%) or sufficient participant characterisation (N=133/211, 63.0%). The risk of measurement bias was low, with nearly all studies using consistent diagnostic methods in all participants (N=198/211, 93.8%). Study precision, defined as sample size greater than 100, was generally adequate (N=201/211, 95.3%, Table A16). Trichomoniasis prevalence was higher in studies with insufficient reporting on participant characteristics (aPR_{TV}: 1.52 [1.22-1.89]) compared to those with more complete reporting (Table A17). Chlamydia and gonorrhoea prevalence were higher in studies with sample sizes less than 100 (aPR_{CT}: 1.37 [1.06-1.76]; aPR_{NG}: 1.93 [1.32-2.82]) than those with higher precision. There was no evidence that other critical appraisal criteria influenced prevalence estimates (Table A17).

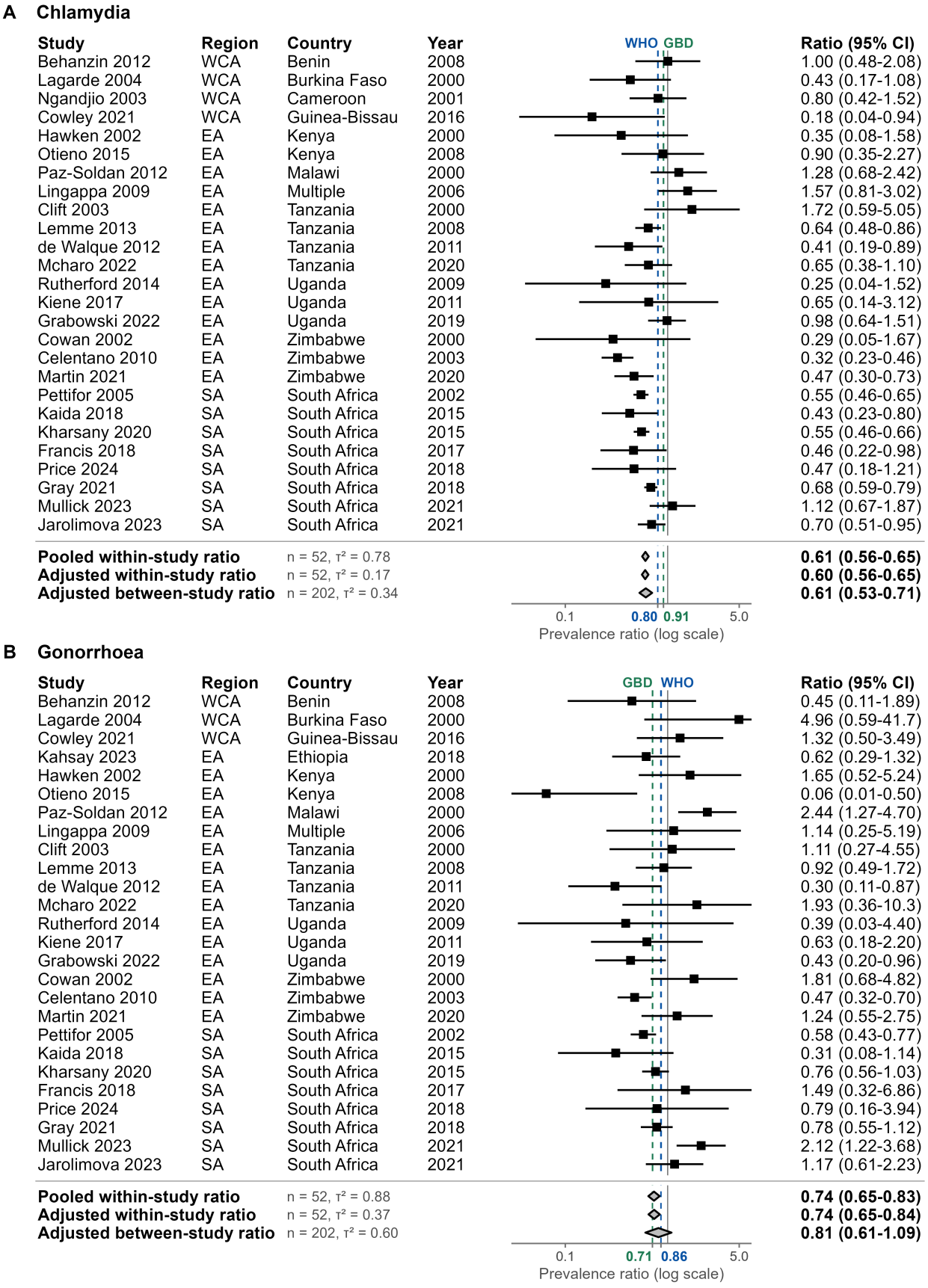


Figure 2.4: Male-to-female sexually transmitted infection prevalence ratios in SSA

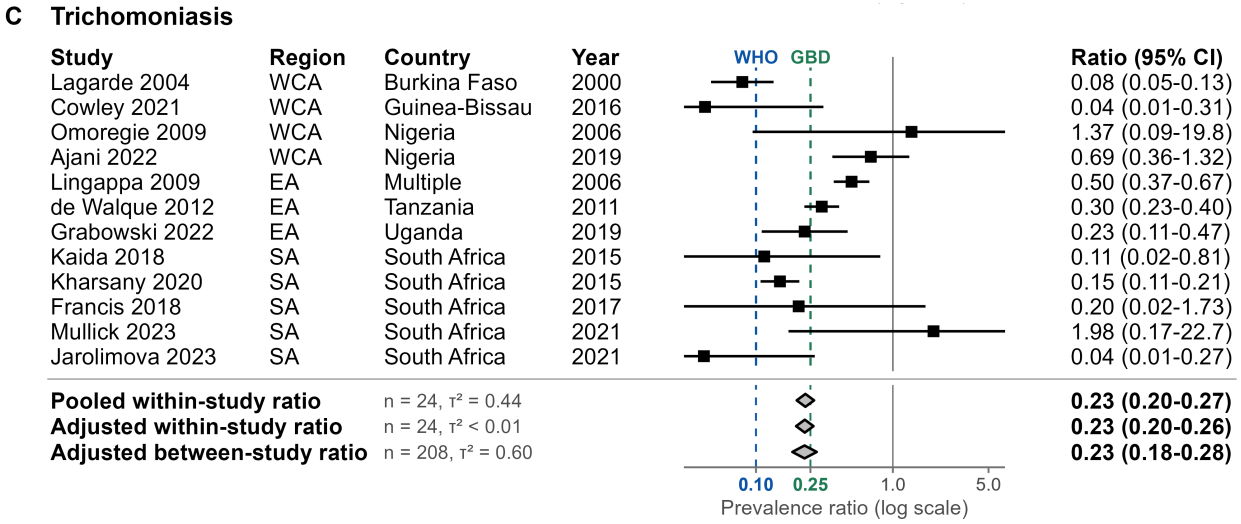


Figure 2.4: Male-to-female sexually transmitted infection prevalence ratios in SSA (continued)

Study observations and aggregate estimates of male-to-female prevalence ratios for (A) chlamydia, (B) gonorrhoea and (C) trichomoniasis. Ratios estimated using log-binomial generalised linear mixed-effects models per infection. Models for within-study ratios used study observations among both sexes and between-study ratios used all study observations. All models included sex as a fixed effect and study-level random intercepts. Pooled ratio models did not adjust for additional covariates. Adjusted ratio models included fixed effects for region, year, age-group, HIV-status, diagnostic test, and population group (between-study only). Points and error lines depict ratios and 95% CIs for studies with observations among both sexes. Diamonds depict population-weighted mean ratios and 95% CIs for SSA, with adjusted ratios reported for 2020. Blue dotted lines and x-axis labels represent WHO global male-to-female prevalence ratios.[227,230] Green dotted lines and x-axis labels represent the GBD study male-to-female prevalence ratios.[234] X-axes are truncated. n: number of observations, τ^2: variance for study random intercept, EA: Eastern Africa, SA: Southern Africa, WCA: Western and Central Africa.

2.4 Discussion

In this chapter, I estimated the prevalence of chlamydia, gonorrhoea and trichomoniasis among general populations in SSA between 2000 and 2024. The burden of curable STIs was higher among females (6.9% chlamydia, 1.8% gonorrhoea, and 7.6% trichomoniasis in 2020) than males (4.2%, 1.5%, and 1.7% respectively), which exceeded global prevalence estimates (3.2%, 0.7%, and 2.7% for both sexes in 2020, respectively).[4] Geographic variation was substantial; prevalence of chlamydia and gonorrhoea was highest in Southern Africa, followed by Eastern Africa and then Western and Central Africa. Trichomoniasis prevalence was similar across sub-regions for females but was highest in Eastern Africa for males. The prevalence of chlamydia increased considerably in Eastern and Southern Africa over the study period, while gonorrhoea was stable, and trichomoniasis decreased in Southern Africa.

My central estimates of STI prevalence in 2020 differed from those reported by WHO for the African region and GBD for SSA.[4,234] For females, estimates were higher for chlamydia (6.9% vs. WHO:5.5%,

GBD:4.5%), similar for gonorrhoea (1.8% vs. WHO:1.6%, GBD:1.7%), and lower for trichomoniasis (7.6% vs. WHO:12.0%, GBD:12.6%). For males, estimates were higher for chlamydia (4.2% vs. WHO:4.0%, GBD:2.5%), gonorrhoea (1.5% vs. WHO:1.2%, GBD:1.2%), and trichomoniasis (1.7% vs. WHO:1.3%, GBD:1.5%).[4,234] These differences reflect my broader study inclusion criteria, incorporation of more recent studies, and analysis of temporal trends to reflect sub-regional changes in prevalence. Compared to existing global male-to-female prevalence ratios, my ratio estimates for SSA were lower for chlamydia (WHO: 0.80 and GBD: 0.91), and intermediate for gonorrhoea (WHO: 0.86 and GBD: 0.71) and trichomoniasis (WHO: 0.10 and GBD: 0.25; Figure 3),[227,230,234] though differences were generally not statistically significant. Future estimation exercises should prioritise using region-specific ratios to capture local epidemiologic differences, although sparse male prevalence data can hinder ratio estimation.

The spatial distribution of chlamydia and gonorrhoea prevalence mirrors that of HIV prevalence in SSA, characterised by highest burden in Southern Africa, followed by Eastern Africa, and lowest in Western and Central Africa.[295] This is likely due to shared behavioural risk factors and biological synergies that facilitate co-infection.[296] Despite evidence suggesting that trichomoniasis increases HIV transmission and acquisition risk,[297] prevalence variations were not observed across sub-regions. This could reflect limitations in study availability and diagnostics, missing covariates, or incomplete adjustment for covariates such as age.[158] Greater integration of STI and HIV services remains important for their effective mitigation in high-burden areas.[1]

The increasing prevalence of chlamydia in Eastern and Southern Africa and decreasing prevalence of trichomoniasis in Southern Africa have not been documented previously. Previous STI burden assessments reported stable prevalence over time for chlamydia and gonorrhoea in Kenya and South Africa[238,239] or did not estimate time trends for curable STIs in SSA.[227,240–242,275] Temporal trend estimates were highly uncertain in this analysis, predominantly due to high heterogeneity across included studies. For chlamydia, even the lower bound of my estimates suggests an upward trend. For trichomoniasis, however, declines in prevalence warrant cautious interpretation due to the transition from wet mount microscopy to more sensitive NAAT in recent studies.

Increases in chlamydia prevalence are particularly concerning given the severe, long-term health consequences of untreated infection, including chronic pelvic pain, infertility, ectopic pregnancy, and other adverse pregnancy outcomes, along with an increased risk of HIV transmission.[1] Co-infection with HIV and other STIs additionally compounds the risk of adverse maternal and neonatal outcomes,[298]

making prevention and control strategies particularly critical in high-burden settings.

Globally, there are major concerns about the impact of rising antimicrobial resistance on gonorrhoea incidence.[181] I did not find evidence of increasing gonorrhoea prevalence in SSA. Data on antimicrobial resistance are sparse in the African region, but available evidence suggests that current first-line and dual therapies continue to be effective, despite resistance to older antibiotics.[182]

Differences in prevalence by sex arise from an interplay of biological, behavioural, and structural factors. Men are more likely to experience symptoms of STI infection than women, leading to higher rates of treatment-seeking.[7,33] Men also have a substantially shorter duration of trichomoniasis infection than women; despite potentially comparable incidence, faster clearance in men results in particularly low prevalence and thus a low male-to-female prevalence ratio.[7] Disparities in treatment coverage, due to healthcare access barriers, sociocultural norms, and stigma,[1] may also influence sex-prevalence differences. These factors, which may have changed over time, drive sub-regional variations in both prevalence and sex-prevalence ratios, but data are limited to attribute effects.

My analysis was primarily limited by sparse and heterogenous data. The systematic search identified only 211 studies in 28 of 48 SSA countries. There were particularly few studies in Central Africa (12 studies in 6 countries) and among men (37 studies in 15 countries). I was therefore unable to generate estimates specific to Central Africa or to determine whether temporal prevalence trends differed by sex. Estimates for Southern Africa relied predominantly on studies conducted in South Africa (51 of 59 studies in the sub-region), which limited my ability to assess the representativeness of both these studies and my estimates for the broader sub-region.

Limitations related to study representativeness and reporting, and adjustments made in my analysis, may have influenced my estimates. Given the lack of population-representative studies, I used ANC attendees as the reference category for my prevalence estimates. At least 65% of studies focused exclusively on sexually active individuals, including antenatal, family planning, and gynaecology clinic attendees, and HIV/STI prevention trial participants. I considered these groups to be representative of the general population, which may result in overestimating STI prevalence compared to the total adult population. Variability in reporting across studies hindered my adjustment of estimates for HIV prevalence, granular and mutually exclusive age groups, or urban/rural location, which likely influenced sub-regional heterogeneities. Furthermore, my adjustments for diagnostic test performance may have overestimated prevalence, particularly for trichomoniasis, due to the poor sensitivity of widely used wet

mount microscopy. My reliance on previously published WHO performance data, without accounting for improvements in diagnostic accuracy over time, may have resulted in over-adjusting recent study observations and thus overestimating temporal trends.

Despite these limitations, this systematic review provides a comprehensive analysis of chlamydia, gonorrhoea, and trichomoniasis prevalence in SSA. I have addressed gaps in previous prevalence estimates by examining temporal trends and sub-regional variation, while incorporating studies from a range of populations considered representative of the general population. My multiple analytical approaches yielded similar male-to-female prevalence ratio estimates, and my extensive sensitivity analyses of diagnostic test performance adjustments have strengthened the robustness of my findings.

The analysis underscores an urgent need to strengthen STI control in SSA, particularly in areas with a high burden and rising chlamydia prevalence. However, more comprehensive surveillance through population-representative studies, sentinel surveys, and research in underrepresented countries and population groups is essential for designing targeted mitigation strategies.[222] These data will enable more strategic allocation of resources and interventions at national and sub-national levels.

In conclusion, I estimated a high and geographically varied prevalence of three curable STIs in SSA, with concerning increases in chlamydia prevalence over time. Male-to-female prevalence ratios for SSA and its sub-regions differed from existing WHO and GBD global ratios, emphasising the need to account for epidemiologic variations in future STI burden estimates and control strategies. Strengthening aetiologic surveillance through regular sentinel surveys and population-representative prevalence studies, with greater focus on male populations and underrepresented geographies, is essential for progressing toward WHO targets and reducing the STI burden in SSA.

Chapter 3

Aetiology of vaginal discharge, urethral discharge, and genital ulcer in sub-Saharan Africa

3.1 Introduction

In SSA, access to laboratory or point-of-care aetiologic diagnostics is limited and syndromic management remains the standard of care.[117] The approach enables rapid treatment, but has several limitations. Many STIs remain untreated due to high rates of asymptomatic infection, and syndromic algorithms have suboptimal diagnostic performance, despite efforts to incorporate evolving STI epidemiology.[116] WHO recommends regular national re-assessment of syndrome aetiologies to ensure the relevance of syndromic algorithms,[222,224] yet few countries report including these assessments in STI surveillance plans.[139] These factors collectively lead to unnecessary, incorrect, or missed treatment for STIs, which is particularly concerning amid rising AMR.[116]

WHO published updated guidelines for symptomatic STI management in 2021, which were informed by global systematic reviews of studies assessing the diagnostic performance of different syndromic management algorithms.[117] Although these guidelines accounted for changes over time in the underlying causes of each syndrome, they lacked a comprehensive review and quantification of the distribution of aetiologies among symptomatic populations in different geographic regions. This information would support modifying syndromic management protocols to align with local epidemiology, and thus improve

treatment outcomes.

In this chapter, I characterise the aetiologies for three prevalent STI symptoms in SSA: vaginal discharge, urethral discharge, and genital ulcer. I perform a systematic review and meta-analysis to estimate the distribution of aetiologies for each symptom, investigate their spatiotemporal changes, and evaluate variation according to sex, HIV status, and age.

3.2 Methods

3.2.1 Search strategy

I systematically searched Embase (Ovid), MEDLINE (Ovid), Global Health (Ovid) and Web of Science from database inception to 20 December 2023 for studies assessing the aetiology of vaginal discharge, urethral discharge, and genital ulcer in SSA. Search term domains included relevant terms and synonyms for "symptoms", "infections" and "sub-Saharan Africa" (Table B1). I also conducted a comprehensive grey literature search up to 20 December 2023 using the same search term domains. Sources included websites and reports by the WHO, UNAIDS, and Ministries of Health, and conference abstracts published between 2000 and 2023 from the STI & HIV World Congress, International AIDS Society, and International Conference on AIDS and STIs in Africa. I contacted authors of relevant conference abstracts to enquire about potential unpublished data.

3.2.2 Eligibility criteria and study selection

I included studies that contained empirical data on the proportion of women with vaginal discharge (VD) diagnosed with bacterial vaginosis (BV), any *Candida* species (CS), *C. albicans* (CA), *C. trachomatis* (CT), *M. genitalium* (MG), *N. gonorrhoeae* (NG), *T. vaginalis* (TV), or unknown aetiology (negative aetiologic test results); proportion of men with urethral discharge (UD) diagnosed with CT, MG, NG, TV, or unknown aetiology; and proportion of men and women with genital ulcer (GU) diagnosed with *H. ducreyi* (HD), herpes simplex virus of unspecified type (HSV) or types 1 or 2 (HSV-1 or HSV-2), lymphogranuloma venereum (LGV) due to *C. trachomatis* serovars L1-L3, *T. pallidum* (TP), or unknown aetiology. Since BV and candidiasis are not classified as STIs, but are common causes of vaginal discharge, the broader term of reproductive tract infections (RTIs) is used in this study.

Studies were eligible if: 1) they included participants who were symptomatic at the time of testing, defined by the presence of either self-reported or clinician-evaluated abnormal vaginal discharge, urethral

discharge, or genital ulcer, 2) participants were aged 10 years and older, 3) the sample size was at least 10, and 4) the diagnostic methodology for each infection was described and assessed as valid. While the primary interest was studies among sexually active adult populations, I used a minimum age of 10 years to avoid excluding studies which enrolled both eligible sexually active adolescent and adult participants; in such studies (number of studies (N) = 18), the majority of participants were adults. I assessed diagnostic method validity by cross-referencing its inclusion in either current or previous published recommendations.[117,140,141,144,145,299,300] I used the UN M49 standard to define SSA and its sub-regions (Table B2).[282] I excluded: 1) qualitative studies, case reports, commentaries, reviews, mathematical modelling studies, and longitudinal and randomised controlled studies reporting outcomes post-baseline only, and 2) studies published in languages other than English, French, or Portuguese.

Search results were managed and de-duplicated in Covidence systematic review software (Veritas Health Innovation, Melbourne, Australia).[283] I worked with co-authors (MKW, MW, OE) to independently double-screen titles and abstracts for eligibility and assess full text articles for inclusion. We resolved any discrepancies through consensus or with a third co-author.

3.2.3 Data extraction

I worked with co-authors (MKW, OE) to independently double-extract data from each study, with discrepancies resolved through consensus or with a third co-author. A study observation was the proportion of symptomatic individuals diagnosed with a given RTI or, if not directly reported, the numerator and denominator to calculate the proportion. In cases of RTI co-infection, we extracted each infection separately, potentially causing the total infected proportion to exceed 1 when aggregating all observations for a given population. We extracted observations with stratification by population type (symptomatic clinic attendees; general populations; higher-risk general populations, such as truck drivers, mineworkers, soldiers, and bar workers; or key populations, such as FSW and MSM), country, year or time-period if not stratified by year, sex, HIV status, and age group as available. Strata subsamples of fewer than 10 participants were excluded. We extracted information on study characteristics, participant characteristics, and diagnostic methods (Table B3). I also prepared and extracted unpublished data from two databases identified during the search (Text B.1.2, Table B4).

If multiple articles reported outcome(s) from the same study, we preferentially retained observations from the largest sample or, if samples were the same size, from the article with the largest number of

RTIs tested. If the sample sizes and number of RTIs were equal, we retained the most recent article. When available, I used observations tabulated directly from databases and excluded corresponding published articles to avoid duplication.

If a study reported outcomes for self-reported and clinician-evaluated symptoms in the same population, we preferentially extracted observations for the latter. If multiple diagnostic tests were conducted, we preferentially extracted outcomes based on the most accurate test method for the pathogen and symptom (Tables B5 - B7). When multiple diagnostic tests were used for syphilis among those with genital ulcer, we preferentially extracted observations for tests using ulcer swab specimens over serology.[140] When syphilis serology was used, observations from the combination of non-treponemal and treponemal tests were prioritized when available. The proportion with "unknown aetiology" was only extracted from studies with observations for three or more RTI pathogens, regardless of the specific pathogens tested.

3.2.4 Data analysis

To adjust reported proportions for diagnostic test performance, I classified diagnostic tests into broad categories with sensitivity and specificity estimates compiled from literature, prioritising characteristics published by the WHO (Text B.1.3; Tables B5 - B7).[145,230] I used a Bayesian approach to estimate the true proportion of symptomatic individuals with a given RTI.[284,285] I then calculated adjusted numerators and denominators based on the mean and standard error of the true proportion and used these to pool observations (Text B.1.3).

The diagnosed proportion for each infection was estimated using observations among adults with non-stratified or unmeasured HIV status. I estimated time trends in the diagnosed proportion by region via generalised linear mixed-effects meta-regressions for each symptom.[301] I specified models a priori to include fixed effects for RTI, the interaction of RTI and year (midpoint date of data collection measured as continuous calendar year), and the interaction of RTI and sex (genital ulcer only), random intercepts and slopes per year for the interaction of RTI and region (Central and Western, Eastern, or Southern Africa), and observation-level random intercepts to account for between-study heterogeneity. I weighted pooled regional means by sex-matched regional population estimates for adults 15 years and older in 2015 from the UN World Population Prospects 2022.[287]

To assess the effects of HIV status and age, I extended the meta-regressions described above to include fixed effects for either HIV status (HIV-positive, HIV-negative) or age group ($<$25 years, $\geq$25 years).

Random slopes were however not included, due to their low standard deviation in the main analysis.

3.2.5 Sensitivity analyses and risk of bias

To assess sensitivity to the diagnostic test adjustments, I compared trend estimates between models using observations as reported and models using observations adjusted for diagnostic test performance. This comparison was conducted for all diagnostic test types (all RTIs) and separately for NAAT (all RTIs, excluding BV, CA, and CS).

I adapted the Joanna Briggs Institute critical appraisal tool for prevalence studies to assess study design (objective of the study), selection bias (clarity of inclusion criteria, appropriateness of recruitment method, adequacy of participation, and detail of participant characterisation), measurement bias (objectivity in symptom definition, consistency of diagnostic methodology, and avoidance of misclassified results), precision (sufficiency of sample size), and reporting quality (ambiguity of results; Table B8).[289] My co-authors (MKW, OE) and I independently double-assessed the 10 criteria for each study, and resolved discrepancies through consensus. I assessed the association of these criteria with the RTI proportions by extending meta-regressions to include fixed effects for each criterion.

Unless stated otherwise, all analyses use observations adjusted for diagnostic test performance. For studies that collected data from countries in multiple regions, but did not report data stratified by country or region, I classified them using the region from which most participants were recruited. I report primary meta-regression pooled results as model predictions for the year 2015, representing the most recent quinquennium within the timespan of substantial available data. Results are presented as means with 95% confidence intervals (95% CIs). Model coefficients are presented as adjusted odds ratios (aORs), with confidence intervals calculated on the log odds scale before exponentiation. I assessed study observation heterogeneity per model as the percentage of total variance attributed to observation-level random effects.[302] Analyses were conducted in R version 4.2.3, Bayesian adjustments for diagnostic test performance were implemented in `rstan` version 2.26.21,[303] and mixed-effects logistic meta-regression models were implemented in `glmmTMB` version 1.1.8.[304]

My study was pre-registered on PROSPERO (CRD 42022348045)[305] and reported per the PRISMA guideline (Table B9).[294] The Imperial College Research Ethics Committee approved analysis of secondary data for this study (ICREC 6389606).

3.3 Results

3.3.1 Search results and scope

A total of 7 780 records were identified through the database search, of which 3 362 were duplicates and 4418 were screened (Figure 3.1). Of these, 745 full-text articles were assessed for eligibility and 199 were included. An additional 40 records were identified through grey literature sources and citation searching, of which 5 full-text articles and two databases were assessed for eligibility and included. The two included databases were from the National Institute for Communicable Diseases (NICD) periodic aetiologic prevalence surveys among symptomatic primary healthcare clinic attendees in South Africa between 2006 and 2022,[306–308] and the Centre for Sexual Health and HIV/AIDS Research (CeSHHAR) female sex worker population size estimation study in Zimbabwe in 2017.[309]

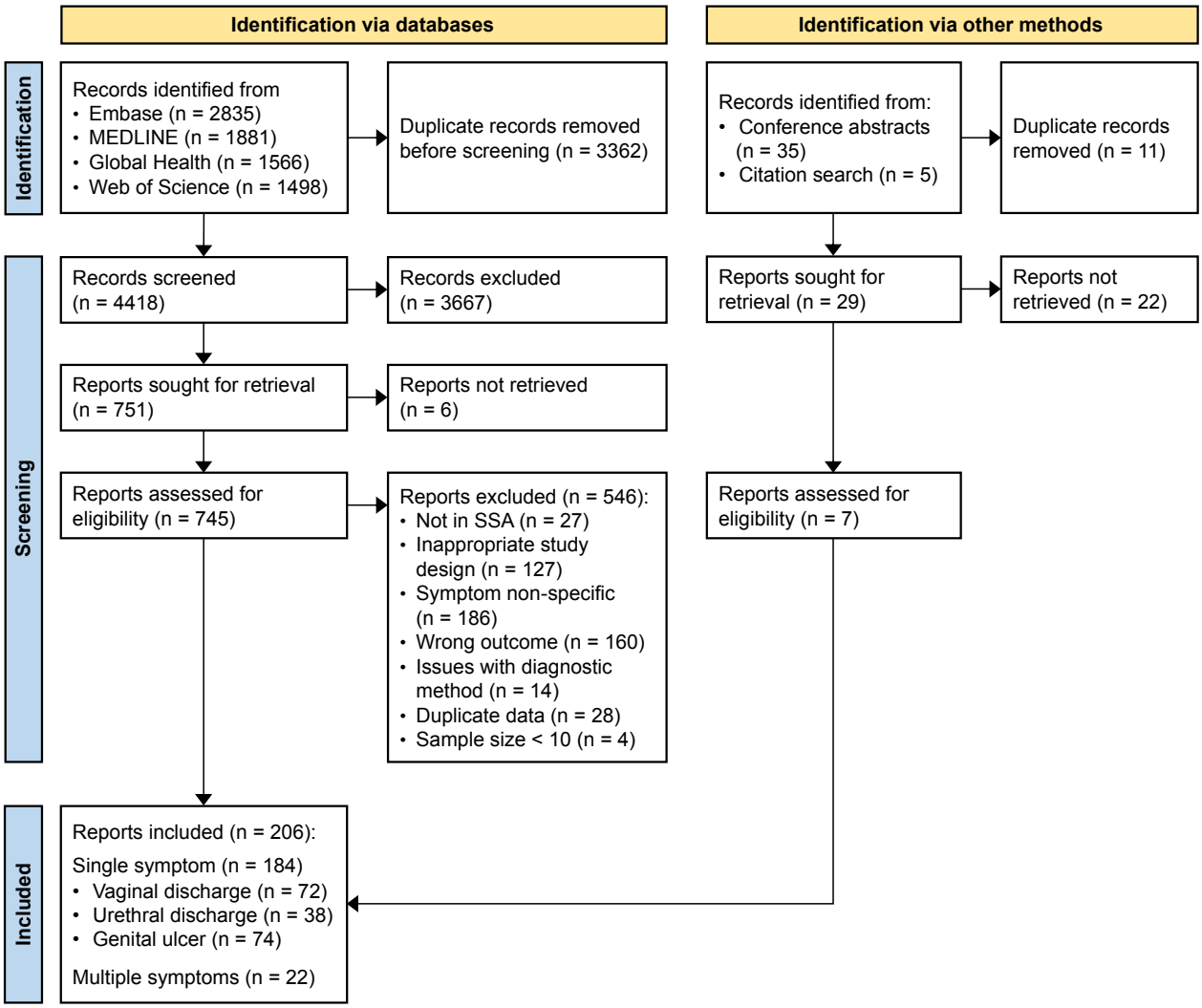


Figure 3.1: Study selection flowchart

Records: titles/abstracts; Reports: full-text articles or databases.

Table 3.1: Summary of characteristics for included studies

	Vaginal discharge	Urethral discharge	Genital ulcer	All symptoms
	$N_{VD} = 87$	$N_{UD} = 55$	$N_{GU} = 80$	$N = 190$
Participant characteristics				
Population group[1]				
Symptomatic clinic attendees	60 (69.0%)	49 (89.1%)	63 (78.8%)	145 (76.3%)
General	25 (28.7%)	2 (3.6%)	6 (7.5%)	32 (16.8%)
Higher-risk general	2 (2.3%)	3 (5.5%)	7 (8.8%)	10 (5.3%)
Key populations	6 (6.9%)	1 (1.8%)	8 (10%)	14 (7.4%)
Youth	16 (18.4%)	6 (10.9%)	6 (7.5%)	21 (11.1%)
Sex				
Male	-	55 (100.0%)	20 (25.0%)	56 (29.5%)
Female	87 (100.0%)	-	21 (26.3%)	86 (45.3%)
Non-stratified	-	-	39 (48.8%)	48 (25.3%)
Mean or median age				
$<$ 25 years	8 (9.2%)	2 (3.6%)	1 (1.3%)	8 (4.2%)
$\geq$ 25 years	18 (20.7%)	15 (27.3%)	21 (26.3%)	49 (25.8%)
NR	61 (70.1%)	38 (69.1%)	58 (72.5%)	133 (70%)
HIV status				
Positive	1 (1.1%)	1 (1.8%)	3 (3.8%)	5 (2.6%)
Negative	4 (4.6%)	1 (1.8%)	1 (1.3%)	5 (2.6%)
Non-stratified	32 (36.8%)	19 (34.5%)	51 (63.8%)	81 (42.6%)
NR	50 (57.5%)	34 (61.8%)	25 (31.3%)	99 (52.1%)
HIV prevalence				
$<$ 25%	12 (13.8%)	4 (7.3%)	5 (6.3%)	19 (10%)
$\geq$ 25%	10 (11.5%)	7 (12.7%)	39 (48.8%)	46 (24.2%)
NR	65 (74.7%)	44 (80.0%)	36 (45.0%)	125 (65.8%)
Study characteristics				
Region[1,2]				
Central Africa	4 (4.6%)	2 (3.6%)	2 (2.5%)	7 (3.7%)
Eastern Africa	35 (40.2%)	32 (58.2%)	50 (62.5%)	93 (48.9%)
Southern Africa	14 (16.1%)	11 (20%)	23 (28.8%)	45 (23.7%)
Western Africa	33 (37.9%)	9 (16.4%)	8 (10%)	47 (24.7%)
Multiple (not stratified)	1 (1.1%)	1 (1.8%)	1 (1.3%)	2 (1.1%)
Study midpoint year[1,3]				
$<$1990	8 (9.2%)	10 (18.2%)	18 (22.5%)	33 (17.4%)
1990 - 1999	21 (24.1%)	18 (32.7%)	36 (45.0%)	63 (33.2%)
2000 - 2009	28 (32.2%)	15 (27.3%)	19 (23.8%)	52 (27.4%)
2010 - 2023	33 (37.9%)	13 (23.6%)	11 (13.8%)	48 (25.3%)
Study design				
Cross sectional	73 (83.9%)	46 (83.6%)	63 (78.8%)	157 (82.6%)
Cohort baseline	11 (12.6%)	8 (14.5%)	12 (15%)	25 (13.2%)
RCT baseline	3 (3.4%)	1 (1.8%)	5 (6.3%)	8 (4.2%)
Sampling method				
Non-probability based	72 (82.8%)	51 (92.7%)	73 (91.3%)	169 (88.9%)
Probability based	9 (10.3%)	3 (5.5%)	6 (7.5%)	13 (6.8%)
NR	6 (6.9%)	1 (1.8%)	1 (1.3%)	8 (4.2%)

Continued...

	Vaginal discharge	Urethral discharge	Genital ulcer	All symptoms
	N_{VD} = 87	N_{UD} = 55	N_{GU} = 80	N = 190
Sample size[4]				
< 100	26 (29.9%)	16 (29.1%)	34 (42.5%)	68 (35.8%)
≥ 100	61 (70.1%)	39 (70.9%)	46 (57.5%)	122 (64.2%)
Pathogen detected[1,5]				
BV	28 (32.2%)	-	-	28 (14.7%)
CA	9 (10.3%)	-	-	9 (4.7%)
CS	34 (39.1%)	-	-	34 (17.9%)
CT	35 (40.2%)	31 (56.4%)	-	56 (29.5%)
HD	-	-	58 (72.5%)	58 (30.5%)
HSV	-	-	31 (38.8%)	31 (16.3%)
HSV-1	-	-	15 (18.8%)	15 (7.9%)
HSV-2	-	-	23 (28.8%)	23 (12.1%)
LGV	-	-	11 (13.8%)	11 (5.8%)
MG	7 (8.0%)	8 (14.5%)	-	13 (6.8%)
NG	42 (48.3%)	50 (90.9%)	-	78 (41.1%)
TP	-	-	68 (85.0%)	68 (35.8%)
TV	60 (69.0%)	28 (50.9%)	-	77 (40.5%)
None[6]	15 (17.2%)	12 (21.8%)	39 (48.8%)	59 (31.1%)
Number pathogens extracted per study				
1	30 (34.5%)	20 (36.4%)	23 (28.8%)	57 (30.0%)
2 - 3	29 (33.3%)	27 (49.1%)	40 (50.0%)	85 (44.7%)
4 - 6	28 (32.2%)	8 (14.5%)	17 (21.3%)	48 (25.3%)

[1]The same study is included in more than one subcategory when it reports across different variable levels or multiple variable levels are relevant. [2]Regions classified according to UN M49 Standard. [3]Mid-point year between start and end dates of data collection. If dates were not reported, year of publication was used as proxy. [4]Sample size defined as the number of symptomatic individuals tested in the study. If a study used different samples across tests, the largest sample size was reported. [5]Number of studies that tested for and reported results for a given pathogen; studies that mentioned testing but did not report results were not counted. [6]Unknown aetiology reported for studies with three or more pathogens extracted. N: number of studies, BV: Bacterial vaginosis, CA: *Candida albicans*, CS: *Candida* species (any), CT: *Chlamydia trachomatis*, HD: *Haemophilus ducreyi*, HSV: Herpes simplex virus (unspecified), HSV-1: Herpes simplex virus type 1, HSV-2: Herpes simplex virus type 2, LGV: lymphogranuloma venereum, MG: *Mycoplasma genitalium*, NG: *Neisseria gonorrhoeae*, TP: *Treponema pallidum*, TV: *Trichomonas vaginalis*, None: unknown aetiology. NR: Not reported.

Overall, 206 reports (204 articles and 2 databases) were included from 190 independent studies (N_{VD}=87, N_{UD}=55, N_{GU}=80) spanning 1969 to 2022 (Tables 3.1 and B10). Of these, 166 studies focused on a single symptom, 16 studies on two symptoms, and 8 studies on all three symptoms. Studies were conducted in 32 of 48 SSA countries included in the search, with a median of 5 studies per country. Most were in Eastern Africa (N_{VD}=35/87, N_{UD}=32/55, N_{GU}=50/80) and few were in Central Africa (N_{VD}=4/87, N_{UD}=2/55, N_{GU}=2/80). Studies were predominantly in South Africa (N_{VD}=13/87, N_{UD}=10/55, N_{GU}=18/80), along with Nigeria (N_{VD}=15/87) for vaginal discharge and Kenya (N_{GU}=18/80) for genital ulcer (Figure B1). Most studies were after 2000 (N_{VD}=61/87, N_{UD}=28/55) for vaginal

discharge and urethral discharge and between 1990-1999 (N_{GU}=36/80) for genital ulcer. Studies were predominantly among symptomatic clinic attendees (N_{VD}=60/87, N_{UD}=49/55, N_{GU}=63/80) and among individuals with non-stratified HIV status (N_{VD}=32/87, N_{UD}=19/55, N_{GU}=51/80). Few studies reported HIV prevalence (N_{VD}=22/87, N_{UD}=11/55, N_{GU}=44/80) or the mean or median age (N_{VD}=26/87, N_{UD}=17/55, N_{GU}=22/80) of participants. Studies were mostly cross-sectional (N_{VD}=73/87, N_{UD}=46/55, N_{GU}=63/80). Most had sample sizes ≥100 (N_{VD}=61/87, N_{UD}=39/55, N_{GU}=46/80) and tested for more than one pathogen (N_{VD}=57/87, N_{UD}=35/55, N_{GU}=57/80). The most frequently assessed aetiologies were TV (N_{VD}=60/87) and NG (N_{VD}=42/87) for vaginal discharge; NG (N_{UD}=50/55) and CT (N_{UD}=31/55) for urethral discharge; and TP (N_{GU}=68/80) and HD (N_{GU}=58/80) for genital ulcer. Study characteristics varied by sub-analysis (Tables B11, B12, B13).

3.3.2 Regional aetiologic trends

In 2015, CS and BV, rather than STIs, were the primary aetiologies for vaginal discharge (Figure 3.2A, Table 3.2A). The proportion of VD cases with CS was 69.4% (95% CI: 44.3-86.6%; number of observations per symptom (n): n_{VD}=50), with BV was 50.0% (32.3-67.8%, n_{VD}=39), and with CA was 31.5% (12.4-59.9%, n_{VD}=9). CT (16.2% [8.6-28.5%], n_{VD}=50) and TV (12.9% [7.7-20.7%], n_{VD}=80) were the most prominent STIs, while a relatively low proportion of cases were due to NG (6.6% [3.3-12.7%], n_{VD}=63) and MG (5.4% [1.8-14.8%], n_{VD}=20). A high proportion (25.1% [10.7-48.4%], n_{VD}=28) of cases did not have an identified aetiology, despite apparent high levels of co-infection. Diagnosed proportions increased over time for CS (aOR per year: 1.10 [95% CI: 1.05-1.15]), decreased for TV (aOR: 0.94 [0.91-0.97]), and were relatively constant for other RTIs (Table B14). There were limited data to assess temporal trends for MG, for which most studies were conducted after 2005.

For urethral discharge, NG has remained the primary aetiology from the 1970s to present (Figure 3.2B, Table 3.2B). In 2015, NG was diagnosed in 77.1% (95% CI: 68.1-84.1%, n_{UD}=68) of urethral discharge cases. CT was the predominant (21.9% [15.4-30.3%], n_{UD}=48) non-gonococcal cause of urethral discharge, followed by MG (5.5% [3.7-8.3%], n_{UD}=29) and TV (2.8% [1.8-4.2%], n_{UD}=46). No aetiology was detected in 10.0% (6.8-14.5%, n_{UD}=26) of cases. The odds of MG (aOR: 0.95 [95% CI: 0.91-1.00]) and TV (aOR: 0.93 [0.90-0.96]) decreased over time, with no significant change for NG (aOR: 1.01 [0.99-1.03]) or CT (aOR: 1.02 [0.99-1.06]; Table B14). The proportion per year with unknown aetiology also decreased (aOR: 0.95 [0.91-0.99]).

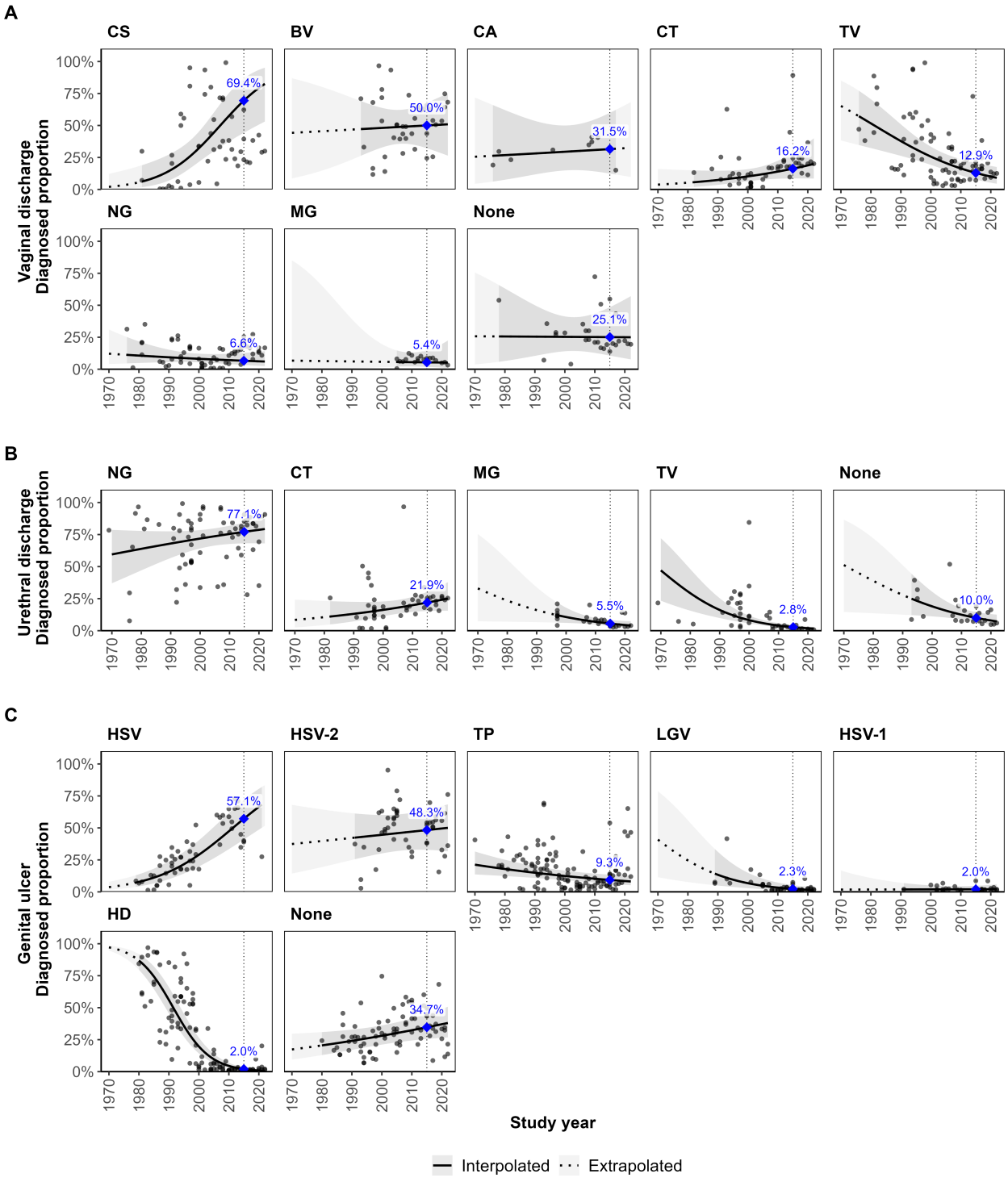


Figure 3.2: Estimated diagnosed proportion per pathogen among symptomatic adults in SSA, 1970-2023

Diagnosed proportion per pathogen among adults of non-stratified or unmeasured HIV status symptomatic with (A) vaginal discharge, (B) urethral discharge, and (C) genital ulcer. Proportions estimated via generalised linear mixed-effects model for each symptom. Lines and shaded areas represent sex-matched population-weighted mean proportions and 95% CIs. Solid lines and darker shading denote estimates and CIs within the observed data range (interpolated), while dotted lines and lighter shading indicate estimates and CIs beyond that time frame (extrapolated). Blue points and labels represent mean proportions in 2015. Vertical dotted lines are through the year 2015. Grey points represent study observations adjusted for diagnostic test performance.

In 2015, HSV-2 was the leading cause of genital ulcer (Figure 3.2C, Table 3.2C). The diagnosed proportion of cases for HSV was 57.1% (95% CI: 40.9-71.9%, n_{VD}=55), HSV-2 was 48.3% (32.9-64.1%, n_{GU}=47), and HSV-1 was 2.0% (0.8-4.7%, n_{UD}=31). TP was the second most prevalent aetiology (9.3% [6.4-13.4%], n_{GU}=117), followed by LGV (2.3% [1.0-5.3%], n_{GU}=43) and HD (2.0% [1.2-3.3%], n_{GU}=107). No aetiology was identified in 34.7% (25.5-45.1%, n_{GU}=83) of cases. The distribution of genital ulcer pathogens changed substantially over time. Since most observations for HSV-1 (97%) and HSV-2 (85%) occurred after the year 2000, trends are more reliably assessed for unspecified HSV. In 1980, estimates were highest for HD (86.9% [79.7-91.8%]) and lowest for HSV (7.8% [4.3-13.7%]; Figure 3.2). The odds of HSV increased per year (aOR: 1.08 [95% CI: 1.06-1.11]) and decreased for HD (aOR: 0.85 [0.83-0.86]), LGV (aOR: 0.93 [0.89-0.96]), and TP (aOR: 0.98 [0.96-0.99]; Table B14C). The odds of having no identified aetiology (aOR: 1.02 [1.00-1.04]) increased over time. Men with genital ulcer had higher odds (aOR: 1.98 [1.27-3.08]) of HD diagnosis than women, but sex was not a significant predictor for other aetiologies.

Across all three symptoms, there was negligible evidence of sub-regional variation in the diagnosed proportion per pathogen over time (Figure B2, Table B14). Study observations were heterogeneous for all three symptoms, especially vaginal discharge. The percentage of total variance attributed to observation-level random effects was 43.0% for VD, 22.3% for UD, and 25.0% for GU (Table B14).

3.3.3 Factors associated with symptom aetiology

For each symptom, HIV status and age were significantly associated with the diagnosis of several RTIs, although the infections with the largest proportions per symptom generally remained the same for all sub-groups (Table 3.2). The main exception was for women with HIV, where the leading causes of vaginal discharge were BV (45.5% [95% CI: 34.4-56.9%], n_{VD}=20), CS (31.4% [19.9-45.8%], n_{VD}=17), and TV (17.1% [11.7-24.4%], n_{VD}=22) in 2015.

For vaginal discharge, the odds were higher among women with HIV than women without HIV for diagnosis with MG (aOR: 2.3 [95% CI: 1.6-3.4], n_{VD}=36), TV (2.2 [1.6-2.9], n_{VD}=45), BV (1.8 [1.3-2.5], n_{VD}=42) and NG (1.6 [1.2-2.2], n_{VD}=41), and lower for CS (0.5 [0.3-0.7], n_{VD}=34; Figure 3.3A, Table B15). No HIV-stratified observations were available for CA. The odds were higher among women <25 years than ≥25 years for diagnosis with CT (2.4 [1.7-3.3], n_{VD}=38), MG (2.0 [1.3-3.0], n_{VD}=31), CS (1.8 [1.3-2.7], n_{VD}=37), and NG (1.7 [1.2-2.3], n_{VD}=43), and lower for those without an identified aetiology (0.7 [0.5-0.9], n_{VD}=36; Figure 3.4A, Table B16).

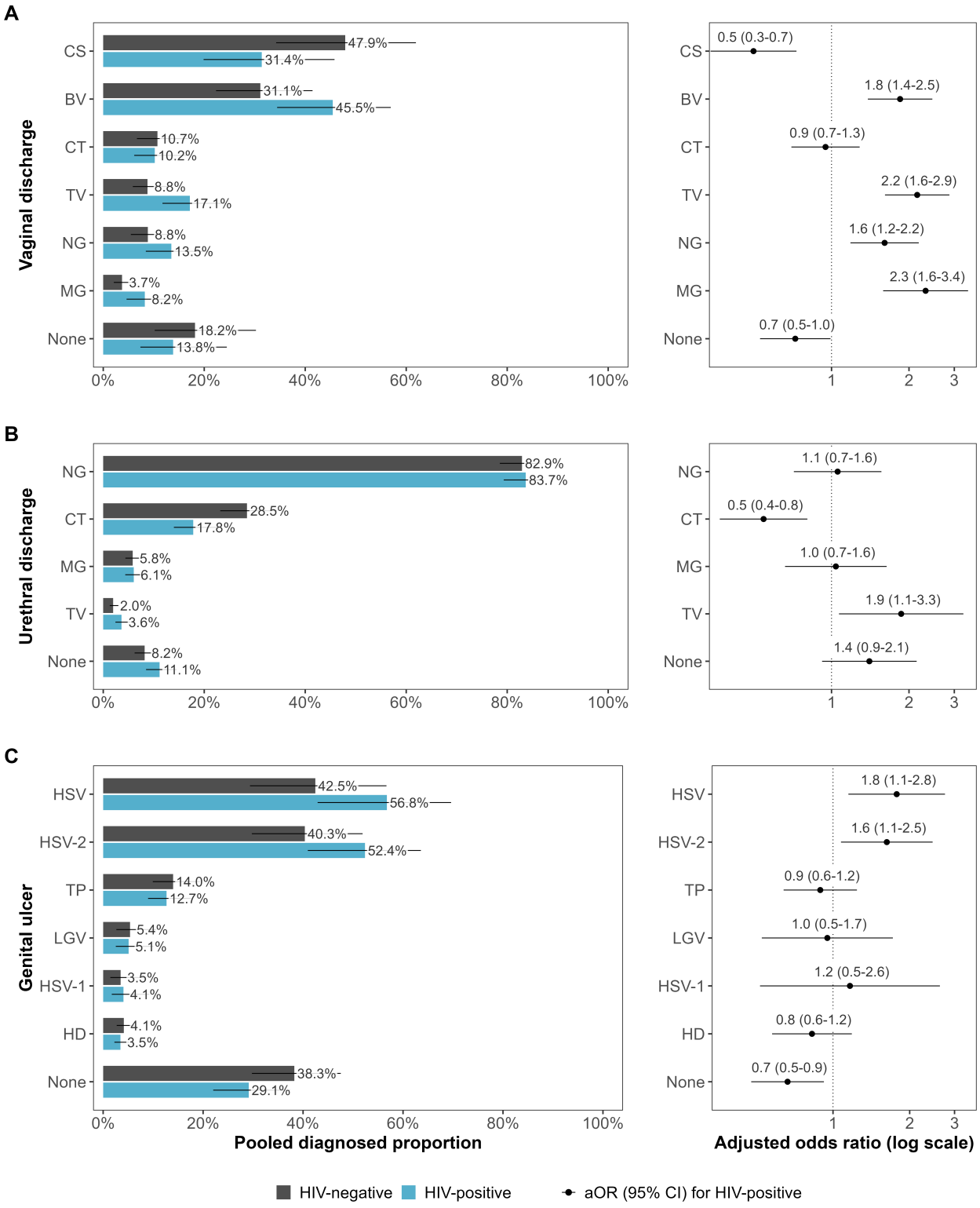


Figure 3.3: Comparison of diagnosed proportion per pathogen by HIV status in SSA

Estimated diagnosed proportion per pathogen among adults with and without HIV in 2015 (left) and adjusted odds of diagnosis per pathogen among adults with HIV compared to without HIV (right) symptomatic with (A) vaginal discharge, (B) urethral discharge, and (C) genital ulcer. Proportions and odds estimated via generalised linear mixed-effects model for each symptom. Bars represent sex-matched population-weighted mean proportions in 2015. Points represent the adjusted odds of diagnosis among adults with HIV. Solid lines represent 95% CIs.

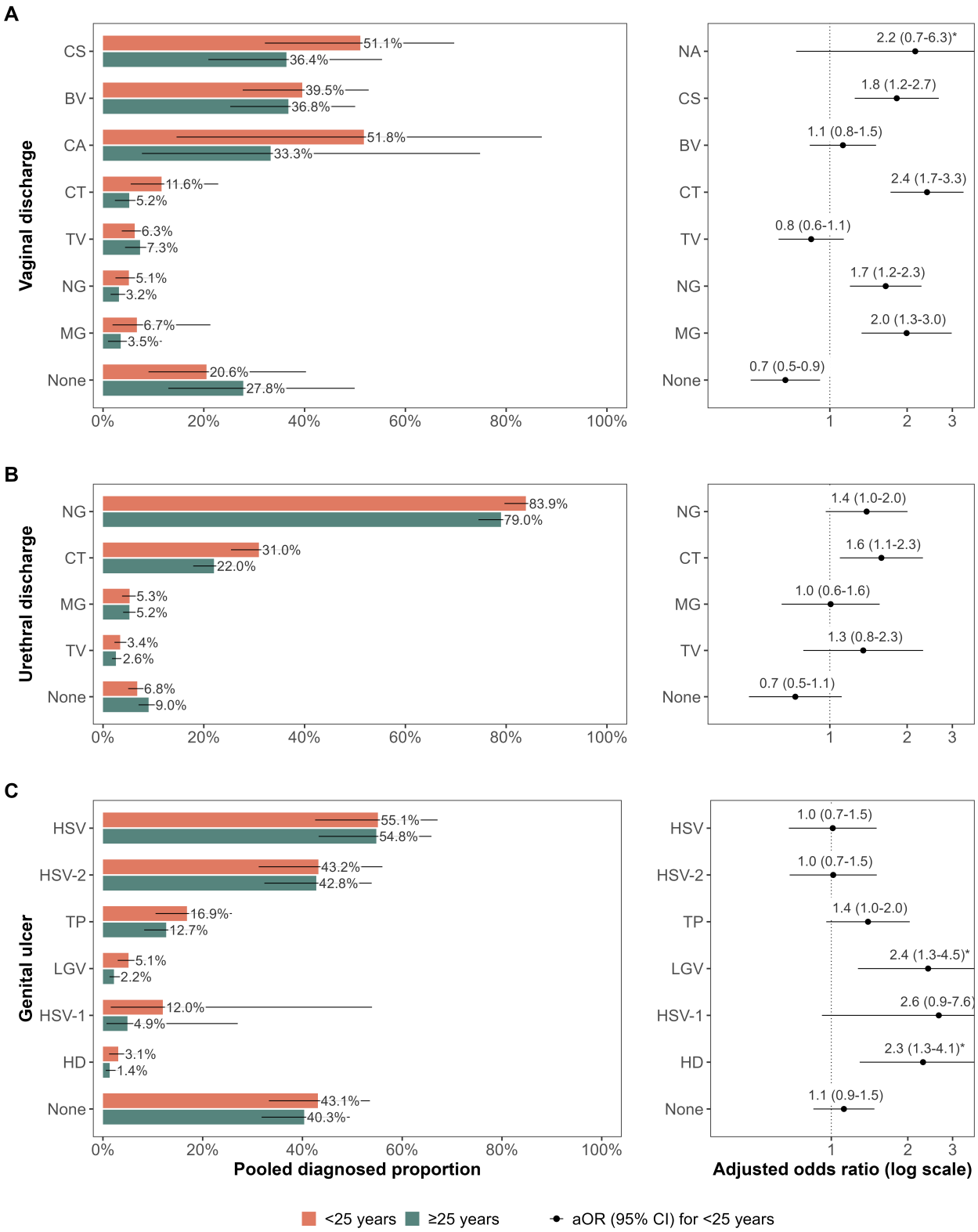


Figure 3.4: Comparison of diagnosed proportion per pathogen by age group in SSA

Estimated diagnosed proportion per pathogen among youth <25 years and adults ≥25 years in 2015 (left) and adjusted odds of diagnosis per pathogen among youth compared to adults (right) symptomatic with (A) vaginal discharge, (B) urethral discharge, and (C) genital ulcer. Proportions and odds estimated via generalised linear mixed-effects model for each symptom. Bars represent sex-matched population-weighted mean proportions in 2015. Points represent the adjusted odds of diagnosis among youth. Solid lines represent 95% CIs.

Table 3.2: Estimated diagnosed proportion per pathogen in SSA in 2015

RTI	Overall			HIV status						Age group					
				HIV positive			HIV negative			< 25 years			≥ 25 years		
	N_s	N_o	% (95% CI)	N_s	N_o	% (95% CI)	N_s	N_o	% (95% CI)	N_s	N_o	% (95% CI)	N_s	N_o	% (95% CI)
A. Vaginal discharge															
CS	31	50	69.4 (44.3-86.6)	4	17	31.4 (19.9-45.8)	4	17	47.9 (34.2-61.9)	5	18	51.1 (32.1-69.7)	6	19	36.4 (20.9-55.4)
BV	23	39	50.0 (32.3-67.8)	6	20	45.5 (34.4-56.9)	8	22	31.1 (22.3-41.4)	6	21	39.5 (27.7-52.7)	7	21	36.8 (25.2-50.0)
CA	9	9	31.5 (12.4-59.9)	0	0	-	0	0	-	2	2	51.8 (14.6-87.1)	2	2	33.3 (7.7-74.8)
CT	30	50	16.2 (8.6-28.5)	5	19	10.2 (6.1-16.5)	8	22	10.7 (6.7-16.8)	6	20	11.6 (5.5-22.9)	4	18	5.2 (2.4-11.1)
TV	55	80	9.2 (7.8-20.7)	8	22	17.1 (11.7-24.4)	8	23	8.8 (5.8-12.9)	11	25	6.3 (3.7-10.4)	11	25	7.3 (4.4-12.1)
NG	38	63	6.6 (3.8-12.7)	6	20	13.5 (8.4-20.8)	7	21	8.8 (5.5-13.9)	7	21	5.1 (2.5-10.3)	8	22	3.2 (1.5-6.5)
MG	5	20	5.4 (1.8-14.8)	3	17	8.2 (4.6-14.3)	5	19	3.7 (2.1-6.6)	1	15	6.7 (1.9-21.3)	2	16	3.5 (1.0-11.7)
None	13	28	25.1 (10.7-48.4)	3	17	13.8 (7.4-24.5)	5	19	18.2 (10.2-30.2)	4	18	20.6 (9.0-40.3)	4	18	27.8 (13.0-49.9)
B. Urethral discharge															
NG	47	68	77.1 (68.1-84.1)	5	19	83.7 (79.3-87.3)	4	19	82.9 (78.5-86.5)	5	19	83.9 (79.7-87.4)	3	17	79.0 (74.5-82.8)
CT	27	48	21.9 (15.4-30.3)	5	19	17.8 (14.0-22.4)	4	19	28.5 (23.2-34.5)	3	17	31.0 (25.4-37.1)	1	15	22.0 (17.9-26.8)
MG	8	29	5.5 (3.7-8.3)	2	16	6.1 (4.4-8.3)	2	17	5.8 (4.4-7.7)	1	15	5.3 (3.8-7.3)	1	15	5.2 (4.0-6.8)
TV	25	46	3.1 (1.8-4.3)	4	18	3.6 (2.4-5.4)	3	17	2.0 (1.3-3.0)	5	19	3.4 (2.3-5.1)	2	16	2.6 (1.8-3.7)
None	11	26	10.0 (6.8-14.5)	3	17	11.1 (8.5-14.5)	1	16	8.2 (6.2-10.8)	1	15	6.8 (5.0-9.2)	1	15	9.0 (7.1-11.5)
C. Genital ulcer															
HSV	28	55	57.1 (40.9-71.9)	11	29	56.8 (42.9-69.6)	8	24	42.5 (29.4-56.7)	1	12	55.1 (42.5-67.1)	1	15	54.8 (43.3-65.9)
HSV-2	22	47	48.3 (32.9-64.1)	14	29	52.4 (41.0-63.6)	13	28	40.3 (29.8-51.9)	3	11	43.2 (31.2-56.0)	4	17	42.8 (32.4-53.9)
TP	64	117	9.3 (6.4-13.4)	22	57	12.7 (9.0-17.6)	21	53	14.0 (9.9-19.4)	5	28	16.9 (10.6-25.8)	3	31	12.7 (8.3-19.0)
LGV	11	43	2.3 (1.0-5.3)	4	29	5.1 (2.5-10.1)	4	28	5.4 (2.6-10.6)	1	18	5.1 (3.0-8.7)	1	25	2.2 (1.3-3.6)
HSV-1	14	31	2.0 (0.8-4.7)	6	17	4.1 (1.2-13.2)	5	16	3.5 (1.4-8.3)	1	7	12.0 (1.8-50.8)	2	13	4.9 (0.2-57.0)
HD	56	107	2.0 (1.2-3.3)	23	56	3.5 (2.3-5.3)	23	54	4.1 (2.7-6.3)	3	24	3.1 (1.2-7.4)	3	31	1.4 (0.6-3.2)
None	38	83	34.7 (25.5-45.1)	15	48	29.1 (22.0-37.4)	14	44	38.3 (29.8-47.5)	2	22	43.1 (33.3-53.5)	3	31	40.3 (31.8-49.5)

Diagnosed proportion per pathogen among populations symptomatic with (A) vaginal discharge, (B) urethral discharge, and (C) genital ulcer. Proportions are sex-matched population-weighted means with 95% confidence intervals and were estimated using generalised linear mixed-effects model for each symptom in three sub-analyses: overall, stratified by HIV status, and stratified by age group. Multiple study observations possible when outcomes were stratified. N_s: number of unique studies, N_o: number of observations, BV: Bacterial vaginosis, CA: *Candida albicans*, CS: *Candida* species (any), CT: *Chlamydia trachomatis*, HD: *Haemophilus ducreyi*, HSV: Herpes simplex virus (unspecified), HSV-1: Herpes simplex virus type 1, HSV-2: Herpes simplex virus type 2, LGV: lymphogranuloma venereum, MG: *Mycoplasma genitalium*, NG: *Neisseria gonorrhoeae*, TP: *Treponema pallidum*, TV: *Trichomonas vaginalis*, None: unknown aetiology.

For urethral discharge, the odds of TV diagnosis were higher (aOR: 1.9 [95% CI: 1.1-3.3], n_{UD}=35) among men with HIV than men without HIV, and lower for CT (0.5 [0.4-0.8], n_{UD}=38; Figure 3.3B, Table B15). CT was the only RTI associated with age for urethral discharge, with higher odds among men <25 years (1.6 [1.2-2.3], n_{UD}=32; Figure 3.4B, Table B16).

For genital ulcer, the odds of unspecified HSV (aOR: 1.8 [95% CI: 1.1-2.8], n_{GU}=53) and HSV-2 (1.6 [1.1-2.5], n_{GU}=57) were higher among participants with HIV than those without HIV, but lower for those with unidentified aetiology (0.7 [0.5-0.9], n_{GU}=92; Figure 3.3C, Table B15). LGV (2.4 [1.3-4.5], n_{GU}=43) and HD (2.3 [1.3-4.1], n_{GU}=55) had higher odds of diagnosis among participants <25 years (Figure 3.4C, Table B16). In both analyses, men with genital ulcer had lower odds (0.4 [0.3-0.7]; 0.6 [0.4-1.0]) of diagnosis with HSV-2 than women (Table B15 and B16).

3.3.4 Sensitivity analyses and risk of bias

In sensitivity analyses, estimates derived for RTIs with large diagnostic test performance adjustments (CS, BV, TV, and NG) were higher than those based on reported observations, although trends were relatively similar (Figure B3, Table B17). Estimates derived using NAAT only were generally consistent with trend estimates based on all test types, irrespective of diagnostic test performance adjustment (Figure B4). Notable exceptions when restricted to NAAT-based estimates were for vaginal discharge, where TV was stable and NG increased over time, and for genital ulcer, where TP increased over time. However, NAAT-based estimates were interpolated from shorter time spans than estimates derived using all diagnostic tests.

Fewer than half of studies aimed to assess the aetiology of genital symptoms (N_{VD}=37/87, N_{UD}=32/55, N_{GU}=40/80, Table B18). Most studies were at risk of selection bias; participant inclusion criteria were often clearly defined (N_{VD}=82/87, N_{UD}=50/55, N_{GU}=73/80), but few studies recruited participants using consecutive or random sampling methods (N_{VD}=32/87, N_{UD}=27/55, N_{GU}=46/80), had adequate participation rates (N_{VD}=34/87, N_{UD}=21/55, N_{GU}=40/80), or provided sufficient detail on participant characteristics to determine their representativeness (N_{VD}=36/87, N_{UD}=21/55, N_{GU}=56/80). Most studies were not susceptible to measurement bias; the majority defined participant symptoms objectively (N_{VD}=48/87, N_{UD}=36/55, N_{GU}=75/80), employed consistent diagnostic methodologies (N_{VD}=81/87, N_{UD}=47/55, N_{GU}=75/80), and avoided misclassifying infections by testing for multiple pathogens (N_{VD}=43/87, N_{UD}=22/55, N_{GU}=48/80). Studies generally had sample sizes of at least 100 participants (N_{VD}=61/87, N_{UD}=39/55, N_{GU}=46/80) and reported results unambiguously (N_{VD}=51/87, N_{UD}=37/55,

N_{GU}=52/80). Several appraisal criteria were associated with the odds of RTI diagnosis, particularly for vaginal discharge, but the hierarchy of aetiologies per symptom remained the same (Figure B5).

3.4 Discussion

In this chapter, I estimated the distribution, trends, and determinants of aetiologies for STI-related symptoms in SSA from 1970 to 2022. I identified the primary aetiologies for vaginal discharge as candidiasis, bacterial vaginosis, and trichomoniasis, for urethral discharge as gonorrhoea and chlamydia, and for genital ulcer as herpes and syphilis. Additionally, I found that over 5% of vaginal discharge and urethral discharge cases were attributed to *M. genitalium*, and over a quarter of vaginal discharge and genital ulcer cases lacked an identified cause in 2015. Symptom aetiologies changed over time, particularly for genital ulcer where the leading cause shifted from chancroid in 1990 to HSV-2 by 2010. I found no evidence of systematic sub-regional variation in the aetiologic distributions. Although HIV status and age group were significantly associated with the diagnosis of several RTIs, the overall hierarchy of aetiologies for each symptom largely remained unchanged.

The estimated distribution of aetiologies for each symptom was consistent with infections prioritised by WHO syndromic management guidelines.[117] My analysis adds to previous reviews[125,126,132,134,138] that informed WHO syndromic algorithms by incorporating a wider range of studies reporting underlying syndrome aetiologies, specifically in SSA. However, I found no evidence of needing to adapt the algorithms for SSA settings or populations. Guidelines currently recommend assessing for *M. genitalium* only in instances of recurrent or persistent discharge,[117] which would potentially leave this infection untreated. Additional attention to MG is therefore warranted, particularly amid ongoing debates on aetiologic testing in higher-income settings.[310] The high proportion of cases without an identifiable cause poses a persistent unaddressed treatment challenge, even with expanded access to diagnostic testing.

Changes over time in genital ulcer aetiology are consistent with existing evidence indicating the near eradication of chancroid.[135] The reduction in chancroid cases is largely attributed to the introduction of antibiotics for genital ulcer management, illustrating that widespread empirical treatment can yield substantial public health benefits when pathogens are highly susceptible. As a typically symptomatic infection, chancroid responded well to syndromic management, but similar gains have been harder to achieve for STIs that are often asymptomatic or diagnostically complex. The prevalence of HSV-2 has also declined, only much more slowly.[5] Furthermore, the HIV epidemic has also shaped the

epidemiology of ulcerative infections over time, with documented synergy between HSV-2 and HIV.[311] I estimated that 2% and 48% of genital ulcer cases were attributed to HSV-1 and HSV-2 in 2015, respectively, consistent with other systematic review findings of 1.2% and 51% in SSA between 1990 and 2015.[5,312]

These temporal changes underscore the need for regular aetiologic assessment of syndromes. However, among 32 countries with data in this review, the publication rate approximated one study every ten years (median of 5 studies per country during 1969 and 2022), falling short of the WHO's recommended assessment frequency of 2 years.[313] In the absence of local studies, my findings suggest that data from neighbouring sub-regions or countries can be used to inform syndromic management protocols, although national-level monitoring of AMR for key pathogens may still be necessary.[146]

Aetiologic proportions among symptomatic populations are a component of characterising RTI burden, but the two are not equivalent. Diagnosed proportions reflect the prevalence of all possible aetiologies for a particular symptom, conditional on individuals being symptomatic and seeking care. For example, while HSV-2 prevalence has decreased in the region, I found that the proportion of genital ulcer cases attributed to HSV-2 increased over time because the prevalence of chancroid and syphilis declined more rapidly.[5,314–316] My estimates of decreasing aetiologic proportions for syphilis and chancroid were consistent with general population prevalence trends in SSA.[227,230,314–316] Diagnosed proportions also do not reflect the relative RTI burden among different population groups. Although I found that aetiologic proportions were distributed similarly by HIV status, age group, and sex (genital ulcer) for each symptom, the prevalence of RTIs in SSA is higher among women and people living with HIV, and varies by age.[317,318] Using these results to estimate overall STI burdens requires additional data on the prevalence of STI syndromes, care seeking, and aetiologies, collected from representative samples of the general population.

My analysis had limitations, mainly related to features of the studies and data. I extensively searched grey literature to identify all available data, particularly surveillance reports that might not appear in peer-reviewed academic literature. My grey literature search used similar criteria to the database search but was not strictly systematic. I did not find studies in 16 countries, particularly in central Africa (5/9 countries had no studies). However, the consistency of results across countries with data provides some confidence that key syndromic management recommendations are generalisable to countries that did not have any or recent studies. Most studies were not nationally representative and were conducted among a convenience sample of symptomatic individuals seeking healthcare at specific facilities. I

did not account for factors influencing treatment access, such as urban or rural location,[184,187,196] or symptom severity, recurrence, or treatment history, which could have influenced the aetiologic distributions among study participants, as these data were not consistently available. Additionally, as most studies did not report HIV prevalence or age among symptomatic participants, I was unable to adjust for these factors in the overall estimates. These factors may have contributed to heterogeneity in the study observations, which was particularly large for vaginal discharge. The definition of vaginal or urethral discharge based on abnormal symptoms may have captured a subset of those meeting criteria for diagnosis with vaginal discharge syndrome (abnormal discharge, vulval irritation, and/or itching) or urethral discharge syndrome (abnormal discharge, dysuria, and/or itching). Alternatively, the symptom-based definition may have included more individuals with non-infectious aetiologies than a clinical syndromic diagnosis. I chose the symptom-based definition to accommodate variations in reporting across studies and over time, but this may have influenced estimated aetiologic distributions. Several studies did not differentiate between HSV type or *Candida* species; I considered these data representative of HSV-2 and CA trends, which may have overestimated their contribution. I estimated the diagnosed proportion for HSV-2 at 85% that of unspecified HSV, which was consistent with previous studies.[5] The flatter trend observed for HSV-2 compared to unspecified HSV likely reflects the limited availability of typed observations in earlier years, rather than large changes over time in the share of genital herpes cases due to HSV-2. In contrast, I found that the diagnosed proportion for CA was 45% relative to CS, which was below the expected range of 70-90%.[319,320] My adjustments to the performance of gram stain and/or wet mount may have overestimated CS proportions, while CA may have been underestimated due to the limited number of observations. Other RTI proportions may also have been over- or under-estimated due to assigned diagnostic test sensitivity and specificity values, despite my efforts to ensure their accuracy. My modelling approach and estimates did not account for relationships between similar outcomes, such as the contribution of HSV-1 and HSV-2 to unspecified HSV; co-infection rates between RTIs; or the negative correlation between proportions for the same symptom, which depend on co-infection rates. Finally, variation in which pathogens were tested across studies may have influenced aetiologic attributions, particularly in cases of co-infection or where few pathogens were included. In such studies, undetected pathogens could have been misclassified or over-attributed to those that were tested, potentially overestimating the contribution of some individual RTIs. Due to the different numbers of pathogens examined across studies, I subjectively defined an unknown aetiology among study participants tested for three or more RTIs, irrespective of the pathogens examined or the number of potential pathogens present. This may have overestimated the

proportion with no identified aetiology.

In conclusion, I found that the aetiology of three common STI-related symptoms were remarkably similar across sub-regions in SSA but have evolved over time, underscoring a changing STI transmission landscape and the need for regular re-assessment to inform syndromic management protocols. The observed aetiologic distributions in SSA were largely consistent with WHO recommended syndromic management algorithms without strong evidence of variation by country, context, or population strata, strengthening the generalisability of my findings to settings lacking data in SSA.

Chapter 4

Prevalence and treatment of self-reported sexually transmitted infection symptoms in sub-Saharan Africa

4.1 Introduction

Despite the high burden of STIs in SSA, gaps in surveillance undermine effective programmatic responses and impede progress towards the WHO 2030 incidence reduction targets.[1] Aetiologic prevalence studies are infrequent, geographically sparse, and rarely population-representative, with few including men.[224] Syndromic case surveillance captures only symptomatic infections among individuals who seek care, primarily in the public sector, excluding those who seek care in private or informal settings or not at all.[224] Consequently, it remains challenging to identify geographies and populations with disproportionate disease burden or inadequate treatment.

Nationally representative household surveys, such as Demographic and Health Surveys (DHS) and AIDS Indicator Surveys (AIS; part of The DHS Program),[226] measure self-reported genital symptoms and associated care-seeking behaviours, and have potential to complement traditional surveillance by quantifying the relative burden and trends of STIs. These surveys are more consistently available and population representative than aetiologic prevalence studies, and capture individuals missed by facility-

based case surveillance. However, the symptoms typically measured — vaginal discharge, urethral discharge, and genital ulcer — are non-specific and may result from a range of infectious and non-infectious causes. The distribution of aetiologies for each symptom was quantified in Chapter 3, showing that vaginal discharge is most frequently caused by bacterial vaginosis and candidiasis, which are not sexually transmitted.[321] Moreover, while these aetiologic distributions were estimated at the regional level, data were insufficient to characterise variation across countries and sub-populations, despite likely differences in infection epidemiology.[321] Cultural norms, stigma, and health literacy may also affect reporting, complicating interpretation across countries and over time. Prior analyses of DHS/AIS have predominantly focused on socio-demographic or behavioural determinants of self-reported symptoms or treatment-seeking within specific countries[194,195,322–328] or sub-regions[184,186,329–331] of SSA, with limited attention to regional trends over time. More critically, it remains unclear whether symptom reporting patterns reflect the burden of their underlying sexually transmitted aetiologies.

In this chapter, I evaluate whether self-reported genital symptoms from population-representative surveys can inform estimates of STI prevalence levels and trends in SSA. Specifically, I estimate national and sub-regional trends in the prevalence of reported symptoms, treatment-seeking for these symptoms, and the distribution of healthcare sectors utilised for treatment between 2000 and 2024. I assess the extent to which symptom patterns align with underlying infection burden by evaluating correlations between the estimated prevalence of vaginal and urethral discharge and observed aetiologic prevalence of chlamydia, gonorrhoea, and trichomoniasis.

4.2 Methods

4.2.1 Data sources

The main data source for this analysis was DHS and AIS conducted in SSA between 2000 and 2024. These were cross-sectional nationally-representative surveys using two-stage stratified cluster sampling, in which all women aged 15-49 years in selected households were eligible to participate, and men aged 15-59 years were eligible from a subsample of approximately one-third of those households. The analysis included sexually active men and women aged 15–49 years who reported whether they had experienced genital discharge (vaginal discharge among females and urethral discharge among males) or genital ulcer in the past 12 months. Among those reporting symptoms, surveys also assessed whether respondents sought treatment and the types of facilities or providers from which they received it (specific questions in Table C1). I defined healthcare sectors utilised for treatment by aggregating reported treatment

providers into five categories: public, private, pharmacy or shop, traditional or other, and multiple sectors (Table C2). I defined variables according to the Guide to DHS Statistics.[226]

The second data source comprised aetiologic prevalence observations of chlamydia, gonorrhoea, and trichomoniasis identified through the systematic review in Chapter 2. These data were drawn from peer-reviewed studies conducted between 2000 and 2024 in SSA among populations broadly representative of the general populations. Most identified studies were not nationally representative. All prevalence observations were adjusted for diagnostic test sensitivity and specificity, as described in Chapter 2. I classified countries into sub-regions of Western Africa (WA), Central Africa (CA), Eastern Africa (EA), and Southern Africa (SA) according to the UN M49 standard (Table C3).[282]

4.2.2 Analysis

For each survey, I calculated point estimates and variances for the prevalence of genital discharge and genital ulcer among sexually active individuals, and the proportion treated among those reporting either symptom, accounting for the complex sampling design employed by DHS/AIS. Estimates were stratified by sex (male, female), five-year age group (15-19, 20-24, 25-29, 30-34, 35-39, 40-44, 45-49 years) and residence type (urban, rural). Using the same approach, I also calculated point estimates and variances for the proportion treated in each healthcare sector among those who reported treatment for either symptom, stratified by sex. I derived effective sample sizes for each point estimate using a binomial variance approximation, for incorporation into regression models.

To estimate sub-regional prevalence trends for genital discharge and genital ulcer, I fit generalised linear mixed-effects models for each symptom category. The models included fixed effects for sex, sub-region, an interaction term for sex and age, an interaction term for sex, sub-region, and year, and an interaction term for sex, sub-region, and residence. Country-level random intercepts, random slopes per year, and random effects per sex were included to account for country-specific variation over time and by sex. Random intercepts per survey were included to account for between-survey heterogeneity. To estimate sub-regional trends in the proportion treated for either symptom, I fit a generalised linear mixed-effects model using the same model structure, excluding the interaction term for sex, sub-region, and residence due to smaller sample sizes in the treatment-seeking sub-population. To estimate sub-regional trends in the proportion seeking treatment for their symptom in each healthcare sector, among those who sought treatment, I fit a generalised linear mixed-effects multinomial model using the multinomial-Poisson transformation.[332] Fixed effects included an interaction term for sector, sex, and sub-region, and an

interaction term for sector, sex, sub-region, and year. Random intercepts and sex-specific random effects were included for each country-sector combination. Random intercepts were also included per survey. All mixed-effects models were implemented with sum-to-zero constraints for country random effects within each sub-region, to ensure sub-regional estimates represented the average across countries. National means were weighted by sex-, age-, and year-matched national population estimates for adults 15-49 years from the UN World Population Prospects 2024.[287] Population weights for symptom models were stratified by urban and rural residence using year-matched national urbanisation proportions from the UN World Urbanisation Prospects 2018, assuming the same sex and age distribution for urban and rural populations.[333] The year 2020 was selected as the reference year for results reporting because the majority of surveys containing data for all three outcome types were conducted prior to this date (Figure C1). Results report the mean and 95% confidence intervals (CIs), estimated by simulating predicted probabilities from posterior model samples and taking the 2.5th and 97.5th percentiles.

To assess correlation between reported symptom prevalence in surveys and aetiologic STI prevalence in localised studies, I aggregated reported genital discharge prevalence (defined as vaginal discharge for females and urethral discharge for males) to match characteristics of aetiologic prevalence study observations. Estimates were matched by country, study midpoint year, sex (male, female), age group (younger adults, all adults), and residence (urban, rural, both or unknown). Age group definitions differed between data sources. Discharge estimates were stratified as 15-24 years for younger adults and ≥15 years for all adults, while aetiologic prevalence studies were classified as 12-25 years and ≥12 years, respectively, though underlying age distributions in aetiologic studies were generally unknown. Pearson's correlation coefficients were determined on the logit scale, stratified by sex, sub-region (Western and Central, Eastern, Southern), and diagnostic test method (NAAT, all tests).

Analyses were conducted in R version 4.4.1. Individual-level survey data were extracted using `rdhs` version 0.8.1.[334] Survey point estimates and variances were calculated using `survey` version 4.4.2.[335] Mixed-effects models used `R-INLA` version 24.12.11.[336] The Imperial College Research Ethics Committee approved analysis of secondary data for this study (ICREC 6365329).

4.3 Results

4.3.1 Data

Of 123 DHS and AIS conducted in SSA between 2000 and 2024, I included 113 surveys from 37 countries with questions assessing genital symptoms (Figure 4.1, Table C4). The analysis included 1,165,839 sexually active females and 417,706 sexually active males aged 15-49 years who reported genital symptoms in the past year. Among these surveys, 104 collected data on treatment-seeking among 123,379 females and 21,654 males who reported symptoms, and 81 collected data on treatment location among 66,934 females and 13,125 males who reported receiving treatment.

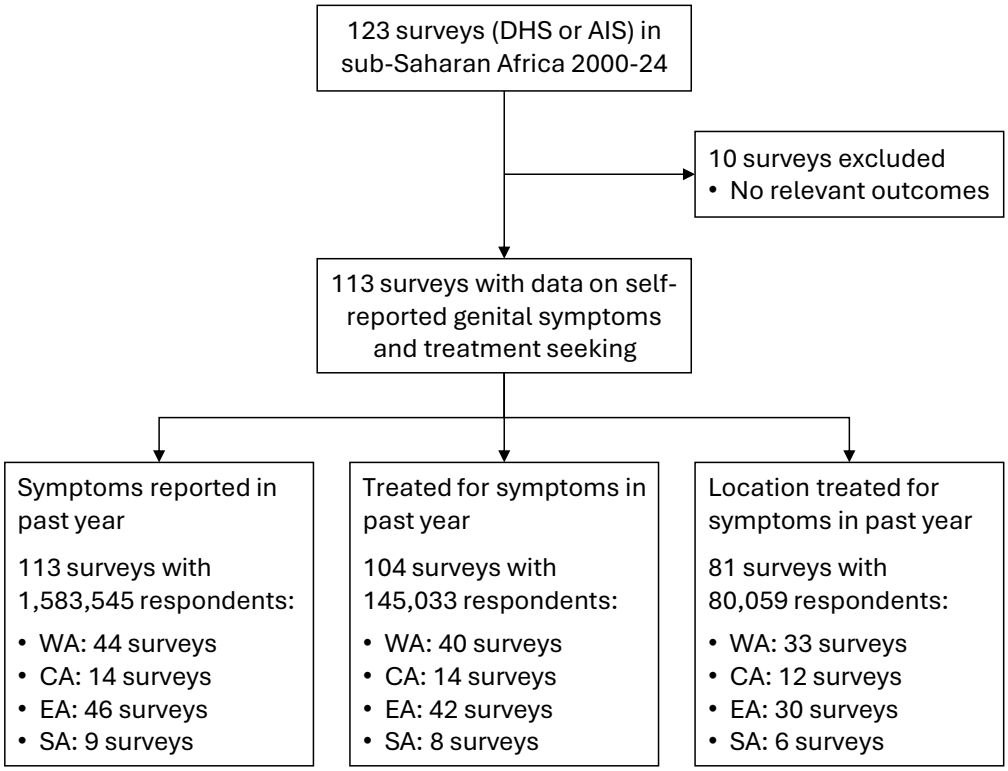


Figure 4.1: Flowchart of survey inclusion for each type of analysis

WA: Western Africa, CA: Central Africa, EA: Eastern Africa, SA: Southern Africa.

4.3.2 Genital symptom prevalence

In SSA, the estimated percentage of sexually active adults 15-49 years who reported symptoms in the past year in 2020 was 9.1% (95% CI: 8.0-10.4%) for vaginal discharge, 4.0% (3.4-4.6%) for urethral discharge, and 3.7% (3.2-4.3%) for genital ulcer (4.7% [4.1-5.5%] among females and 2.9% [2.5-3.4%] among males; Tables 4.1 and C5, Figure 4.2). Between 2000 and 2020, the reporting prevalence more

than doubled in SSA for vaginal discharge (aPR: 2.39 [1.83-3.14]), urethral discharge (2.22 [1.69-2.92]) and genital ulcer (2.25 [1.72-2.88]; Table 4.1). These increases were largely driven by Western and Eastern Africa, where reporting prevalence approximately tripled and doubled, respectively, for all three symptoms. There was no evidence of changes over time in Central or Southern Africa (Table 4.1, Figures C2 and C3).

Sub-regionally, prevalence of reporting vaginal discharge in 2020 was highest in Western Africa (13.8% [11.1-17.0%]), followed by Southern (11.2% [6.8-17.7%]), Central (9.6% [6.3-14.4%]), and Eastern Africa (5.8% [4.9-6.9%]; Table 4.1, Figure 4.2). Prevalence of reporting urethral discharge was relatively similar across sub-regions, ranging from 5.1% (2.9-8.6%) in Southern Africa to 3.3% (2.7-4.0%) in Eastern Africa in 2020. Prevalence patterns for genital ulcer were similar to those of genital discharge, though sub-regional differences were less pronounced. Among females, reporting genital ulcer was highest in Western Africa (6.4%, [4.9-8.3%]) and lowest in Eastern Africa (3.6% [2.9-4.4%]), while among males, it was more consistent across sub-regions. Urban-rural differences in symptom prevalence varied by sub-region (Table C7). In Central Africa, prevalence was higher in urban than rural areas for vaginal discharge (aPR: 1.47 [1.31-1.64]) and genital ulcer (1.32 [1.15-1.51]) in 2020, while in Southern Africa, prevalence was lower in urban areas for vaginal discharge (0.92 [0.84-1.00]), urethral discharge (0.76 [0.66-0.88]), and genital ulcer (0.66 [0.58-0.73]).

Several countries had a high prevalence of symptom reporting, particularly in Western Africa (Tables C9 and C10, Figures 4.2, C2, and C3). Vaginal discharge prevalence was high in Liberia (40.8% [31.4-50.3%]), Guinea (38.2% [28.4-48.0%]) and Mali (29.2% [21.0-38.6%]) in Western Africa, and in Uganda (21.9% [15.7-29.8%]) in Eastern Africa. Genital ulcer prevalence was also high in Liberia (19.5% [13.4-27.0%]) and Uganda (15.6% [10.2-21.5%]).

4.3.3 Comparison of discharge prevalence and aetiologic prevalence

Genital discharge reporting prevalence was not expected to match absolute prevalence estimates for chlamydia, gonorrhoea, or trichomoniasis, since these infections are often asymptomatic and genital discharge results from several additional aetiologies. However, alignment in relative patterns by sex, sub-region, and time would suggest symptom reporting reflects the underlying burden of these STIs.

Sex differences in discharge reporting were smaller than anticipated based on STI prevalence and symptom aetiology. Vaginal discharge is associated with a broader range of aetiologies than urethral discharge (Chapter 3). This most commonly includes non-STIs, as well as chlamydia and trichomoniasis,

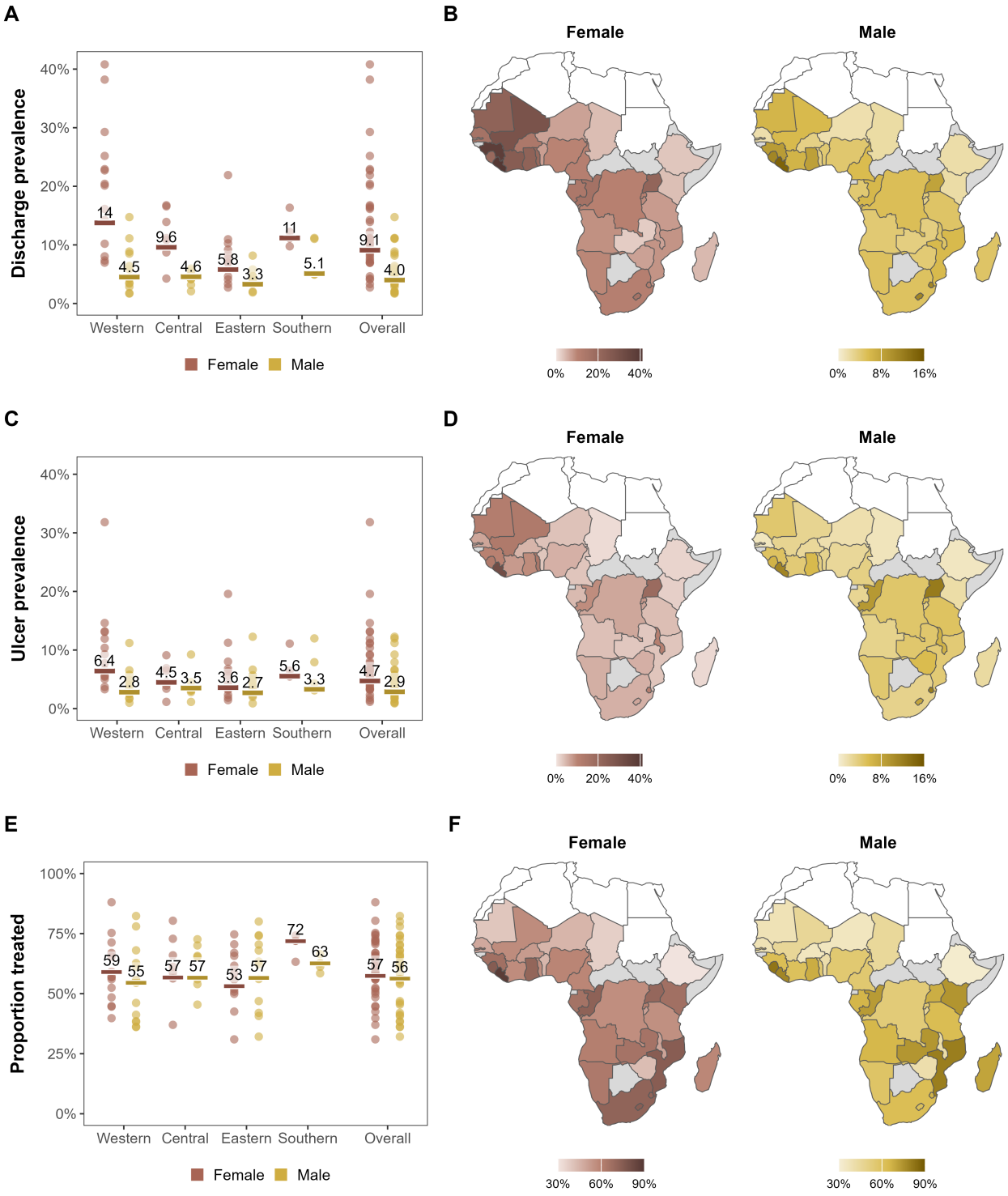


Figure 4.2: Prevalence and treatment of self-reported genital symptoms in SSA, 2020

Estimated prevalence of self-reported genital symptoms and the proportion of symptomatic individuals who reported seeking treatment, among ever sexually active adults aged 15–49 years in SSA in 2020. Panels A–B show prevalence of genital discharge, panels C–D show prevalence of genital ulcer, and panels E–F show proportion treated for either symptom. Left panels (A, C, E) present sub-regional estimates, where horizontal lines indicate population-weighted means matched by sex, age, and year (with values shown), and points represent national means. Right panels (B, D, F) present corresponding national estimates generated using generalised linear mixed-effects models, where colour intensity corresponds to percentages shown in the scales, and grey shading indicates data not available.

Table 4.1: Prevalence and treatment of self-reported genital symptoms in SSA

Sub-region	Estimate in 2020 % (95 % CI)			Relative change since 2000* aPR (95 % CI)		
	Female	**Male**	**Overall**	**Female**	**Male**	**Overall**
Discharge prevalence						
WA	13.8 (11.1-17.0)	4.5 (3.6-5.7)	8.0 (6.2-10.0)	3.39 (2.19-5.19)	2.86 (1.74-4.52)	3.13 (1.97-4.78)
CA	9.6 (6.3-14.4)	4.6 (2.8-7.3)	6.7 (4.3-10.0)	1.55 (0.59-3.44)	1.70 (0.61-3.90)	1.62 (0.59-3.66)
EA	5.8 (4.9-6.9)	3.3 (2.7-4.0)	4.4 (3.7-5.3)	2.17 (1.64-2.85)	1.93 (1.42-2.55)	2.04 (1.54-2.66)
SA	11.2 (6.8-17.7)	5.1 (2.9-8.6)	7.6 (4.4-12.3)	2.76 (0.71-8.04)	3.40 (0.79-9.79)	2.98 (0.73-8.69)
SSA	9.1 (8.0-10.4)	4.0 (3.4-4.6)	6.1 (5.3-7.0)	2.39 (1.83-3.14)	2.22 (1.69-2.92)	2.30 (1.75-3.03)
Ulcer prevalence						
WA	6.4 (4.9-8.3)	2.8 (2.1-3.7)	4.2 (3.3-5.3)	2.39 (1.56-3.49)	3.73 (2.33-5.52)	2.97 (1.91-4.33)
CA	4.5 (2.7-7.1)	3.5 (2.1-5.5)	4.0 (2.4-6.3)	1.35 (0.49-2.94)	1.57 (0.54-3.62)	1.45 (0.52-3.14)
EA	3.6 (2.9-4.4)	2.7 (2.2-3.3)	3.1 (2.5-3.8)	2.08 (1.50-2.87)	2.24 (1.60-3.09)	2.15 (1.57-2.96)
SA	5.6 (3.1-9.1)	3.3 (1.8-5.6)	4.3 (2.3-7.0)	2.59 (0.57-7.53)	3.19 (0.68-9.62)	2.81 (0.63-8.27)
SSA	4.7 (4.1-5.5)	2.9 (2.5-3.4)	3.7 (3.2-4.3)	1.99 (1.53-2.57)	2.56 (1.92-3.30)	2.25 (1.72-2.88)
Proportion treated						
WA	59.0 (52.7-65.5)	54.5 (47.5-61.5)	56.8 (50.6-62.9)	1.00 (0.82-1.22)	0.76 (0.63-0.91)	0.87 (0.72-1.03)
CA	56.7 (44.9-68.5)	56.6 (44.1-68.4)	56.1 (43.8-68.2)	0.91 (0.58-1.38)	0.69 (0.49-0.93)	0.77 (0.53-1.10)
EA	53.1 (46.5-58.9)	56.6 (49.6-62.9)	54.8 (48.5-61.4)	0.93 (0.77-1.11)	0.87 (0.73-1.03)	0.90 (0.75-1.06)
SA	71.9 (58.3-82.5)	62.6 (47.2-76.8)	68.2 (53.9-80.2)	1.09 (0.69-2.15)	0.92 (0.54-1.70)	1.00 (0.62-1.91)
SSA	57.4 (53.0-61.5)	56.3 (51.7-60.9)	56.8 (52.7-60.7)	0.94 (0.83-1.07)	0.78 (0.69-0.89)	0.85 (0.75-0.97)

*Ratio of prevalence in 2020 compared to prevalence in 2000. Ratios derived from model simulations. WA: Western Africa, CA: Central Africa, EA: Eastern Africa, SA: Southern Africa, SSA: Sub-Saharan Africa, CI: confidence interval.

which are substantially more prevalent among females than males (Chapter 2). Although males are more likely to develop symptoms from individual STIs, particularly gonorrhoea, the wider range of causes and higher overall STI prevalence among females suggest that discharge reporting should have exceeded the observed two-fold difference between females and males.

Geographic patterns in discharge reporting were not consistent with those of STI prevalence. Vaginal discharge was most frequently reported in Western Africa, whereas chlamydia and gonorrhoea prevalence were highest in Southern Africa and lowest in Western and Central Africa, and trichomoniasis prevalence was similar across sub-regions (Chapter 2). Urethral discharge reporting more closely reflected gonorrhoea prevalence patterns, with highest prevalence among males in Southern Africa.

Temporal trends in discharge reporting also diverged from those of STI prevalence. Reporting of both vaginal and urethral discharge more than doubled over the past two decades in SSA, while only chlamydia prevalence increased and gonorrhoea and trichomoniasis prevalence remained stable during the same period (Chapter 2). Given that chlamydia is predominantly asymptomatic, especially among females, it unlikely accounted for these increases in discharge reporting.

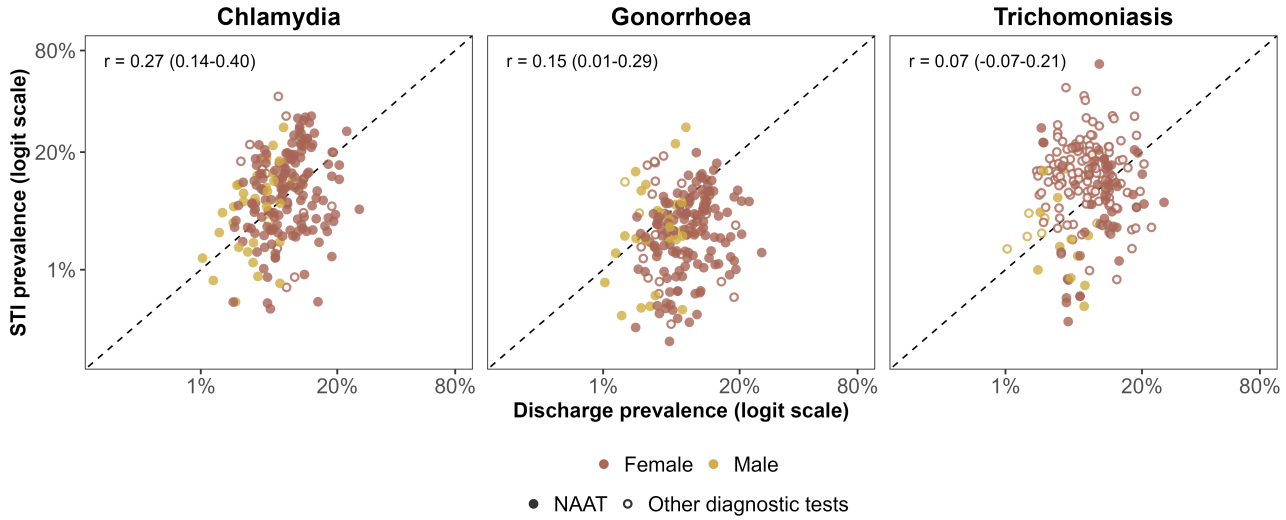


Figure 4.3: Correlation between reported discharge prevalence and aetiologic prevalence

Comparison of reported vaginal discharge and urethral discharge to aetiologic prevalence observations of chlamydia, gonorrhoea, and trichomoniasis, matched according to country, year, age group, and residence. r: Pearson's correlation coefficient, calculated for values on the logit scale. NAAT: nucleic-acid amplification test.

Table 4.2: Correlation between reported discharge prevalence and aetiologic prevalence

Group	Chlamydia		Gonorrhoea		Trichomoniasis	
	n	r (95% CI)	n	r (95% CI)	n	r (95% CI)
Vaginal discharge						
Sub-region						
Western and Central	19	0.50 (0.05-0.78)	15	0.60 (0.13-0.85)	12	0.38 (-0.25-0.78)
Eastern	82	0.12 (-0.10-0.33)	76	0.20 (-0.02-0.41)	16	-0.10 (-0.57-0.42)
Southern	46	0.47 (0.21-0.67)	45	0.41 (0.13-0.63)	24	-0.12 (-0.50-0.30)
Residence						
Urban	93	0.21 (0.01-0.40)	86	0.23 (0.02-0.42)	32	0.23 (-0.13-0.53)
Rural	43	0.26 (-0.04-0.52)	39	0.40 (0.10-0.64)	15	-0.02 (-0.53-0.50)
Non-stratified	11	0.36 (-0.31-0.79)	11	0.05 (-0.57-0.63)	5	-0.67 (-0.98-0.52)
Overall	147	0.24 (0.08-0.39)	136	0.28 (0.12-0.43)	52	0.15 (-0.12-0.41)
Urethral discharge						
Sub-region						
Western and Central	2	-	2	-	0	-
Eastern	15	0.26 (-0.29-0.68)	14	0.12 (-0.44-0.61)	2	-
Southern	14	0.58 (0.08-0.85)	12	0.10 (-0.50-0.64)	9	-0.46 (-0.86-0.29)
Residence						
Urban	16	0.40 (-0.12-0.75)	13	0.13 (-0.45-0.64)	5	-0.45 (-0.95-0.72)
Rural	11	0.38 (-0.29-0.80)	11	0.20 (-0.45-0.71)	4	-0.97 (-1.00–0.20)
Non-stratified	4	1.00 (0.96-1.00)	4	0.87 (-0.55-1.00)	2	-
Overall	31	0.44 (0.10-0.69)	28	0.30 (-0.08-0.61)	11	-0.53 (-0.86-0.10)

Aetiologic prevalence observations diagnosed using nucleic-amplification tests only. Table C8 provides correlation results for all diagnostic tests. n: number of observations, r: Pearson's correlation coefficient. CI: confidence interval.

Correlations between reported vaginal discharge prevalence and NAAT-diagnosed STI prevalence were weak for chlamydia (r = 0.24 [95% CI: 0.08-0.39], number of observations (n) = 147) and gonorrhoea (0.28 [0.12-0.43], n=136), and not significant for trichomoniasis (Table 4.2, Figure 4.3). Reported urethral discharge prevalence was moderately correlated with chlamydia (0.44 [0.10-0.69], n=31), but not significantly associated with gonorrhoea or trichomoniasis. Some sub-regions had stronger associations. Vaginal discharge correlated moderately with chlamydia and gonorrhoea in Southern (CT: 0.47 [0.21-0.67], NG: 0.41 [0.13-0.63]) and Western and Central Africa (CT: 0.50 [0.05-0.78], NG: 0.60 [0.13-0.85]), suggesting partial alignment of relative symptom reporting within these sub-regions (Table 4.2). Stratifying by urban or rural residence did not strengthen these associations (Table 4.2).

4.3.4 Treatment seeking

In SSA, among those reporting symptoms in the past year, an estimated 57.4% (53.0–61.5%) of females and 56.3% (51.7–60.9%) of males reported seeking treatment in 2020 (Tables 4.1 and C5, Figure 4.2). Treatment-seeking was highest in Southern Africa for both females (71.9% [58.3–82.5%]) and males (62.6% [47.2–76.8%]), and relatively similar across other sub-regions for each sex. Between 2000 and 2020, the proportion seeking treatment remained stable among females overall (aPR: 0.95 [0.83–1.07]) but declined by 22% (0.78 [0.69–0.89]) among males overall, driven by significant decreases in Western (0.76 [0.63–0.91]) and Central Africa (0.69 [0.49–0.93]; Figure C4).

The proportion who reported seeking treatment was particularly low in several countries (Table C11, Figures 4.2 and C4). In Western Africa, the lowest proportions were in Mauritania (both sexes combined: 39.3% [28.1-51.4%]), Niger (40.3% [23.6-59.0%]), Benin (43.4% [31.6-56.6%]), and Senegal (43.6% [34.8-53.4%]). In Central Africa, treatment-seeking was lowest in Chad (40.7% [24.2-58.4%]), and in Eastern Africa, in Ethiopia (31.2% [20.5-43.7%]). In contrast, the highest estimated proportions were in Liberia (83.6% [76.8-89.0%]), Sierra Leone (79.1% [70.5-85.7%]), Mozambique (77.4% [67.3-86.1%]), and Congo (72.5% [53.9-88.2%]).

4.3.5 Treatment sector utilisation

In SSA, among those who sought treatment for symptoms in 2020, 59.3% (57.5-61.0%) used the public sector, 14.7% (13.5-16.2%) used the private sector, 13.7% (12.5-14.9%) used pharmacies or shops, 8.8% (7.9-9.7%) used traditional or other sources of care, and 3.6% (3.2-4.1%) accessed multiple sectors (Tables 4.3 and C6, Figure 4.4). Public sector utilisation was highest in Southern (78.0% [72.0-83.2%]) and Eastern Africa (69.1% [66.2-71.8%]), and lowest in Western (46.9% [44.6-49.1%]) and Central

Africa (55.9% [50.9-60.9%]; Table 4.3, Figure 4.4). This pattern corresponded with higher utilisation of pharmacies or shops in Western Africa (24.8% [22.9-26.9%]), the private sector in Central Africa (23.0% [18.3-28.1%]), and traditional or other sources in both Western (12.3% [10.8-13.8%]) and Central Africa (12.3% [9.3-15.6%]).

Among those who sought treatment in SSA, females were more likely than males to use the public sector (aPR: 1.14 [1.08-1.21]), similarly likely to use private providers (0.93 [0.79-1.08]) or traditional or

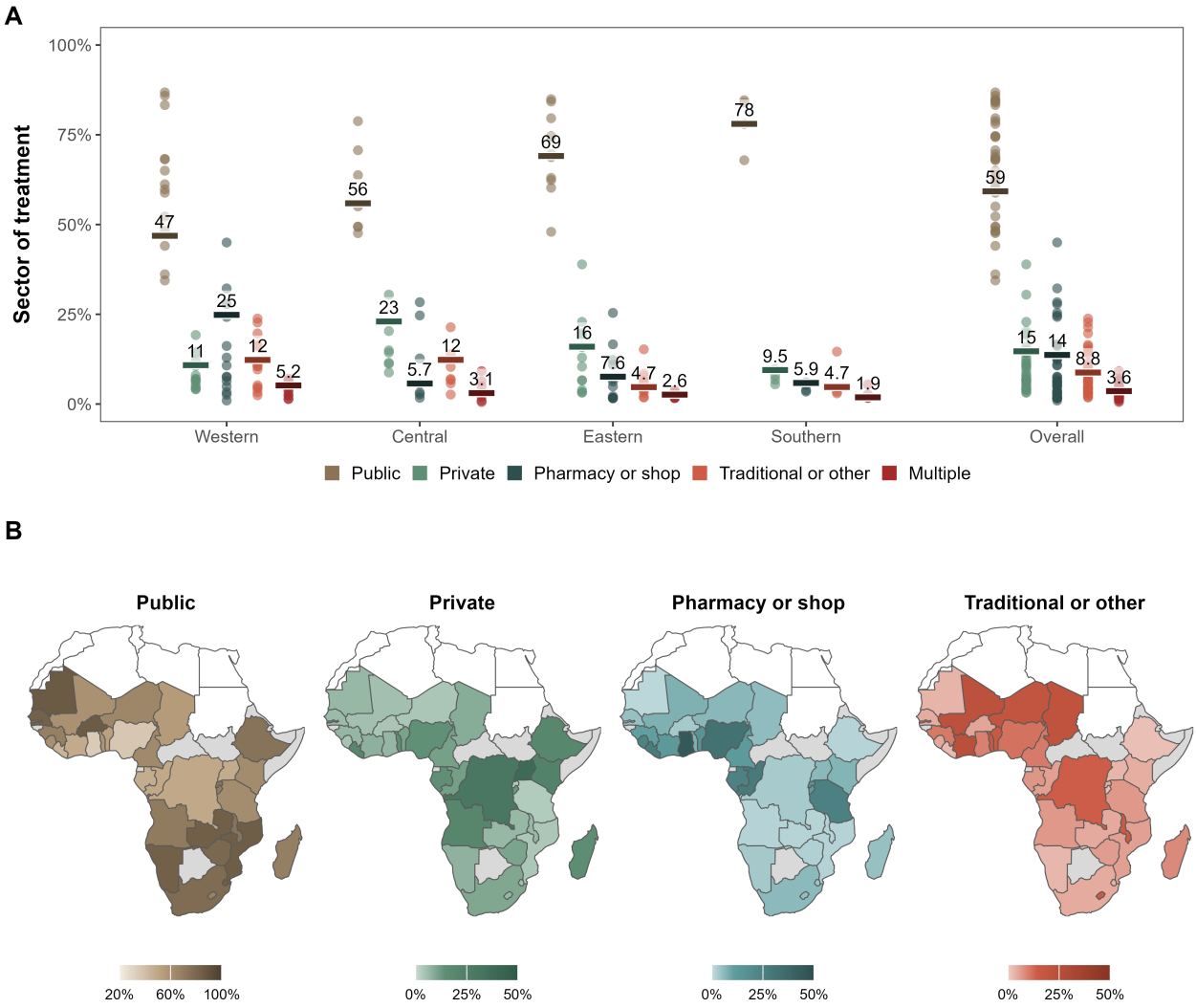


Figure 4.4: Distribution of healthcare sectors accessed for treatment in SSA, 2020

Estimated distribution of healthcare sectors accessed for treatment, among ever sexually active 15-49 year-old adults who reported treatment for genital symptoms in SSA in 2020. Panel A presents sub-regional estimates, where horizontal lines indicate population-weighted means matched by sex, age, and year (with values shown), and points represent national means. Panel B presents corresponding national estimates generated using a generalised mixed-effects multinomial model with the Poisson transformation, where colour intensity corresponds to percentages shown in the scales, and grey shading indicates data not available.

Table 4.3: Proportion treated for genital symptoms per health sector in SSA

Sub-region	Proportion in 2020 % (95% CI)			Relative ratio for sex†	Relative change since 2000*
	Female	Male	Overall	aPR (95% CI)	aPR (95% CI)
Public					
WA	51.7 (49.9-53.5)	41.9 (38.0-45.7)	46.9 (44.6-49.1)	1.24 (1.13-1.37)	1.04 (0.93-1.16)
CA	57.2 (52.0-62.3)	54.5 (46.6-62.4)	55.9 (50.9-60.9)	1.05 (0.89-1.24)	0.95 (0.77-1.16)
EA	73.9 (71.1-76.6)	64.4 (59.8-68.7)	69.1 (66.2-71.8)	1.15 (1.07-1.24)	1.09 (1.02-1.18)
SA	78.1 (70.6-83.9)	78.0 (68.2-86.3)	78.0 (72.0-83.2)	1.00 (0.87-1.17)	1.18 (0.91-1.63)
SSA	63.1 (61.4-64.7)	55.3 (52.4-58.1)	59.3 (57.5-61.0)	1.14 (1.08-1.21)	1.06 (0.99-1.13)
Private					
WA	9.6 (8.7-10.6)	12.0 (9.8-14.5)	10.8 (9.6-12.2)	0.81 (0.64-1.01)	0.57 (0.45-0.70)
CA	25.5 (20.2-30.9)	20.0 (13.4-28.5)	23.0 (18.3-28.1)	1.33 (0.85-1.97)	2.34 (1.24-4.07)
EA	14.3 (12.3-16.7)	17.6 (14.3-21.3)	16.0 (13.9-18.3)	0.82 (0.65-1.04)	1.17 (0.92-1.49)
SA	9.8 (6.7-14.3)	9.1 (4.0-17.3)	9.5 (6.2-13.8)	1.23 (0.52-2.64)	0.51 (0.19-1.19)
SSA	14.1 (12.8-15.6)	15.3 (13.3-17.6)	14.7 (13.5-16.2)	0.93 (0.79-1.08)	0.91 (0.75-1.09)
Pharmacy or shop					
WA	23.1 (21.6-24.9)	26.6 (22.7-30.2)	24.8 (22.9-26.9)	0.88 (0.75-1.03)	3.18 (2.51-3.88)
CA	3.7 (2.8-4.9)	8.0 (5.4-11.5)	5.7 (4.3-7.6)	0.48 (0.30-0.73)	2.05 (1.04-3.81)
EA	5.0 (3.6-6.9)	10.1 (7.1-14.0)	7.6 (5.8-10.0)	0.51 (0.33-0.77)	3.74 (2.10-6.30)
SA	9.1 (4.7-16.8)	2.2 (0.5-5.8)	5.9 (3.1-10.5)	5.89 (1.42-17.94)	3.06 (0.27-14.20)
SSA	11.9 (11.0-13.1)	15.5 (13.5-17.9)	13.7 (12.5-14.9)	0.77 (0.66-0.90)	2.95 (2.28-3.75)
Traditional or other					
WA	12.3 (11.0-13.5)	12.3 (9.8-14.8)	12.3 (10.8-13.8)	1.01 (0.80-1.27)	0.68 (0.52-0.87)
CA	11.3 (8.2-14.7)	13.5 (8.7-19.2)	12.3 (9.3-15.6)	0.87 (0.53-1.36)	1.02 (0.50-1.80)
EA	4.1 (3.3-5.1)	5.3 (4.0-7.0)	4.7 (3.9-5.7)	0.79 (0.56-1.09)	0.44 (0.30-0.61)
SA	0.9 (0.4-1.6)	9.1 (4.3-16.3)	4.7 (2.4-8.0)	0.11 (0.04-0.24)	1.90 (0.42-5.78)
SSA	8.1 (7.4-9.0)	9.4 (8.0-11.0)	8.8 (7.9-9.7)	0.87 (0.72-1.03)	0.66 (0.53-0.83)
Multiple					
WA	3.2 (2.8-3.8)	7.2 (5.5-9.2)	5.2 (4.3-6.2)	0.46 (0.34-0.60)	0.56 (0.38-0.79)
CA	2.3 (1.5-3.3)	3.9 (2.2-6.7)	3.1 (2.1-4.4)	0.62 (0.31-1.10)	0.24 (0.11-0.51)
EA	2.7 (2.0-3.5)	2.6 (1.8-3.6)	2.6 (2.0-3.3)	1.06 (0.66-1.63)	0.28 (0.17-0.45)
SA	2.1 (0.9-4.1)	1.6 (0.3-5.7)	1.9 (0.9-3.8)	2.32 (0.29-8.31)	1.28 (0.10-5.66)
SSA	2.8 (2.4-3.2)	4.5 (3.7-5.4)	3.6 (3.2-4.1)	0.63 (0.49-0.81)	0.38 (0.27-0.50)

†Ratio of proportion for females compared to males in 2020. *Ratio of overall (non sex-stratified) proportion in 2020 compared to 2000. Ratios derived from model simulations. WA: Western Africa, CA: Central Africa, EA: Eastern Africa, SA: Southern Africa, SSA: Sub-Saharan Africa.

other sources (0.87 [0.72-1.03]), and less likely to use pharmacies or shops (0.77 [0.66-0.90]) or multiple sectors (0.63 [0.49-0.81]; Table 4.3). Sub-regionally, pharmacy or shop utilisation was approximately 50% lower among females than males in Central (0.48 [0.30-0.73]) and Eastern Africa (0.51 [0.33-0.77]), while traditional sector utilisation was substantially lower among females in Southern Africa (aPR: 0.11 [0.04-0.25], Table 4.3).

Between 2000 and 2020, there was no evidence of change in public or private sector utilisation overall in SSA (Table 4.3, Figure C5). However, pharmacy or shop use nearly tripled (2.95 [2.28-3.75]), while

use of traditional or other providers declined by 34% (0.66 [0.53-0.83]). Sub-regionally, private sector utilisation more than doubled over time in Central Africa (2.34 [1.24-4.07]) but declined in Western Africa (0.57 [0.45-0.70]). Use of the traditional sector or other providers decreased in both Western (0.68 [0.52-0.87]) and Eastern Africa (0.44 [0.30-0.61]).

Several countries were outliers for their share of treatment sectors utilised (Tables C12 - C16, Figures 4.4 and C5). Public sector utilisation was higher in Mauritania (86.8% [82.4-90.5%]), Burkina Faso (85.9% [80.9-89.9%]), and Senegal (83.3% [80.0-86.3%]) than other Western African countries. Private sector use in Eastern Africa was high in Uganda (38.9% [35.5-42.4%]), while below 4% in Tanzania, Rwanda, and Mozambique. Pharmacy utilisation was highest in Ghana (45.0% [40.6-49.5%]) and Nigeria (32.2% [29.7-35.1%]) in Western Africa, Congo (28.4% [20.7-37.2%]) and Gabon (24.7% [20.5-29.9%]) in Central Africa, and Tanzania in Eastern Africa (25.4% [18.2-34.0%]), while almost negligible (<2%) in several countries such as Mauritania, Ethiopia, and Zambia. Traditional sector use was high in Cote d'Iviore (23.8% [19.6-28.4%]), Mali (22.7% [19.1-26.8%]), and Niger (19.7% [14.4-26.5%]) in Western Africa, and Chad in Central Africa (21.4% [15.0-29.0%]).

4.4 Discussion

In this chapter, I estimated trends in the prevalence of self-reported genital symptoms and treatment-seeking behaviours across SSA. The percentage who reported having STI symptoms in the past year more than doubled between 2000 and 2020, reaching 9.1% for vaginal discharge, 4.0% for urethral discharge, and 3.7% for genital ulcer in 2020. Among females, symptom reporting varied substantially by sub-region and was highest in Western Africa, while patterns among males were more uniform across the region. Less than 60% of symptomatic individuals reported seeking treatment, with higher rates in Southern Africa than other sub-regions. Among those who sought care, the distribution of healthcare sectors utilised varied widely between countries and sub-regions. The public sector remained the predominant source of treatment between 2000 and 2020, while use of pharmacies or shops nearly tripled (reaching 13.7% in 2020) and use of traditional providers declined (8.8% in 2020).

Geographic distributions and temporal trends in reported symptom prevalence were not consistent with underlying aetiologic patterns. Vaginal discharge, primarily caused by bacterial vaginosis (roughly 50% of cases; Chapter 3) and candidiasis (70%),[321] and genital ulcer, primarily caused by HSV-2 (50%) and syphilis (10%),[321] were most frequently reported among females in Western Africa, despite these aetiologies being least prevalent in that sub-region.[5,240,279,337] Geographic patterns of reported urethral

discharge aligned more closely with its primary aetiology, gonorrhoea (nearly 80% of cases; Chapter 3),[321] being most prevalent among males in Southern Africa and more uniformly distributed elsewhere in SSA.[338] Temporally, symptom reporting increased substantially in Western and Eastern Africa, despite evidence that prevalence has remained stable for bacterial vaginosis[321] and gonorrhoea,[338] and declined for trichomoniasis,[338] HSV-2[5] and syphilis[241,316] in SSA. Only candidiasis[321] and chlamydia[338] have evidence of increasing prevalence over time in SSA. These inconsistent patterns in geographic variation and temporal trends likely explain the weak correlations between discharge symptoms and laboratory-confirmed infections. The relatively stronger correlation for chlamydia may reflect closer alignment in temporal trends with reported discharge, whereas the negative correlation for trichomoniasis likely arises from opposing trends over time between the two data sources.

The high burden of reported vaginal discharge in Western Africa may partially reflect the sub-region's high prevalence of female genital mutilation (FGM). In several countries, including Guinea, Mali, and Sierra Leone, more than 90% of females aged 15-49 years have undergone FGM, with prevalence persistently high over the past three decades.[339,340] FGM is associated with chronic genitourinary sequelae, including vaginal discharge, itching, and pelvic inflammatory disease, as well as increased risk of bacterial vaginosis and other reproductive tract infections.[341,342] However, evidence on its association with HIV or other STI acquisition remains inconclusive.[342–344] Thus, to the extent that higher vaginal discharge burden in Western Africa is related to FGM, it likely reflects non-STI reproductive tract infections rather than higher STI prevalence, though this warrants further investigation.

Estimated increases in symptom reporting over time likely reflect sociocultural factors and changes in survey implementation, in addition to underlying epidemiological patterns. Over the past two decades, the expansion of HIV services and sexual and reproductive health education has likely improved STI knowledge and symptom awareness in SSA.[2] Evolving attitudes towards sexual and reproductive health and rights, though progressing unevenly across the region,[345] may have also influenced individuals' willingness to acknowledge or disclose genital symptoms. Additionally, methodological variations in survey implementation, question formulation, or translation may have affected response patterns over time or across sub-regions, although systematic changes have not been documented.

Persistently low and uneven treatment-seeking for genital symptoms undermines STI control efforts across SSA. Factors such as social stigma, limited health literacy, and socioeconomic barriers continue to restrict access to care.[199,346] Declines in treatment coverage among males in Western and Central Africa also highlight gender-specific disparities in sexual health service utilisation.[203] These challenges

contribute to delayed or missed diagnoses, which increases the risk of STI transmission and potential development of long-term reproductive health sequelae. Interventions are therefore needed to improve access to and uptake of appropriate treatment, particularly in countries with chronically low coverage.

Improving treatment coverage will require health system strategies that are tailored to the diverse and often fragmented sectors through which individuals seek care across the region. Patterns characterised for treatment sector utilisation reflect the structural differences in national health systems in SSA. Higher public sector utilisation in Southern (78.0%) and Eastern Africa (69.1%) likely reflects stronger public healthcare infrastructure and access to primary care services relative to other sub-regions,[347] though may also indicate limited availability of affordable alternatives. In contrast, greater reliance on pharmacies or shops in Western Africa (24.8%) and private providers in Central Africa (23.0%) suggests more fragmented health systems,[347,348] where patients often face high out-of-pocket costs to access care.[349] The growing reliance on pharmacy or shops over time emphasises both persistent structural gaps in the public health system[347] and patient preferences for care that is perceived as easily accessible and discreet.[192] These trends necessitate strengthening regulation of informal pharmacy providers, improving quality assurance of medicines and services across both formal and informal sectors, and better integrating pharmacies into national health systems.[350]

This study had several limitations related to the measurement and analysis of reported symptoms and treatment behaviours. All outcomes relied on self-reported data, which is subject to social desirability bias,[351] potentially leading to under-reporting of symptoms and over-reporting of treatment-seeking. Recall bias,[351] misinterpretation or non-recognition of symptoms,[117] and the exclusion of other relevant manifestations (e.g., dysuria, lower abdominal pain) may have further underestimated symptom prevalence. Several potential confounders were not accounted for due to data limitations. Non-sexually acquired genitourinary conditions, such as female genital schistosomiasis[352] or FGM-associated sequelae,[342] can produce similar symptoms and may have contributed to high reported symptom prevalence. I did not consider the geographic and temporal distributions of these conditions in my analysis. I also did not adjust for socioeconomic or cultural factors that affect symptom reporting and care-seeking,[184,199] and which likely varied between countries and over time. Analyses of treatment-seeking and sector utilisation were not stratified by urban-rural residence due to small sample sizes, limiting my ability to assess known geographic disparities.[199] HIV status was also not included, although people living with HIV are more likely to experience reproductive tract infections and have more frequent contact with the health system.[242] Differences in the quality and accessibility

of each treatment sector may have also shaped care-seeking behaviours, but were not considered.

Limitations in aetiologic prevalence data likely affected the assessment of correlations with self-reported symptoms. I considered only a subset of potential infectious aetiologies for vaginal and urethral discharge, which was particularly limiting for the former, as cervical infections are not the predominant cause and have low diagnostic sensitivity and specificity.[117] I also did not account for the large proportions of asymptomatic infection, particularly among females,[33] which would not have been captured through symptom-based reporting. Moreover, aetiologic prevalence data were geographically sparse and highly heterogenous, with limited observations among males, preventing robust assessment of correlations between reported urethral discharge and gonorrhoea or chlamydia.[117] Furthermore, I did not assess correlations between reported genital ulcer and underlying aetiologies such as HSV-2 and syphilis, since prevalence data for these infections were not readily available. This may have resulted in stronger associations, given the higher sensitivity and specificity of ulcer symptoms for identifying these aetiologies,[117] and could be explored in future analyses.

In conclusion, this analysis drew on 113 nationally representative surveys across 37 countries to estimate trends in the prevalence of self-reported genital symptoms and treatment-seeking in SSA. I identified substantial geographic and temporal discrepancies between reported genital discharge and underlying aetiologic prevalence, notwithstanding known limitations in both data sources. I also found uneven and often low treatment-seeking that occurred through a fragmented mix of healthcare providers, which varied considerably across the region. These findings highlight an urgent need to strengthen aetiologic surveillance, particularly in settings with exceptionally high symptom reporting that is inconsistent with expected infection patterns. Addressing structural barriers and improving access to effective treatment are essential for a comprehensive regional response aiming to improve STI control.

Chapter 5

Comparison of self-reported genital symptoms and syndromic case surveillance for estimating symptomatic sexually transmitted infection burden in Malawi

5.1 Introduction

Effective STI surveillance is essential to monitor epidemic trends, identify high burden populations and geographies, and guide programmatic responses.[1] In SSA, however, surveillance remains constrained by limited diagnostic testing capacity and infrequent aetiologic prevalence surveys.[222] The most accessible data are on experience of STI symptoms, captured through health facility syndromic case reports or household surveys recording self-reported symptom history. These sources, while potentially more consistently available and population representative than sporadic aetiologic prevalence surveys, have not been used to monitor STI trends due to several limitations. Most notably, both data sources fail to capture the substantial proportion of asymptomatic infections that contribute to ongoing transmission. Syndromic case surveillance is prone to misclassification due to the non-specific nature of symptoms, lacks standardised case definitions, and is often incomplete or reported in aggregate without age or sex

stratification.[224] Household surveys provide individual-level data, but may capture symptoms caused by non-infectious conditions and are susceptible to recall and reporting biases.[226]

In Chapter 4, I assessed the potential utility and limitations of self-reported genital symptoms and treatment-seeking from population-representative surveys across SSA. However, it remains unclear whether symptom trends from survey data align with those from facility-based syndromic surveillance. In this chapter, I explore whether self-reported symptoms can inform estimates of symptomatic STI burden and trends. I assess the consistency of determinants, trends, and spatial patterns from symptoms reported in household surveys and syndromic cases in health management information systems in Malawi, and compare the distribution of symptoms across both data sources.

5.2 Methods

5.2.1 Data sources

I compared two data sources to assess symptomatic STIs in Malawi. The first was the Malawi DHS, conducted in 2000, 2004, 2010 and 2015-16.[226] As described in Chapter 4, DHS are cross-sectional nationally representative household-based surveys. Women aged 15-49 years and men aged 15-54 years were eligible to participate if they were permanent residents or visitors in selected households. All men in one-third of the sampled households were selected for the male survey. Variable definitions were informed by the Guide to DHS Statistics (Table D1).[353]

I analysed self-reported genital ulcer, genital discharge, and known STI (defined in the DHS as infection acquired through sexual contact, other than HIV) in the last 12 months as outcomes, and among those, self-reported care or treatment sought for genital symptoms or known STI in the last 12 months. Among participants reporting care or treatment, I also assessed the sector (public, private, or other) where they sought care. I selected covariates based on factors associated with STI acquisition in previous literature.[329,354] Socio-demographic variables were age (15-19, 20-24, 25-29, 30-34, 35-39, 40+ years), sex (male, female), place of residence (urban, rural), marital status (never married, formerly married, married or living together), education (none, primary, secondary or higher), and current employment. Behavioural variables were age at first sex (<15, 15-19, 20-24, 25+ years), number of sex partners in the last 12 months (none, one, two or more), condom use at last sex in the last 12 months (yes, no or unknown), and ever tested for HIV (yes, no or unknown). Knowledge of STIs was measured as awareness that diseases can be transmitted through sexual contact (yes, no or unknown).

The second data source was health facility-level aggregate STI syndrome case counts recorded in the Malawi Department of HIV and AIDS Management Information System (DHAMIS), a database that records public sector supported HIV and related services in Malawi, provided through clinics, health posts, dispensaries, health centres, and hospitals, among others. Each quarter, monthly STI case reports are manually aggregated from paper STI clinic registers and verified.[355] Data were analysed from January 2014 to December 2021. During this period, reporting facilities increased gradually from 708 in 2014 to 794 in 2021. Aggregate STI case reports were stratified by sex (male, female), age (0-19, 20-24, 25+ years), and HIV status (HIV-positive, HIV-negative, unknown). STI diagnoses, which were not age-stratified, included genital ulcer disease (GUD), urethral discharge syndrome (UDS), low-risk vaginal discharge syndrome (VDS), high-risk VDS, lower abdominal pain, among others. As diagnoses could include multiple syndromes, the number of diagnoses was typically larger than the number of clients (Figure D1). I defined outcome variables for this analysis as cumulative annual diagnoses of GUD, UDS, and VDS (low- and high-risk combined) to align with DHS outcomes.

I used adjusted district-level population estimates by sex, age-group, and year for 2014-2021 as denominators for STI diagnosis rates. The Malawi National Statistics Office projected population totals by district, sex, and five-year age-group from the 2008 household census, accounting for anticipated population growth. These annual projections were adjusted proportionally in each district, sex, and age-group to align with national census results in 2018.[356,357]

5.2.2 Analysis

DHS respondent characteristics and self-reported symptoms and treatment-seeking were summarised as frequencies and survey-weighted proportions with design-based standard errors and 95% confidence intervals (95% CI). I assessed treatment by health sector using only the 2010 and 2015-16 DHS, due to inconsistent questionnaire formulation in earlier surveys.

To identify factors associated with self-reported symptoms, I fit four Bayesian logistic mixed-effects models to estimate the odds of self-reported genital ulcer and genital discharge in the last 12 months among ever sexually active males and females. To identify factors associated with self-reported treatment-seeking, I fit two Bayesian logistic mixed-effects models to estimate the odds of seeking treatment among males and females reporting a genital symptom or known STI in the last 12 months. All models included random intercepts and slopes over calendar year for the 33 geographic areas (29 health districts and four metropolitan cities). Survey cluster-level random effects were included to

account for the clustered sampling design.[353] Fixed effects included covariates for sociodemographic and behavioural characteristics, and STI-related knowledge. The models did not use sampling weights.

I checked health facility syndromic case reports in DHAMIS for consistency and completeness. I defined severely high outliers as monthly cases five times larger than the third quartile for the relevant health facility, year, and diagnosis. I assumed these outliers were reporting errors and replaced them with the median number of diagnoses per month for the corresponding health facility and year. I calculated annual rates of syndromic diagnoses per district and year by dividing the total number of diagnoses of each syndrome across all ages by the adult population aged 15-54 years.

To assess similarities in the distribution and characteristics of syndromic diagnoses in health facility case reports and symptom care seeking in the past year in household surveys, I estimated the probability of treatment for genital ulcer and genital discharge in the last 12 months among all adult DHS respondents. I fit Bayesian logistic mixed-effects models with random intercepts and slopes over calendar year for each geographic area and fixed effects for year and age interacted with sex. I used the models to predict the proportion of all adults aged 15-54 years seeking treatment for each symptom in the last year at district, region, and national level for the period 2014-2021, assuming a flat trend after the year 2015 (the year of the final survey). To compare the extent of district-level variability between data sources, I calculated coefficients of variation for each symptom based on average district rates during 2014-2021.

Regression model results were presented as adjusted odds ratios (aORs) with 95% credible intervals (95% CrI). All regression models used diffuse priors. Analyses were performed in R version 4.2.1, using the `rstanarm` package version 2.21.1.[358] The Imperial College Research Ethics Committee approved analysis of secondary data for this study (ICREC 6365329).

5.3 Results

5.3.1 Survey-reported symptoms and treatment

The four DHS included 60 039 women aged 15-49 years and 17 703 men aged 15-54 years who reported having ever had sex (Table D2). During 2010 and 2015-16, 11.0% (95% CI: 10.7-11.4%) of adults reported having a genital symptom or STI in the last year, of whom 54.2% (52.8-55.7%) sought treatment. More women than men reported genital ulcer (8.4% [8.1-8.8%] vs. 4.8% [4.4-5.2%]) and genital discharge (5.0% [4.7-5.3%] vs. 3.9% [3.5-4.3%]; Figure 5.1A). Among those reporting symptoms, women were slightly more likely to seek treatment than men (55.3% [53.8-56.9%] vs. 48.8% [44.9-52.7%];

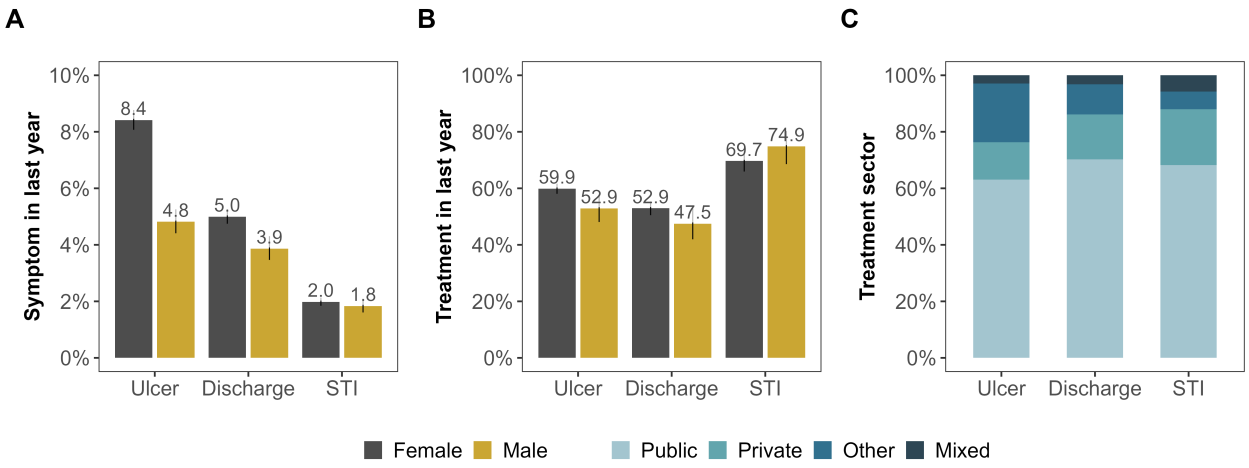


Figure 5.1: Self-reported symptoms and treatment-seeking in Malawi, 2000-2015

Percentage of ever sexually active survey respondents reporting (A) genital ulcer, genital discharge, or known STI in the last year (2000, 2004, 2010 and 2015-16); (B) treatment-seeking for genital ulcer, genital discharge, or known STI in the last year (2000, 2004, 2010 and 2015-16); and (C) health sectors accessed for treatment in the last year (2010 and 2015-16). Error lines represent 95% confidence intervals.

Figure 5.1B). Among those seeking treatment, 64.8% (62.3-67.3%) used the public sector, 15.0% (13.2-16.8%) the private sector, and 17.3% (15.4-19.3%) other services (Figure 5.1C). This distribution was similar across symptoms, although a higher proportion of adults with ulcer reported other treatment sources. More men than women (28.1% [22.4-33.7%] vs. 12.8% [11.0-14.6%]) used the private sector.

Factors associated with reporting a genital symptom varied by sex (Table 5.1). Among women, reporting ulcer and discharge was higher among those formerly married than never married (aOR: 1.45 [95% CrI: 1.23-1.73], 1.33 [1.09-1.62]), primary school educated than never educated (1.27 [1.16-1.40], 1.25 [1.10-1.41]) and employed than unemployed (1.13 [1.06-1.21], 1.18 [1.08-1.29]). Women aged 35-39 years were most likely to report genital ulcer (1.15 [1.03-1.28]), compared to 25-29 years. Among men, only primary school education (1.50 [1.09-2.12]) was associated with ulcer, and employment (1.38 [1.09-1.76]) was associated with discharge. Men (2.26 [1.63-3.15], 2.36 [1.66-3.33]) and women (2.73 [2.14-3.49], 2.93 [2.16-3.98]) with two or more sex partners in the last year were much more likely to report ulcer and discharge than those with no partners. As expected, the odds of reporting either symptom was higher among men and women who reported having an STI in the last year.

Spatially, symptom reporting was higher in Central and Southern Malawi (Figure D2). The odds of ulcer were almost four times higher among women in Central (aOR: 3.62 [2.67-4.82]) and Southern (3.63 [2.71-4.80]) than Northern region, and about two times higher among men (Central 1.98 [1.25-3.13], Southern 2.36 [1.52-3.71]). There was no evidence of linear time trend in symptom reporting among

Table 5.1: Adjusted odds of self-reported genital symptoms in the last year among sexually active adults in Malawi, estimated via generalised linear mixed-effects models

	Ulcer		Discharge	
	Female	**Male**	**Female**	**Male**
	aOR (95% CrI)	aOR (95% CrI)	aOR (95% CrI)	aOR (95% CrI)
Intercept	0.01 (0.01-0.02)	0.01 (0.00-0.02)	0.01 (0.01-0.02)	0.02 (0.01-0.04)
Year (centred at 2010)	1.00 (0.98-1.02)	1.02 (0.99-1.04)	1.01 (0.99-1.03)	1.01 (0.98-1.03)
Age group				
15-19	0.69 (0.60-0.79)	1.11 (0.80-1.55)	1.08 (0.93-1.26)	1.25 (0.86-1.84)
20-24	0.82 (0.74-0.90)	0.95 (0.72-1.23)	0.96 (0.87-1.08)	1.08 (0.80-1.48)
25-29	Ref	Ref	Ref	Ref
30-34	1.12 (1.01-1.23)	1.06 (0.82-1.35)	1.06 (0.93-1.20)	0.94 (0.67-1.31)
35-39	1.14 (1.03-1.27)	1.05 (0.79-1.39)	1.08 (0.94-1.24)	1.08 (0.76-1.51)
40+	0.88 (0.79-0.98)	0.93 (0.73-1.21)	0.90 (0.78-1.03)	0.66 (0.47-0.91)
Residence type				
Rural	Ref	Ref	Ref	Ref
Urban	1.14 (1.02-1.27)	1.24 (0.98-1.54)	1.06 (0.93-1.22)	1.07 (0.80-1.40)
Region				
Northern	Ref	Ref	Ref	Ref
Central	3.62 (2.67-4.82)	1.98 (1.25-3.13)	1.68 (1.28-2.20)	1.71 (1.06-2.80)
Southern	3.63 (2.71-4.80)	2.36 (1.52-3.71)	1.71 (1.31-2.24)	1.41 (0.88-2.28)
Marital status				
Never married	Ref	Ref	Ref	Ref
Formerly married	1.45 (1.23-1.73)	1.17 (0.79-1.70)	1.33 (1.09-1.62)	0.63 (0.38-1.03)
Married or living together	1.28 (1.08-1.51)	0.83 (0.62-1.13)	1.15 (0.96-1.39)	0.43 (0.31-0.60)
Highest education				
None	Ref	Ref	Ref	Ref
Primary	1.27 (1.16-1.40)	1.50 (1.09-2.12)	1.25 (1.10-1.41)	1.18 (0.82-1.72)
Secondary or higher	1.09 (0.96-1.25)	0.99 (0.69-1.44)	0.94 (0.79-1.11)	0.74 (0.50-1.13)
Employment				
No or unknown	Ref	Ref	Ref	Ref
Yes	1.13 (1.06-1.21)	1.09 (0.88-1.36)	1.18 (1.08-1.29)	1.38 (1.09-1.76)
Age at first sex				
<15	1.04 (0.96-1.13)	1.15 (0.96-1.39)	1.05 (0.95-1.17)	1.21 (0.97-1.51)
15-19	Ref	Ref	Ref	Ref
20-24	0.94 (0.83-1.05)	0.83 (0.67-1.02)	0.93 (0.80-1.08)	0.83 (0.63-1.09)
25+	0.75 (0.50-1.09)	0.69 (0.43-1.08)	0.94 (0.57-1.46)	1.00 (0.56-1.68)
Number of sex partners in past 12 months[1]				
None	Ref	Ref	Ref	Ref
1	1.20 (1.06-1.35)	1.15 (0.84-1.55)	1.48 (1.26-1.73)	1.29 (0.94-1.79)
2+	2.73 (2.14-3.49)	2.26 (1.63-3.15)	2.93 (2.16-3.98)	2.36 (1.66-3.33)
Condom use at last sex in past 12 months[1]				
No or unknown	Ref	Ref	Ref	Ref
Yes	1.38 (1.23-1.56)	0.87 (0.69-1.10)	1.21 (1.06-1.40)	0.81 (0.63-1.04)

Continued...

	Ulcer		Discharge	
	Female	**Male**	**Female**	**Male**
	aOR (95% CrI)	aOR (95% CrI)	aOR (95% CrI)	aOR (95% CrI)
Ever tested for HIV				
No or unknown	Ref	Ref	Ref	Ref
Yes	1.15 (1.04-1.27)	1.19 (1.00-1.42)	1.11 (0.98-1.25)	1.04 (0.84-1.29)
Knowledge of STIs				
No or unknown	Ref	Ref	Ref	Ref
Yes	0.98 (0.87-1.10)	1.28 (0.90-1.85)	0.71 (0.62-0.81)	0.83 (0.58-1.20)
Any STI in past 12 months[2]				
No or unknown	Ref	Ref	Ref	Ref
Yes	11.4 (10.0-13.0)	18.1 (14.2-23.1)	17.8 (15.3-20.5)	36.3 (27.2-48.4)
Random effects				
τ_{00} geographic area	0.28	0.37	0.29	0.42
τ_{00} cluster	0.38	0.21	0.32	0.57
τ_{11} geographic area	0.04	0.05	0.04	0.04
ρ_{01} geographic area	0.47	0.07	0.47	0.54

[1]Measured among those who reported sex in the past 12 months. [2]Measured among those who knew diseases can be transmitted through sexual contact. aOR: adjusted odds ratio, 95% CrI: 95% credible interval, Ref: Reference, τ_{00}: random intercept variance, τ_{11}: random slope variance, ρ_{01}: correlation between intercept and slope.

women (ulcer 1.00 [0.98-1.02], discharge 1.01 [0.99-1.03]) or men (ulcer 1.02 [0.99-1.04], discharge 1.01 [0.98-1.03]), and no consistent temporal trend across and within sub-regional areas (Figure D3).

Treatment seeking, among those reporting a genital symptom or known STI, was higher for urban than rural residents (aOR women: 1.43 [1.21-1.69], men: 1.69 [1.19-2.43]), and women with ulcers rather than discharge (1.70 [1.50-1.94]; Table D3). The odds of treatment decreased by 5% per year (0.95 [0.93-0.97]) for women and 4% (0.96 [0.92-1.00]) for men but did not vary substantially by region or district (Figures D4 and D5).

5.3.2 Facility-recorded syndromic case diagnoses

Between 2014 and 2021, 2 547 737 STI client case reports were recorded in DHAMIS (Table D4). Most cases (96.7%) were reported by facilities in the public sector. Of all cases, 59.4% were female, 67.7% were over 25 years of age, and 11.2% were asymptomatic partners. The majority (74.6%) of clients had not been treated previously for an STI. The Southern and Central regions recorded 51.9% and 34.8% of total cases, respectively, compared to 43.3% and 43.8% of the adult population in 2021. Of 2 657 076 syndromic diagnoses, the highest proportions were for VDS (30.2%), UDS (25.4%), GUD (14.0%), and lower abdominal pain (13.9%).

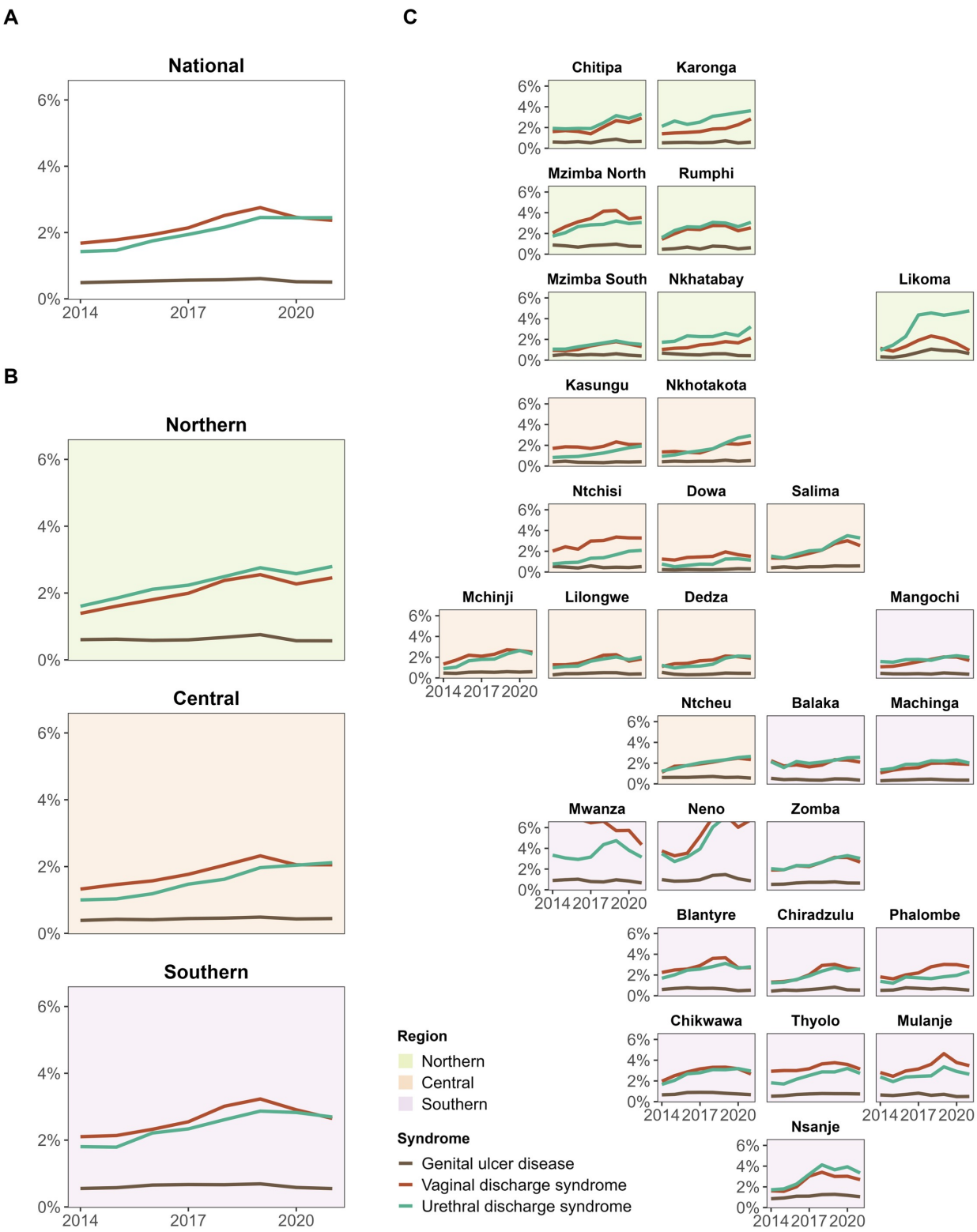


Figure 5.2: Syndromic case diagnosis trends by geographic level in Malawi, 2014-2021

Annual rate of syndromic case diagnoses among adults aged 15-54 years between 2014-2021 at (A) national, (B) regional, and (C) district level according to geographic proximity. Diagnosis rates were calculated using total reported syndromic diagnoses and sex-matched census population estimates per year per district. *Y-axes truncated at 6%; maximum rate of 9.7% (2014) for vaginal discharge syndrome in Mwanza, and 7.2% (2019) and 7.3% (2021) for vaginal discharge and urethral discharge syndromes in Neno.

Nationally, the annual case diagnosis rate among adults aged 15-54 years between 2014-2021 was 3.7% for all cases and 3.3% for symptomatic cases. The rate of VDS diagnoses among adult women increased from 1.7% in 2014 to 2.4% in 2021. The rate of UDS diagnoses increased from 1.4% to 2.5% over the same period. GUD diagnosis rates remained consistent, averaging 0.5% annually (Figure 5.2A). Southern and Northern Malawi had the highest case rates across all three diagnoses (Figure 5.2B). Mean case diagnosis rates were particularly high for VDS and UDS in Mwanza (6.7% and 3.6%) and Neno (5.4% and 5.2%) in the South and for UDS in Likoma (3.5%), a small island district in Lake Malawi (Figure 5.2C).

5.3.3 Comparison of survey and facility reporting

In household surveys, most adults reporting treatment for genital ulcer and genital discharge in the last year were female (67.5% [66.3-67.5%], 62.0% [60.8-63.6%]) and over 25 years (73.1% [72.7-73.4%], 68.2% [68.0-68.4%]). These were broadly consistent with the sex distribution (59.4% female) and age distribution (67.7% over 25 years) recorded in health facility case reporting.

In household surveys, nationally, 3.8% (95% CrI: 2.3-6.1%) of adults reported seeking treatment for genital ulcer in the last year, 3.8% (2.0-6.7%) for vaginal discharge, and 2.6% (1.2-4.7%) for urethral discharge. Although lower, the annual rate of health facility syndromic diagnoses among adults was within the credible intervals of the survey predictions for urethral discharge and vaginal discharge (Figure 5.3A). However, the average facility diagnosis rate for genital ulcer was much lower (0.5%) than treatment seeking proportions reported in the surveys.

Discrepancies between self-reported and facility-reported measures averaged during 2014-2021 were most striking in Central and Southern Malawi (Figure 5.3B). In these regions, self-reported treatment for genital ulcer was 9.6 times higher (4.2% [2.5-6.6%] vs. 0.4%) and 7.1 times higher (4.4% [2.7-6.7%] vs 0.6%) than facility-reported diagnosis rates. Vaginal discharge self-reporting was 1.7 (3.2% [1.6-5.7%] vs. 1.9%) and 1.9 (5.0% [2.7-8.4%] vs. 2.6%) times higher and urethral discharge self-reporting was 1.3 (2.1% [1.0-4.0%] vs. 1.6%) and 1.4 (3.4% [1.7-6.0%] vs. 2.4%) times higher than facility case diagnosis rates in the Central and Southern regions.

Coefficients of variation, averaged across all symptoms, indicated higher district-level variation for survey self-reported treatment (74.4%) than facility case diagnosis rates (38.6%). District-level variation was highest in the South, in both survey self-reporting (South: 64.5% vs. Central: 53.1% and Northern: 56.8%) and facility case-reporting (38.5% vs. 23.5% and 25.3%).

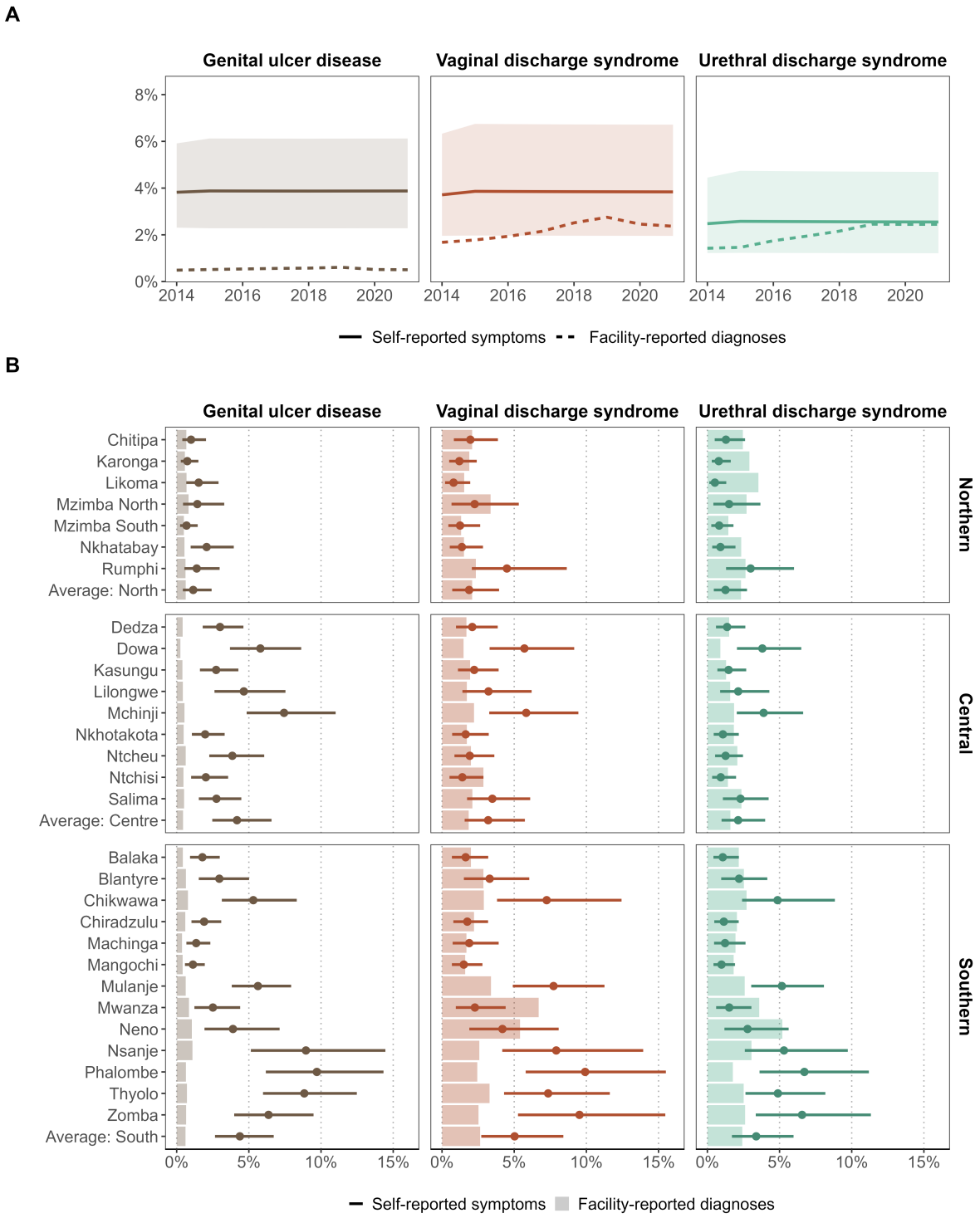


Figure 5.3: Comparison of self-reported treated symptoms and facility-reported syndromic case diagnoses in Malawi, 2014-2021

Comparison of the prevalence of self-reported symptoms with treatment in the last 12 months and annual facility-reported syndromic diagnosis rates, among adults aged 15-54 years in Malawi between 2014-2021. (A) National comparison of annual rates per symptom. Dotted lines represent aggregate health facility case reports. Solid lines represent self-reported symptoms from household surveys between 2000 and 2015-16, predicted to 2014-2021. Shading represents 95% credible intervals around self-reported symptoms. (B) District and regional comparison of mean rates per symptom. Solid bars represent aggregate health facility case reports. Points and error bars represent self-reported symptoms with 95% credible intervals.

5.4 Discussion

In this chapter, I assessed the consistency between symptoms reported in nationally representative household surveys and syndromic case diagnoses recorded by health facilities in Malawi. The two data sources indicated divergent rates, distributions, and temporal trends in STI symptoms. In household surveys between 2000 and 2015-16, around 11% reported having a genital symptom or known STI in the last year, of whom around half sought treatment for their last infection, implying that 6.0% of adults per year present for STI care. In health facility case reports between 2014 and 2021, the mean annual symptomatic case diagnosis rate was only 3.3%.

Symptom distribution also differed between data sources. In surveys, genital ulcer and vaginal discharge were most common among adults seeking treatment. In national case reports, facility diagnosis rates were highest for vaginal discharge and urethral discharge syndromes, and substantially lower for genital ulcer. This diagnostic distribution aligns with previous research among STI clients attending an urban and rural hospital in Malawi.[359,360] Furthermore, surveys indicated a higher proportion of symptomatic women treated for genital ulcer than men. The absence of sex-stratified case report diagnoses precluded direct comparison, but prior research identified the opposite sex trend among STI clients.[359,360] It is unlikely that the 90% lower GUD rate recorded in health facility data from 2014-2021, compared to survey data from 2000-2015, is explained by the decreasing prevalence of ulcerative STIs (HSV-2, syphilis, and chancroid) over time in SSA.[5,316,361]

STI care seeking outside predominantly public sector facilities reporting to the national health information system may partly account for differences in the magnitude and distribution of symptoms across data sources. In 2021, DHAMIS case-reporting included 794 health facilities. The 2018 master facility list documented 963 public, private, and traditional STI service providers in Malawi, but this likely underestimated the count due to difficulties capturing unregistered or informal sources of care.[188] Most survey participants reported seeking care in the public health sector, but over 30% sought treatment from the private and traditional sectors. Men reported using the private sector more than women. This was consistent with previous research that men in SSA prefer private facilities for perceived increased quality of care and privacy, despite higher costs.[187,188,362] Individuals with genital ulcer reported using other means of treatment, such as traditional healers, more often than those with other symptoms. Although the traditional health sector is widely used in Malawi,[363] differences in traditional care-seeking by STI symptom have not been previously documented. Moreover, frequent

antibiotic stockouts in the public sector may result in periods of increased reliance on private and traditional providers.[364] However, I did not evaluate these patterns.

Temporal trends differed between the two data sources, though my assessment was limited by not having survey data after 2015 for direct comparison with health facility data. While facility case diagnosis rates increased between 2015 and 2020, household surveys showed no significant change in STI symptom reporting and a decline in STI care seeking between 2000 and 2015. The trends could have reversed, particularly trends in care seeking coinciding with health system improvements supported through HIV service decentralisation. One possible explanation for the rise in syndromic diagnoses is the large scale-up of HIV testing in Malawi since 2015,[365] which may have facilitated STI detection and management, underscoring the importance of integrated health service provision.[1] The upcoming Malawi DHS will help assess if increasing facility diagnoses are reflected in self-reporting, and if so whether they are attributable to higher rates of symptomatic infection or treatment seeking.

Despite discrepancies in overall levels, both data sources had similar demographic and spatial patterns, consistent with the burden of STIs. I found a higher burden of symptoms among women, individuals above 25 years, and in Southern Malawi. Specifically, self-reported genital ulcer in surveys was highest among women 35-39 years old and in the Southern region, aligning with the prevalence of HSV-2 and HIV. HSV-2, the main cause of genital ulcer in SSA,[5] is more prevalent among women within this older age group due to the lifelong nature of infection.[275] HIV prevalence in Malawi is also higher among women aged 35-54 years and in the South.[366] These complementary trends suggest household surveys can provide insight into the relative prevalence of symptomatic STIs. However, future analyses aiming to reconcile the burden and distribution of STI symptoms with STIs should incorporate factors influencing the aetiology of these symptoms. For instance, a recent study among STI clinic attendees with genital ulcer in Malawi identified a substantial proportion of cases attributed to syphilis, indicative of shifts in genital ulcer aetiology over time, and a considerably higher proportion of HSV-attributed cases among those HIV-positive than HIV-negative.[367]

District level differences between survey self-reporting and facility case-reporting require further investigation to inform programme planning and prioritisation. Districts with high self-reported treatment but low facility diagnoses might rely more on the private and traditional sectors for STI service provision. Programmes should examine this further and consider expanding public sector services in these areas to address potential unmet treatment needs. Moreover, I found greater district-level variation for self-reported treatment than facility case diagnoses. Surveys may thus better identify

districts with a higher infectious burden for prioritised programming.

Limitations in the two data sources may contribute to the identified inconsistencies. Self-reporting in household surveys may under-represent symptomatic infection, due to recall and social desirability biases,[351] and potential lack of awareness of STI symptoms.[117,368] Over-reporting may have resulted from using non-specific questions on STI symptoms. Analysis of facility-reported routine cases may have overestimated syndromic diagnosis rates among adults, due to limited stratification available in DHAMIS. Case diagnoses were included from all age groups, yet 8.8% of total diagnoses were among those younger than 19 years. Additionally, clients may have been double counted if they sought care from multiple health facilities or experienced recurrent symptoms in a year.

In conclusion, in Malawi, where aetiologic STI surveillance is not widely implemented, I found non-negligible discrepancies in the magnitude, trends, and distribution of STI symptoms between household survey self-reporting and health facility syndromic case reporting. Spatio-demographic trends were however similar across data sources, suggesting that survey self-reporting could potentially aid programme prioritisation and investment in the absence of aetiologic prevalence data and reliable case-reporting data. My analysis did not account for asymptomatic infections, which constitute a high proportion of the overall infectious burden. Localised STI sentinel surveys could help adjudicate some of the inconsistencies identified, such as the different relative burden of STI symptoms, to facilitate interpretation of both data sources for a more comprehensive understanding of STI epidemiology and surveillance.

Chapter 6

Optimal deployment of point-of-care tests for gonorrhoea in Kenya

6.1 Introduction

Gonorrhoea poses a substantial public health challenge in SSA.[227] The infection can cause severe reproductive tract complications, including pelvic inflammatory disease and infertility.[369] In pregnant women, untreated infections can cause preterm labour, low birth weight, and perinatal mortality.[71] The emergence of drug-resistant strains has led WHO to classify *N. gonorrhoeae* as a high-priority pathogen requiring urgent action.[370]

Syndromic management is the primary approach for treating symptomatic STIs in SSA, but its poor diagnostic performance for cervical infections like gonorrhoea often results in inadequate care.[117] WHO guidelines now recommend incorporating molecular diagnostics into syndromic algorithms for managing vaginal and urethral discharge syndromes, but implementation in many global settings is impeded by requirements for laboratory infrastructure and trained personnel, along with high per-test costs.[116,117] High rates of asymptomatic gonorrhoea have prompted several high-income countries to implement screening among higher-risk and pregnant populations, but such screening programmes have not been implemented at scale in SSA.[156,157]

Point-of-care tests (POCTs) offer an opportunity to expand access to diagnostic testing and screening. Although existing gonorrhoea POCTs or near-POCTs do not meet WHO TPP requirements for diagnostic performance, usability, and cost, several promising new diagnostic platforms are in

development.[140,176] Maximising public health impact with a constrained initial supply of and limited resources for next-generation POCTs will require strategic targeting during early implementation.

Several trade-offs need to be considered for test allocation.[179] First, while targeting individuals with symptoms of urethral or vaginal discharge would yield higher positivity rates per test, screening for asymptomatic infections may identify cases that would otherwise go untreated, as symptomatic individuals are more likely to seek treatment through existing channels. A second consideration is how to prioritise among groups of men or women with different characteristics. Testing men could achieve greater reductions in onward transmission due to higher male-to-female transmission probability.[7] Diagnostic testing among symptomatic men may also have a higher yield because gonorrhoea causes a higher proportion of urethral than vaginal discharge cases,[321] and men more likely to seek treatment when symptomatic.[188,191] However, targeting women could directly prevent severe sequelae and, despite the potentially lower diagnostic yield, offer greater incremental benefit over syndromic management. Third, prioritising high-risk populations may most effectively reduce transmission at population-level,[371] but once test availability increases, coverage in these groups could saturate and miss substantial infection burden in the general population, also necessitating expansion to lower-risk groups.

In this chapter, I use mathematical modelling to assess how a limited supply of gonorrhoea POCTs could be allocated across different testing strategies and target populations to maximise reductions in population-level incidence and morbidity in Kenya between 2025 and 2030. I develop a compartmental transmission model to quantify the burden of infection and associated sequelae across five target populations: FSW, clients of female sex workers (CFSW), pregnant women, sexually active AGYW, and men in the general population. My analysis compares the impact of testing symptomatic individuals and screening routine healthcare service attendees (i.e. both symptomatic and asymptomatic) to inform prioritisation both within and between target populations.

6.2 Methods

6.2.1 Mathematical model structure

I developed a deterministic compartmental model of heterosexual gonorrhoea transmission among individuals aged 15–49 years in Kenya. The model represented an open and growing population, stratified by sex (male, female), sexual risk (low, intermediate, high), age (15–24 years [younger], 25–49 years [older]), and gestational status (non-pregnant, pregnant), denoted as $s \in \{m, f\}$, $r \in \{l, i, h\}$,

$a \in \{y, o\}$, and $g \in \{n, p\}$, respectively. High-risk strata represented FSW and CFSW, and risk classifications were fixed over time for all strata. Individuals progressed through age groups, and females transitioned between gestational states based on age- and risk-specific fertility rates. New individuals entered the model as aged 15–24 years and not yet sexually active (U), and transitioned unidirectionally to the sexually active population either upon sexual debut or ageing into the 25–49-year-old group. Individuals exited the model either upon reaching age 50 or due to all-cause mortality.

Sexual partnerships were formed between sexually active individuals through proportionate mixing across risk, age, and gestational strata. FSW formed partnerships exclusively with CFSW, while CFSW could partner with females across all risk groups. Same-sex partnerships were not incorporated. The per capita force of infection governed the rate at which susceptible sexually active individuals (S) acquired gonorrhoea. It depended on the annual rate of new partner acquisition, the number of sex acts per partnership per year, the proportion of those acts protected by condoms, the per-act probability of gonorrhoea transmission, and the prevalence of infection among potential partners.

The natural history structure for gonorrhoea followed the framework described by Garnett *et al.*[32] Following exposure, individuals entered either an asymptomatic (X) or symptomatic infection state, where men had a higher probability of symptomatic infection than women. Symptomatic individuals were further stratified based on whether they would access care (Y) or not (Z). The probability of accessing care was fixed at symptom onset and reflected both active care-seeking for symptoms and opportunistic treatment during unrelated healthcare visits. Treated individuals recovered and returned to the susceptible state, with treatment assumed to be fully effective. Untreated individuals cleared infection naturally, at a slower rate, and subsequently returned to the susceptible state. All recovered individuals were assumed to have no temporary or partial immunity. The model did not distinguish between anatomical sites of infection, with natural history parameter values chosen to represent an average across heterosexual routes of transmission.

Three diagnostic and treatment scenarios were considered: (i) a baseline scenario with syndromic management for individuals with symptomatic infection who accessed care, (ii) a diagnostic testing scenario in which POCTs were used in place of syndromic management for a proportion of symptomatic individuals who accessed care, and (iii) a screening scenario in which POCTs were offered to routine healthcare service attendees, regardless of symptoms. At baseline (Figure 6.1A), syndromic management was provided to individuals with urethral or vaginal discharge syndromes who accessed care. A proportion of these individuals were assumed to be appropriately treated for gonorrhoea through

syndromic management algorithms (M^y), while the remainder received inappropriate treatment (R^y) due to imperfect diagnostic sensitivity of the syndromic algorithms. No treatment was provided to symptomatic individuals who did not access care, or to asymptomatic individuals. The diagnostic testing scenario (Figure 6.1B) introduced POCTs among individuals accessing care for urethral or vaginal discharge syndromes. POCTs replaced syndromic management for the individuals tested, such that the treatment pathways were interdependent and mutually exclusive. Available tests were allocated across all symptomatic cases, irrespective of the underlying aetiology. For this allocation, the total number of eligible cases was estimated annually using the gonorrhoea aetiologic proportion (the proportion of symptomatic cases attributable to gonorrhoea). Those tested were either appropriately treated (T^y) or not appropriately treated (R^y) according to test sensitivity. Individuals who were not tested continued to receive syndromic management (M^y or R^y), as in the baseline scenario. The screening scenario (Figure 6.1C) introduced POCTs among routine healthcare service attendees, regardless of symptoms. Available tests were allocated among individuals based on their probability of attending PHC or ANC services. Tested individuals with infection were either appropriately treated (T^h) or not appropriately treated (R^h) according to test sensitivity. Syndromic management continued among individuals accessing care for symptoms.

Model equations representing population dynamics, sexual mixing, the force of infection, natural history, and treatment access pathways are provided in the Supplement (Text E.1.1).

6.2.2 Model parametrisation and calibration

Model parameter values were derived from multiple sources (Table 6.1, Text E.1.2). Demographic parameters were from UN World Population Prospects (WPP) 2024 projections between 1970 and 2030 (Table E1), including initial population size, annual entry into the 15–24-year-old group, annual pregnancy rates, and annual all-cause mortality rates. Sexual behaviour and care-seeking parameters for the general population were from Kenya DHS in 2003, 2008-09, 2014, and 2022,[372–375] and the Kenya Population-based HIV Impact Assessment in 2018 (Table E2).[376] These included the probability of being sexually active, number of partnerships per person per year, probability of condom use per sex act, probability of seeking care for genital discharge symptoms, and probability of attending PHC or ANC services. For FSW and CFSW, population size, sexual behaviour, and care-seeking parameters were derived from published literature (Table E2). Natural history parameters, including the per-act probability of transmission, probability of symptomatic infection, and annual rates of natural recovery and recovery with treatment, were also estimated from published literature (Table E2).

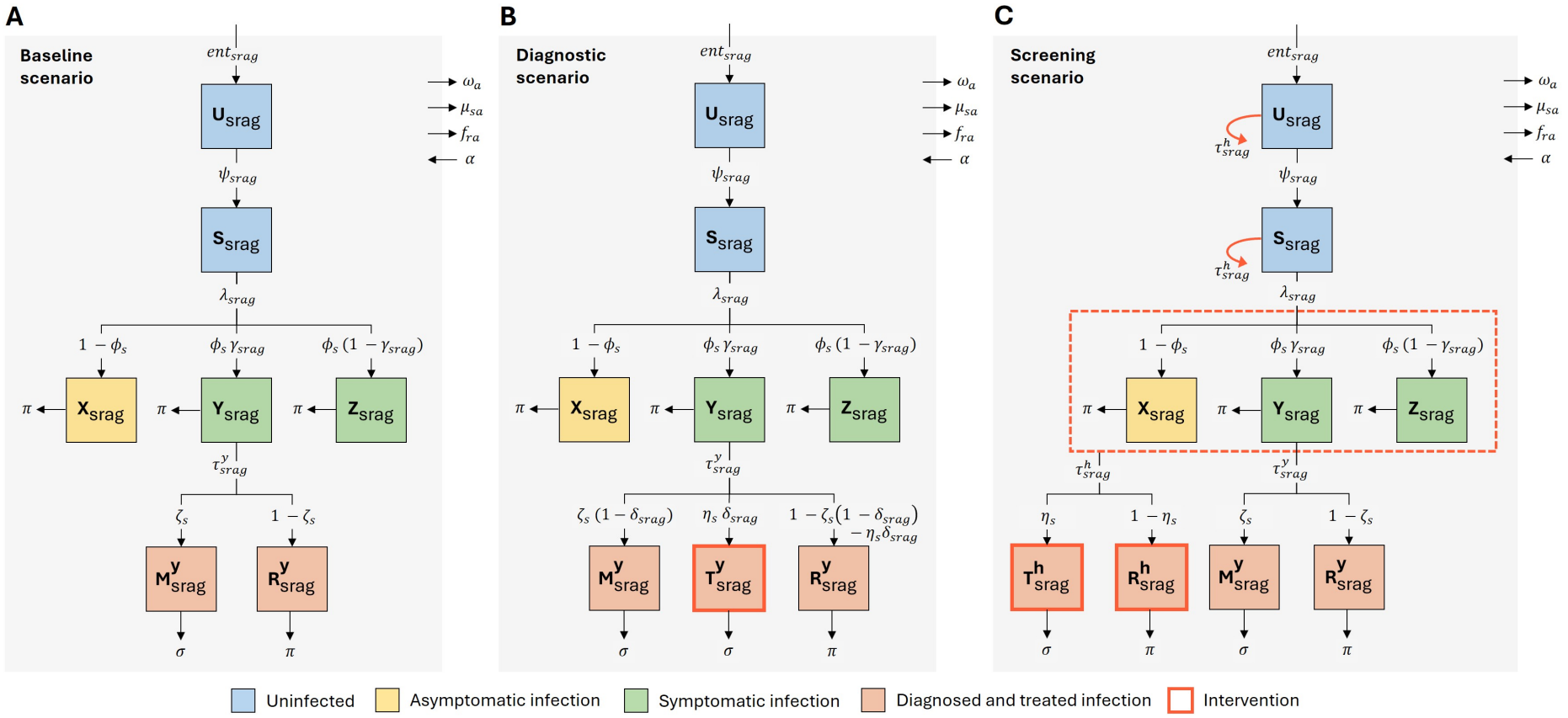


Figure 6.1: Flow diagram of gonorrhoea transmission model

Model structure showing diagnostic and treatment scenarios: (A) baseline with syndromic management, (B) diagnostic testing, and (C) screening. Population states shown as boxes: blue for uninfected (U: never sexually active, S: susceptible), yellow for asymptomatic infection (X), green for symptomatic infection (Y: with healthcare access, Z: without), and orange for diagnosed and treated infection (M^y: appropriate treatment following syndromic management, T^y or T^h: appropriate treatment following POCT for diagnosis or screening, R^y or R^h: inappropriate treatment). Dark orange boxes and arrows indicate POCT intervention components; the dotted box in (C) indicates that the intervention is applied to all three infection compartments. Subscripts denote stratification by sex (s), risk (r), age (a), and gestational status (g). Superscripts denote treatment among symptomatic individuals (y) or routine healthcare attendees (h). All infected individuals return to the susceptible state via natural recovery (π) or treatment (σ); these transitions are omitted for diagrammatic clarity. Outflows (ω_a, μ_{sa}, f_{ra}, α) represent transitions or exits from relevant compartment strata due to ageing, mortality, pregnancy, or return from pregnancy. Parameters are defined in Table 6.1.

Table 6.1: Parameter definitions, priors, and posteriors for transmission model

Description	Symbol	Prior Distribution	Posterior, Median (UI)	Source
Demography				
Initial population size in 1970	N^0_{srag}	Fixed	Table E1	[287]
Annual entrants into population	ent_{srag}	Fixed	Table E1	[287]
Probability of being in sexual risk group				
Male, low risk	υ_{ml}	Calculated	0.70 (0.60-0.80)	$1-\upsilon_{mi}-\upsilon_{mh}$
Male, intermediate risk	υ_{mi}	U(0.10, 0.20)	0.15 (0.10-0.20)	Assumption
Male, high risk (CFSW)	υ_{mh}	U(0.10, 0.20)	0.15 (0.10-0.20)	[372–375]
Female, low risk	υ_{fl}	Calculated	0.73 (0.67-0.79)	$1-\upsilon_{fi}-\upsilon_{fh}$
Female, intermediate risk	υ_{fi}	U(0.20, 0.30)	0.25 (0.20-0.30)	Assumption
Female, high risk (FSW)	υ_{fh}	U(0.01, 0.03)	0.02 (0.01-0.03)	[377]
Mean duration in age group (year)				
15-24 y	$1/\omega_y$	Fixed	10	-
25-49 y	$1/\omega_o$	Fixed	25	-
Rate of becoming pregnant among sexually active females ($year^{-1}$)	f_{ra}	Fixed	Table E1	[287]
Mean duration of pregnancy (year)	$1/\alpha$	Fixed	0.75	-
Rate of mortality ($year^{-1}$)	μ_{sa}	Fixed	Table E1	[287]
Sexual behaviour				
Probability of being sexually active				
All sex, low risk, 15-24 y, non-pregnant	q_{slyn}	U(0.50, 0.70)	0.59 (0.50-0.70)	[372–375]
All sex, all risk, 25-49 y, non-pregnant	q_{sron}	Fixed	1	[372–375]
Female, all risk, all age, pregnant	q_{frap}	Fixed	1	-
Relative probability of being sexually active among all sex, 15-24 y, non-pregnant				
Intermediate:low risk	$rq_{i:l}$	U(1.05, 1.15)	1.10 (1.05-1.15)	Assumption
High:intermediate risk	$rq_{h:i}$	U(1.05, 1.15)	1.10 (1.05-1.15)	Assumption
Rate of becoming sexually active ($year^{-1}$)	ψ_{srag}	Calculated	-	$-log(1-q_{srag})$
Number of partnerships per person ($year^{-1}$)				
All sex, low risk, 15-24 y, non-pregnant	c_{slyn}	U(1.00, 1.50)	1.24 (1.01-1.48)	[372–375]
Relative number of partnerships per person				
Male, intermediate:low risk	$rc_{mi:ml}$	U(3.00, 6.00)	4.20 (3.08-5.87)	Assumption
Male, high:intermediate risk	$rc_{mh:mi}$	U(2.50, 5.00)	2.84 (2.05-3.89)	[372,374]
Female, intermediate:low risk	$rc_{fi:fl}$	U(3.00, 6.00)	4.44 (3.12-5.93)	Assumption
Female, high:intermediate risk	$rc_{fh:fi}$	U(10.0, 25.0)	16.4 (10.4-24.5)	[378–391]
25-49:15-24 y	$rc_{o:y}$	U(0.85, 1.00)	0.92 (0.85-0.99)	[372–375]
Pregnant:non-pregnant	$rc_{p:n}$	U(0.85, 1.00)	0.92 (0.86-1.00)	[372–375]
Number of sex acts per person ($year^{-1}$)	n	U(52.0, 104.0)	79.6 (54.6-102.4)	Assumption
Probability of condom use per sex act				
All sex, low risk, 15-24 y, non-pregnant	χ_{slyn}	U(0.25, 0.35)	0.31 (0.26-0.35)	[372–375]
Relative probability of condom use per sex act				
Intermediate:low risk	$r\chi_{i:l}$	U(2.30, 2.50)	2.40 (2.31-2.50)	[372–376]
High:intermediate risk	$r\chi_{h:i}$	U(1.00, 1.15)	1.08 (1.00-1.15)	[379–383,385,387,388–392]
25-49:15-24 y	$r\chi_{o:y}$	U(0.85, 1.00)	0.94 (0.86-1.00)	[372–375]
Pregnant:non-pregnant, low risk	$r\chi_{lp:ln}$	U(0.10, 0.30)	0.20 (0.11-0.29)	[372–375]

Continued...

Description	Symbol	Prior Distribution	Posterior, Median (UI)	Source
Pregnant:non-pregnant, intermediate risk	$r\chi_{ip:in}$	U(0.10, 0.30)	0.20 (0.10-0.30)	[372–375]
Pregnant:non-pregnant, high risk	$r\chi_{hp:hn}$	U(0.85, 1.00)	0.93 (0.86-0.99)	Assumption
Efficacy of condoms per sex act	e	U(0.90, 1.00)	0.95 (0.90-1.00)	[15, 98, 274, 393]
Diagnosis and treatment				
Probability symptomatic individuals access care				
Male, low risk, 15-24 y, non-pregnant	γ_{mlyn}	U(0.65, 0.75)	0.70 (0.65-0.75)	[372–375]
Relative probability symptomatics access care				
Female:male	$r\gamma_{f:m}$	U(0.70, 0.90)	0.81 (0.70-0.90)	[372–375]
Intermediate:low risk	$r\gamma_{i:l}$	U(1.00, 1.10)	1.05 (1.00-1.10)	Assumption
High:intermediate risk	$r\gamma_{h:i}$	U(1.00, 1.10)	1.05 (1.00-1.10)	[389–391, 394, 395, 373, 374]
25-49 y:15-24 y	$r\gamma_{o:y}$	U(1.00, 1.10)	1.05 (1.00-1.10)	[372–375]
Pregnant:non-pregnant	$r\gamma_{p:n}$	U(1.00, 1.10)	1.05 (1.00-1.10)	[372–375]
Rate of treatment for symptomatic males (year^{-1})	τ_m^y	U(20.0, 40.0)	29.4 (20.6-39.4)	[10, 12, 26, 27, 32, 61, 188, 198, 396–398]
Relative rate of treatment among symptomatics				
Female:male	$r\tau_{f:m}^y$	U(0.50, 1.00)	0.76 (0.52-0.98)	[188, 198, 396–398]
Probability of POCT diagnosis among symptomatic	δ_{srag}	Fixed	Scenario specific	Text E.1.1
Probability of primary healthcare access among all risk, 15-24 y, non-pregnant				
Male	h_{mryn}	Fixed	0.70	[399–403]
Female	h_{fryn}	Fixed	0.75	[399–403]
Relative probability of primary healthcare access among all sex and risk				
25-49 y:15-24 y	$rh_{o:y}$	Fixed	1.05	[403]
Pregnant:non-pregnant	$rh_{p:n}$	Fixed	1.20	[372–375, 404, 405]
Rate of screening with POCT (year^{-1})	τ_{srag}^h	Fixed	Scenario specific	Text E.1.1
Sensitivity of syndromic management				
Male	ζ_m	U(0.80, 0.90)	0.85 (0.80-0.90)	[125]
Female	ζ_f	U(0.40, 0.50)	0.45 (0.40-0.50)	[116, 120]
Sensitivity of POCT				
Male	η_m	Fixed	0.95	[175]
Female	η_f	Fixed	0.95	[175]
Natural history				
Probability of transmission per sex act				
Female-to-male	κ_m	U(0.15, 0.30)	0.23 (0.15-0.30)	[7–9, 11–13]
Male-to-female	κ_f	U(0.10, 0.40)	0.19 (0.10-0.37)	[7, 10–13]
Probability infection is symptomatic				
Male	ϕ_m	U(0.45, 0.90)	0.60 (0.46-0.85)	[7, 11, 12, 30–32]
Female	ϕ_f	U(0.20, 0.45)	0.35 (0.22-0.44)	[7, 11, 12, 30–33]
Rate of natural recovery (year^{-1})	π	U(1.80, 2.80)	2.31 (1.84-2.76)	[7, 12, 14, 32, 40, 41]
Rate of recovery with treatment (year^{-1})	σ	U(26.0, 52.0)	39.0 (26.6-51.5)	[117]
Probability syndrome is caused by gonorrhoea				
Male (urethral discharge)	p_m	U(0.65, 0.8)	0.73 (0.65-0.80)	[321]
Female (vaginal discharge)	p_f	U(0.10, 0.20)	0.14 (0.10-0.20)	[321]

Row shading indicates fixed (light blue) or estimated (white) parameters. CFSW: clients of female sex workers, FSW: female sex workers, UI: Uncertainty interval, U: Uniform distribution (min, max), y: years. Table E2 provides detailed rationale and assumptions for parameter prior distributions and fixed values.

I specified parameter priors as uniform distributions to reflect uncertainty, using broad ranges for parameters for which empirical data were limited (Tables 6.1 and E2). In total, the model incorporated 23 input parameter groups, of which 14 were calibrated and 9 were fixed. Parameter groups were stratified by sex, risk, age, or gestational status, resulting in 41 distinct calibrated parameters.

The model was calibrated to four targets: (i) gonorrhoea prevalence among sexually active females in the general population, (ii) gonorrhoea prevalence among FSW, (iii) the male-to-female ratio for gonorrhoea prevalence, and (iv) the male-to-female ratio for all-cause symptomatic case rates (Table E3). I derived ranges for the first two targets by fitting a generalised linear mixed-effects meta-regression to study observations of gonorrhoea prevalence among general populations and FSW in Kenya between 2000–2024 (Text E.1.3, Table E4, Figure E1). Ranges for the male-to-female ratios were derived from the gonorrhoea sex-prevalence ratio for SSA in Chapter 2, and by calculating a symptomatic case rate ratio for Kenya using the four DHS (Text E.1.3).[372–375] For initial candidate parameter combinations, I generated 50 000 parameter combinations from the uniform prior distributions of the 41 stratified parameters using Latin Hypercube Sampling.[406] For each parameter combination, I simulated the model from 1970 to 2030 and retained those where equilibrium outputs were within pre-specified ranges for all four calibration targets.

6.2.3 Morbidity and health outcomes

Health losses attributed to gonorrhoea related to both acute infection and longer-term sequelae. For men, these included urethritis and epididymitis (EDS); for non-pregnant women, symptomatic cervicitis, pelvic inflammatory disease (PID), chronic pelvic pain (CPP), ectopic pregnancy (EP), and tubal factor infertility (TFI); and for pregnant women, symptomatic cervicitis, stillbirth, low birth weight (LBW), neonatal pneumonia (NP), and ophthalmia neonatorum (ON).

Probability tree models were used to quantify the likelihood of these sequelae following gonorrhoea infection. For men and non-pregnant women, I followed the model frameworks and assumptions described by Li *et al.*[407] For men, urethritis was assumed to occur during each episode of symptomatic infection, while EDS could develop during untreated infection irrespective of symptom status (Figure 6.2A).[407] For non-pregnant women, PID could develop irrespective of treatment or symptom status and could lead to CPP, EP, or TFI, occurring alone or in combination (Figure 6.2B).[407] For pregnant women, I developed a new tree structure where untreated infection episodes could result in either stillbirth or live birth, with the latter potentially leading to LBW, NP, or ON via vertical transmission (Figure

6.2C).[71,369] I excluded other adverse pregnancy outcomes, such as premature rupture of membranes, preterm birth, and neonatal death,[71,369] due to complex interactions with modelled sequelae and insufficient evidence to support parameterisation without introducing substantial uncertainty.

I quantified health losses using quality-adjusted life years (QALYs), where one QALY equals one year lived in perfect health. Each health state was assigned a utility value between 0 (death) and 1 (perfect health), where a utility decrement (1 - utility) represents the fraction of a healthy year lost due to that outcome. To calculate QALY losses at the population level, I used posterior estimates of gonorrhoea incidence and duration, distinguishing between episodes that were symptomatic or asymptomatic, and treated or untreated. For acute infection episodes (urethritis or symptomatic cervicitis), I multiplied person-years of infection for each subgroup by the corresponding utility decrement to capture shorter-term reductions in quality of life. For longer-term sequelae, I multiplied the number of incident infections by the probability that each sequela would result from an infection episode, the duration of the sequela, and its associated utility decrement. Since the probability of developing PID increases

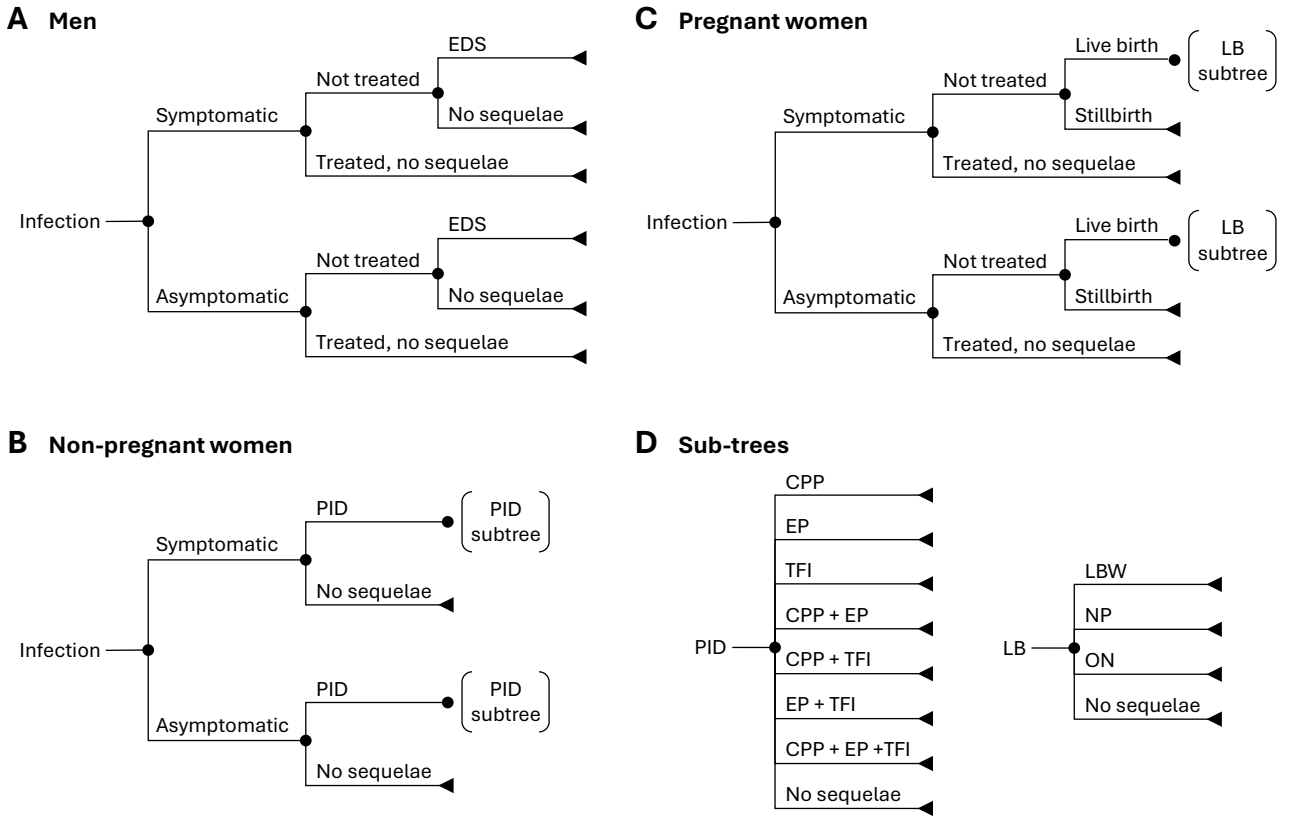


Figure 6.2: Probability trees for gonorrhoea sequelae

Probability trees for sequelae of gonorrhoea among (A) men, (B) non-pregnant women, and (C) pregnant women, with complications of PID and LB shown as separate subtrees (D). CPP: chronic pelvic pain, EDS: epididymitis, EP: ectopic pregnancy, LB: live birth, LBW: low birth weight, NP: neonatal pneumonia, OP: ophthalmia neonatorum, PID: pelvic inflammatory disease, TFI: tubal factor infertility. Trees and subtrees for men and non-pregnant women adapted from Li *et al.* 2023.[407]

Table 6.2: Parameters for the probability, duration, and utility of gonorrhoea sequelae

Sequelae	Probability per infection episode	Duration (years)	Utility weight
Men			
Urethritis, given symptomatic infection	1.000[407]	Simulated[*]	0.961[74,410]
Urethritis, given asymptomatic infection	0[407]	Simulated[*]	1.000[410]
EDS, given untreated infection	0.043[407†]	0.019[74]	0.665[74,410]
Non-pregnant women			
Symptomatic infection	Simulated[*]	Simulated[*]	0.994[410]
Asymptomatic infection	Simulated[*]	Simulated[*]	1.000[410]
PID	Simulated with annual rate[‡]	0.028[74]	0.756[74,410]
CPP, given PID	0.261[407†]	10.00[407,411]	0.759[74,410]
TFI, given PID	0.168[407†]	10.00[407,411]	0.905[74,410]
EP, given PID	0.070[407†]	0.078[74]	0.731[74,410]
Pregnant women			
Symptomatic infection	Simulated[*]	Simulated[*]	0.994[410]
Asymptomatic infection	Simulated[*]	Simulated[*]	1.000[410]
Stillbirth, given untreated infection	0.047[‖]	Neonatal: 64.00[287] Maternal: 10.00[412]	Neonatal: 0 Maternal: 0.862[412]
LBW, given LB with untreated infection	0.176[‖]	Neonatal: 64.00[287] Maternal: 10.00[§]	Neonatal: 0.894[413] Maternal: 0.999[§]
NP, given LB with untreated infection	0.160[414†]	0.167[74]	Neonatal: 0.790[74] Maternal: 0.999[§]
ON, given LB with untreated infection	0.180[414†]	0.500[74]	Neonatal: 0.970[74] Maternal: 0.999[§]

Utilities with two sources were averaged. [*]Probability and duration of symptomatic and asymptomatic gonorrhoea varied per posterior parameter combination. [†]Probabilities of sequelae approximated from studies of chlamydia, due to limited gonorrhoea-specific data.[407] [‡]Probability of PID calculated using mean annual progression rate of 0.15 per year[408] and posterior estimates for duration of infection.[407] [‖]Probabilities of stillbirth and LBW among pregnant women with untreated gonorrhoea estimated by combining odds ratios from meta-analyses with background prevalence in the general population of pregnant women.[71,415,416] [§]Assumption.[417] CPP: chronic pelvic pain, EDS: epididymitis, EP: ectopic pregnancy, LB: live birth, LBW: low birth weight, NP: neonatal pneumonia, ON: ophthalmia neonatorum, PID: pelvic inflammatory disease, TFI: tubal factor infertility.

with infection duration, I based risk on person-years of infection and applied an annual progression rate.[407,408] To estimate QALY losses from combined PID sequelae, I accounted for the probability of these sequelae co-occurring, their duration of overlap, and their joint utility (Text E.1.4).[409]

Published literature and global burden estimates informed parameters on the probability, duration, and utility of each outcome. Probability estimates for most sequelae were derived from longitudinal studies of chlamydia, due to sparse direct evidence for gonorrhoea (Table 6.2).[407,409] I estimated the probability of stillbirth and LBW among pregnant women with untreated gonorrhoea by combining odds ratios from meta-analyses that assessed the association between gonorrhoea and these outcomes with the background prevalence of each outcome in the general pregnant population (Text E.1.4).[71,415,416] I obtained values for utility weights and their associated durations from the Global Burden of Disease

study,[410,413] the Institute of Medicine study,[74] and other published literature. The duration of sequelae varied by type, with CPP and TFI classified as chronic sequelae that manifested 5 years after infection and persisted for 10 years.[407,411] I valued stillbirth-associated losses as equivalent to neonatal deaths (i.e. utility weight of 0 over the life expectancy at birth), due to limited consensus on alternative quantification approaches.[418] Mothers who experienced a stillbirth were assigned a reduction in utility lasting 10 years to reflect the long-term psychological impact.[412] I also included utility decrements for mothers of infants with LBW, NP, or ON.[417]

For outcomes extending beyond the year of infection, I discounted life years to the infection year at 3% annually (Text E.1.4).[407] Discounted lifetime QALYs lost were then aggregated across all probability tree pathways for infections acquired in a given year to estimate annual burden by population group.

6.2.4 Intervention scenarios

I modelled interventions implementing POCTs either for diagnostic testing among individuals accessing syndromic management or for screening among routine healthcare service attendees. Both strategies were alternatively introduced among five distinct but non-mutually exclusive target populations: FSW, CFSW, pregnant women, sexually active AGYW who were non-pregnant, and men in the general population, representing a total of 10 scenarios. To assess the potential impact of these scenarios when POCT availability was limited, I assumed 25 000 tests were available annually. This quantity was informed by early implementation data for tuberculosis rapid diagnostic testing (Xpert MTB/RIF),[419] and was sufficient to test approximately 0.1% of the modelled adult population each year. To assess the maximum potential impact of these scenarios in a situation when POCT availability was not constrained, I simulated all 10 scenarios at full coverage among eligible individuals in each target population. I assumed that POCTs met the WHO optimal TPP (95% sensitivity, 98% specificity).[175] Interventions were introduced into the baseline scenario from the beginning of 2025, where prevalence was at equilibrium, until the end of 2030. I assessed the population-level impact of each intervention scenario by comparing epidemiologic and health outcomes to the baseline scenario within each posterior parameter combination. Model outcomes over the 6-year intervention period were POCT coverage among the eligible target population, the proportion of all incident infections averted, the proportion of all QALYs gained, the number of infections averted per test used, and the number of QALYs gained per test used. I compared these outcomes across testing strategies to assess whether target population prioritisation differed when aiming to reduce transmission versus improve overall health. Results were reported as medians with uncertainty intervals (UIs) corresponding to the 2.5th and 97.5th percentiles.

6.2.5 Sensitivity analyses

To assess which model parameters drove variability in intervention impact estimates, I calculated partial rank correlation coefficients (PRCCs) between posterior parameter values and the proportion of incident infections averted for each scenario.[406] To assess which parameters influenced the relative prioritisation of target populations, I ranked each population group based on the proportion of infections averted for each parameter combination and test strategy, and then calculated PRCCs between posterior parameter values and the resulting prioritisation ranks. I classified PRCCs as having a strong ($|\text{PRCC}| \geq 0.6$), moderate ($0.4 \leq |\text{PRCC}| < 0.6$), weak ($0.2 \leq |\text{PRCC}| < 0.4$), or very weak ($|\text{PRCC}| < 0.2$) correlation.[420] To assess the sensitivity of intervention impact estimates to assumptions about pregnancy-related sequelae, I calculated the proportion of QALY losses averted per scenario after (i) increasing the utility weight for stillbirth from 0 to 0.5 and reducing the duration of infant LBW-associated losses from lifetime to 10 years, and (ii) increasing the utility weight for stillbirth from 0 to 1 to exclude QALY losses associated with stillbirth in infants. All sensitivity analyses were conducted assuming limited POCT availability, to reflect resource constraints expected during early-stage implementation.

All analyses were conducted in R version 4.4.1; ordinary differential equations were implemented in `odin` version 1.5.10,[421] Latin Hypercube Sampling used `lhs` version 1.2.0,[422] diagnostic test performance adjustments used `rstan` version 2.32.6,[291] and meta-regression modelling to synthesise prevalence data for model calibration target ranges used `glmmTMB` version 1.1.9.[292]

6.3 Results

6.3.1 Model calibration

Of 50 000 initial sampled parameter combinations, 536 were retained after calibration. The size of the modelled population by sex, age group, and gestational status aligned over time with national estimates from the UN WPP (Figure E2).[287] Equilibrium outcomes from the calibrated parameter combinations tended toward the lower end of the calibration target ranges for prevalence in sexually active females and for the male-to-female prevalence ratio (Figure E3). Posterior parameter samples had very broad distributions (Figure E4) and weak pairwise correlations (Figure E5).

6.3.2 Gonorrhoea burden at baseline

In 2025, the prevalence of gonorrhoea among sexually active 15-49-year-olds was 1.5% (UI: 0.9-2.8%). Prevalence was higher among FSW (4.7% [2.4-6.5%]) and their clients (3.6% [2.0-6.7%]) than pregnant women (1.9% [1.1-3.6%]), AGYW (1.8% [1.1-3.3%]), and men in the general population (1.3% [0.7-2.3%]; Table E7). The number of incident infections per 100 individuals followed a similar pattern across target populations (Figure 6.3A). There were a total 1 063 670 (617 783 - 1 911 887) incident infections in 2025, of which 50.0% (45.4-55.0%) were among men (Figure 6.3B). A substantially higher proportion of these incident infections remained untreated among females (91.7% [87.9-94.9%]) compared to males (63.7% [50.6-73.8%]; Figure E6). Of the total incident infections, CFSW and FSW accounted for 21.4%

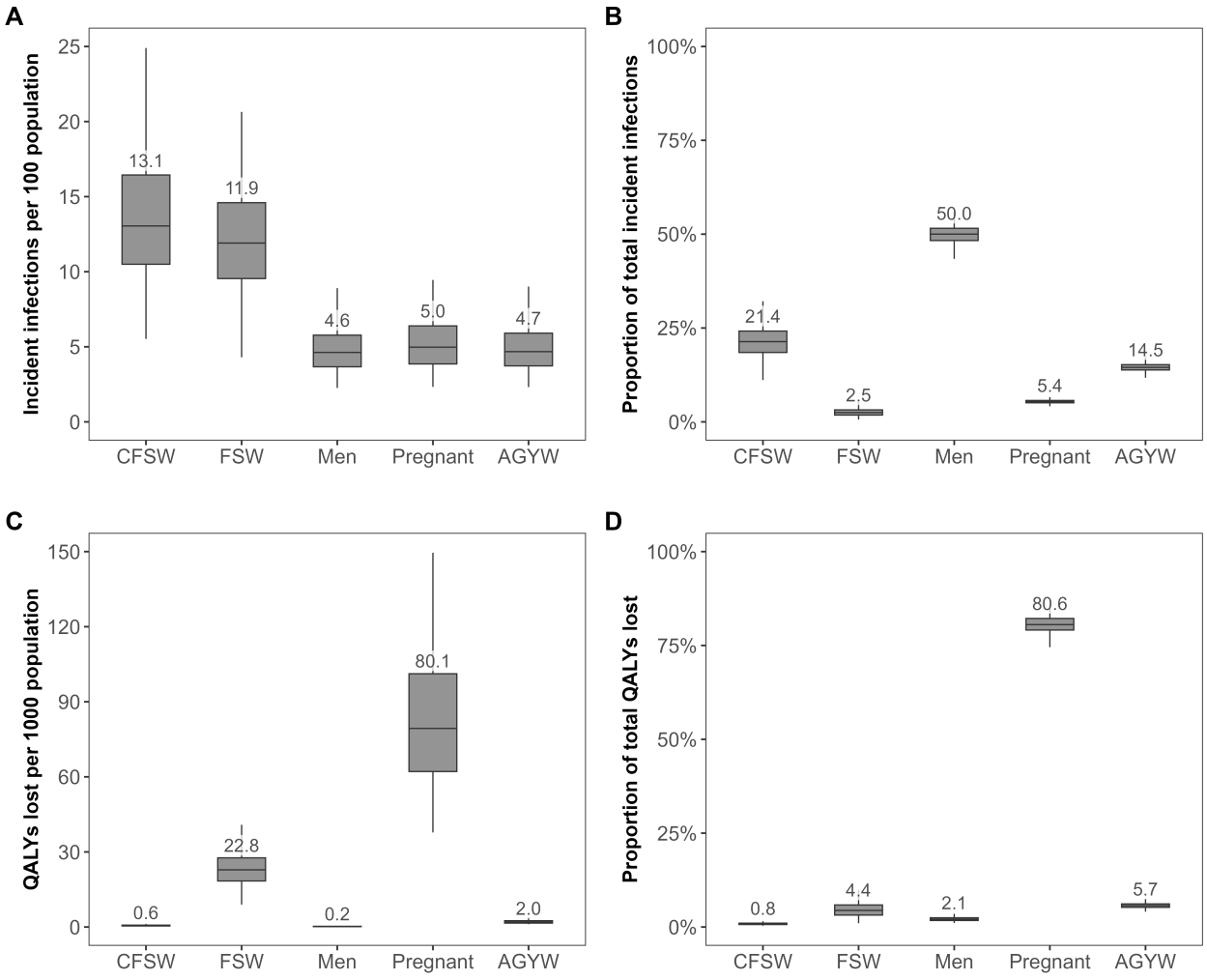


Figure 6.3: Gonorrhoea burden at baseline, 2025

Baseline outcomes per target population: (A) incident infections per 100 individuals, (B) lifetime QALYs lost per 1 000 individuals, (C) share of total incident infections, and (D) share of total QALYs lost, for infections in 2025. Target populations are not mutually exclusive. Boxplots show median (horizontal line), interquartile range (box), and uncertainty interval (whiskers). AGYW: sexually active non-pregnant adolescent girls and young women; CFSW: clients of female sex workers; FSW: female sex workers.

(13.7-29.3%) and 2.5% (1.0-5.6%) of cases, while comprising 14.6% (10.4-29.5%) and 2.0% (1.0-3.0%) of the total male and female populations, respectively.

The burden of gonorrhoea varied substantially across target populations. Lifetime QALYs lost per 1 000 individuals in each population group due to sequelae from infections in 2025 were 80.1 (UI: 45.1-145.0) among pregnant women, 22.8 (11.1-36.0) among FSW, 2.0 (1.2-3.7) among AGYW, 0.6 (0.3-1.2) among CFSW, and 0.2 (0.1-0.5) among men overall (Figure 6.3C). These per-capita differences translated into a disproportionate share of total health losses among pregnant women. Of the 115 105 (66 430 - 207 620) total lifetime QALYs lost due to infections in 2025, 80.6% (76.1-83.8%) were among pregnant women and their infants (Figure 6.3D), of which most losses were attributed to infant outcomes of stillbirth (70.0%) and LBW (26.5%; Figure E7). Modelled estimates of the number of stillbirth and LBW cases were consistent with the published estimates used to inform parametrisation (Table E8).

6.3.3 Intervention impact and prioritisation

Limited POCT availability

With a limited supply of gonorrhoea POCTs (25 000 annually; approximately 0.1% of adult population) between 2025 and 2030, the proportion of eligible individuals tested varied widely by target population and testing strategy (Table E9). When POCTs were allocated for diagnostic testing among individuals accessing care for urethral or vaginal discharge of any aetiology, coverage ranged from 8.0% (UI: 3.9-16.2%) among men in the general population to 66.8% (26.0-100.0%) among FSW. When POCTs were allocated for screening among individuals attending routine healthcare services, coverage ranged from 0.14% (0.14-0.15%) among men to 5.8% (3.9-11.1%) among FSW.

The diagnostic testing strategy averted 2.2% (UI: 0.8-5.3%) of population-level incident infections between 2025 and 2030 when testing CFSW, followed by 2.1% (0.6-5.6%) when testing FSW, 1.3% (0.5-3.0%) when testing general population men, 1.0% (0.5-2.1%) when testing pregnant women, and 0.9% (0.4-2.0%) when testing AGYW (Figures 6.4A and E8, Table E9). This variation reflected differences in POCT coverage and the proportion of symptomatic infections attributed to gonorrhoea (Figure E8). The impact of the screening strategy was substantially smaller, averting 0.6% (0.2-1.5%) of all incident infections when testing FSW and 0.6% (0.4-0.8%) when testing CFSW, followed by 0.13% (0.11-0.16%) when testing AGYW, 0.12% (0.10-0.13%) when testing general population men, and 0.08% (0.06-0.10%) when testing pregnant women (Figure 6.4A). The relative difference between target populations with the highest and lowest impact was narrower for diagnostic testing (2.4-fold [UI:

1.0-5.0] difference for CFSW vs. AGYW) than for screening (7.8-fold [2.4-19.4] for FSW vs. pregnant women). The impact of diagnostic testing overlapped between target populations, while for screening, there were significant differences between groups (7.8-fold [2.4-19.4] higher for FSW vs. pregnant women; 5.0-fold [3.7-7.0] higher for CFSW vs. general population men).

The relative impact on QALYs gained across target populations mirrored that of infections averted, except for strategies among pregnant women, who accounted for the largest share of health losses (Figure 6.4B, Table E9). The diagnostic testing strategy averted 3.5% (UI: 1.8-7.2%) of population-level lifetime QALY losses when testing pregnant women, which exceeded the impact of testing FSW or CFSW. Screening pregnant women averted 0.29% (0.25-0.34%) of QALY losses, which was intermediate between screening high-risk and lower-risk populations. When comparing test strategies, diagnostic testing of pregnant women averted 5.5-fold (UI: 2.3-14.9) more QALY losses than screening FSW.

Patterns in population-level impact across scenarios were consistent whether measured by overall effectiveness (percentage of incident infections averted or QALYs gained; Figure 6.4A,B) or efficiency (number of incident infections averted or QALYs gained per 100 tests used; Figure 6.5A,B).

Maximum POCT availability

Full coverage of gonorrhoea POCTs among eligible individuals in each target population between 2025 and 2030 required substantially more tests relative to limited supply scenarios, particularly for screening, which had larger impacts but with diminishing efficiency (Figures 6.4C,D and 6.5C,D; Table E10). The diagnostic testing strategy averted 15.3% (UI: 6.8-28.9%) of all incident infections when testing general population men, followed by 11.6% (4.9-24.1%) when testing CFSW, 6.9% (3.8-10.7%) when testing AGYW, 3.1% (0.7-10.0%) when testing FSW, and 3.0% (1.6-5.0%) when testing pregnant women (Figure 6.4C). Patterns for QALYs gained across target populations mirrored those of infections averted, although diagnostic testing among pregnant women averted 10.7% (6.4-15.8%) of population-level lifetime QALY losses (Figure 6.4D). Diagnostic testing efficiency was similar under limited and maximum POCT availability across all target populations (Figure 6.5).

The screening strategy averted more infections and QALY losses than diagnostic testing across all target populations (Figure 6.4C,D; Table E10) but was consistently less efficient (Figure 6.5C,D). Screening male populations had the largest impacts, averting 45.8% (40.3-49.1%) of infections and 45.5% (40.1-48.5%) of QALY losses when targeting men, and 36.3% (27.7-42.2%) and 36.1% (27.9-41.9%) when targeting CFSW, respectively, but was far less efficient for men (2.8 [1.7-4.9] infections and 0.3

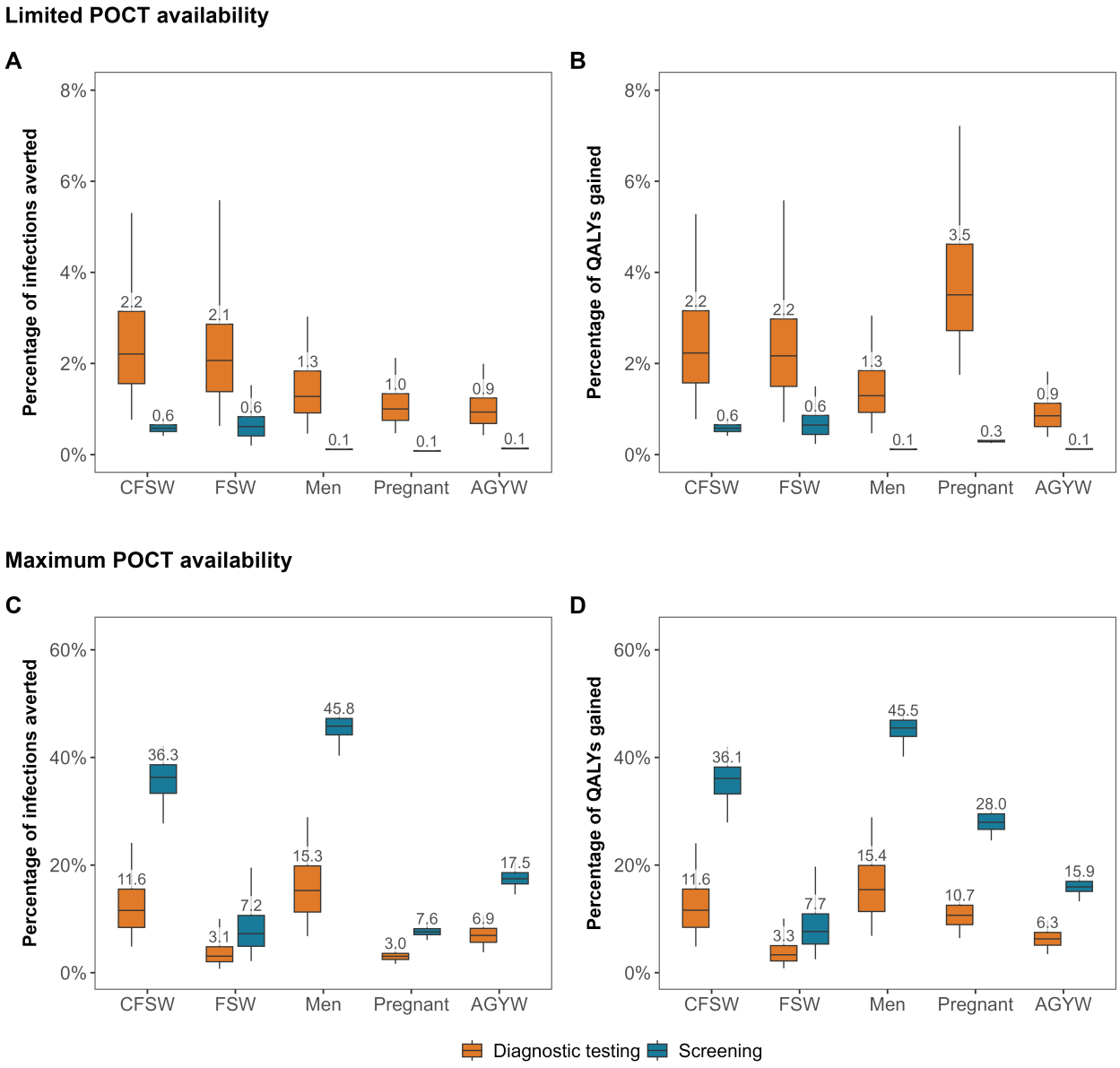


Figure 6.4: Population-level impact of diagnostic testing and screening strategies among each target population under varying POCT availability, 2025-2030

Population-level impact of deploying gonorrhoea POCTs among each of five target populations between 2025 and 2030 through either diagnostic testing of individuals accessing care for urethral or vaginal discharge, or screening of attendees at routine healthcare services. Panels A-B show impact for limited POCT availability (25 000 annually); Panels C-D show impact for maximum POCT availability (100% coverage of eligible target population annually). Impact measured as (A, C) percentage of incident infections averted and (B, D) percentage of QALYs gained, relative to baseline with syndromic management only. Boxplots represent the median (horizontal line), interquartile range (box), and uncertainty interval (whiskers). AGYW: sexually active non-pregnant adolescent girls and young women; CFSW: clients of female sex workers; FSW: female sex workers; POCT: point-of-care test; QALY: quality-adjusted life year.

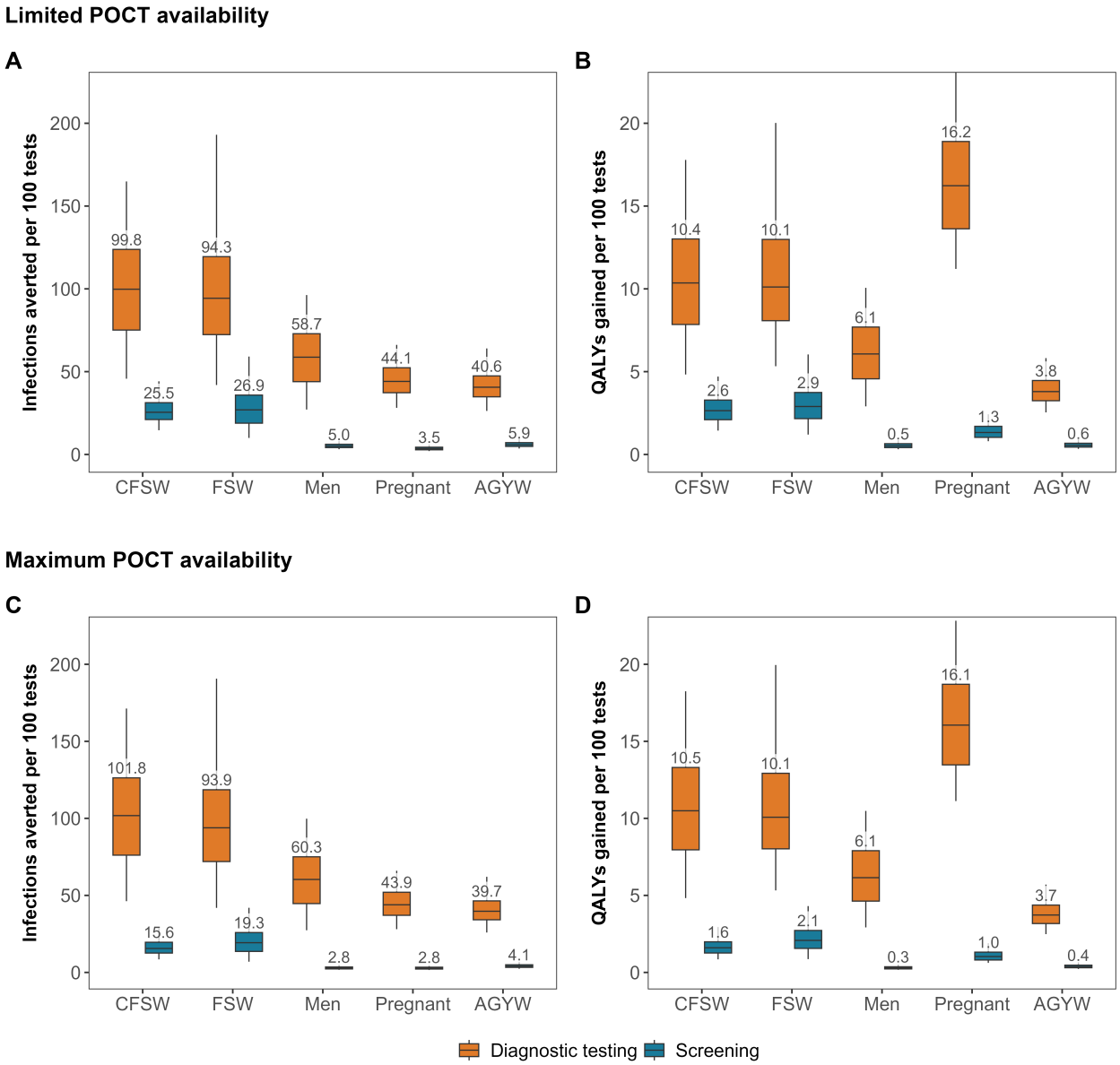


Figure 6.5: Efficiency of diagnostic testing and screening strategies among each target population under varying POCT availability, 2025–2030

Population-level efficiency of deploying gonorrhoea POCTs among each of five target populations between 2025 and 2030 through either diagnostic testing of individuals accessing care for urethral or vaginal discharge, or screening of attendees at routine healthcare services. Panels A-B show outcomes for limited POCT availability (25 000 POCTs annually); Panels C-D show outcomes for maximum POCT availability (100% coverage of eligible target population annually). Outcomes measured as (A, C) number of incident infections averted per 100 POCTs used and (B, D) number of QALYs gained per 100 POCTs used, relative to baseline with syndromic management only. Boxplots represent the median (horizontal line), interquartile range (box), and uncertainty interval (whiskers). AGYW: sexually active non-pregnant adolescent girls and young women; CFSW: clients of female sex workers; FSW: female sex workers; POCT: point-of-care test; QALY: quality-adjusted life year.

[0.2-0.5] QALY losses averted per 100 tests) than CFSW (15.6 [8.5-28.6] and 1.6 [0.9-3.0]). Screening pregnant women averted 28.0% (24.6-31.8%) of QALY losses and was also less efficient than screening higher-risk populations. Screening was less efficient under maximum than limited POCT availability across all target populations, reflecting diminishing returns at higher coverage.

6.3.4 Sensitivity analyses

Several parameters influenced the estimated impact of each testing strategy across targeted populations. For diagnostic testing, the proportion of all infections averted when targeting male populations correlated negatively with the sensitivity of urethral discharge syndrome management (PRCC ζ_m: -0.71 [UI: -0.76; -0.67] for both CFSW and general population men; Figure E9A), reflecting the smaller marginal benefit of POCTs when syndromic algorithms have high diagnostic sensitivity. For screening, the proportion of infections averted when targeting CFSW or FSW correlated negatively with the size of each population (PRCC υ_{mh}: -0.91 [-0.92; -0.89], υ_{fh}: -0.57 [-0.65; -0.52]; Figure E9B), an association not estimated for diagnostic testing. Across both strategies, the proportion of infections averted increased when the targeted population had more sexual partnerships, reflecting the role of testing and treatment in reducing onward transmission.

When prioritising target populations based on the proportion of infections averted through diagnostic testing, population ranking varied across parameter combinations, with only 24% (n=131/536) maintaining the same order as median estimates. Most parameter combinations resulted in prioritising either CFSW (n=299/536, 55.8%) or FSW (n=234/536, 43.7%) as the highest impact target population. Prioritisation order was most strongly associated with urethral discharge syndromic management sensitivity, with positive correlations for general population men (PRCC ζ_m: 0.75 [0.72-0.79]) and CFSW (0.67 [0.62-0.72]) and negative correlations for FSW (-0.67 [-0.71; -0.62]) and pregnant women (-0.59 [-0.64; -0.53]; Figure E10A). For screening, 53% (n=282/536) of parameter combinations maintained the same target population prioritisation order as median estimates, with either FSW (n=297/536, 55.4%) or CFSW (n=239/536, 44.6%) prioritised as highest impact. The relative number of sexual partnerships among FSW was strongly correlated with prioritisation order for both FSW and CFSW (PRCC $rc_{fh:fi}$: -0.69 [-0.75; -0.66] and 0.69 [0.65-0.74]; Figure E10B).

Targeting testing among pregnant women to maximise QALYs gained remained beneficial under alternative assumptions for health losses from stillbirth and LBW (Figure E11). Diagnostic testing among pregnant women averted 3.0% (1.5-6.2%) of population-level QALY losses when the stillbirth

utility weight was increased from 0 to 0.5 and the duration of LBW-associated losses was reduced from lifetime to 10 years, and 2.6% (1.3-5.4%) of QALY losses when the stillbirth utility weight was increased from 0 to 1. In both cases, the impact of testing pregnant women remained higher than that of testing any other target population.

6.4 Discussion

In this chapter, I developed and calibrated a compartmental transmission model to evaluate how a limited supply of gonorrhoea POCTs could be allocated across different testing strategies and target populations to maximise reductions in population-level incidence and morbidity in Kenya between 2025 and 2030. At baseline, gonorrhoea prevalence was highest among FSW and their clients, while the largest QALY losses were associated with untreated infections during pregnancy, predominantly due to severe neonatal sequelae. Introducing an annual supply of 25 000 POCTs, equivalent to testing approximately 0.1% of the adult population each year, had a meaningful impact relative to the limited scale of testing. The diagnostic testing strategy averted between 0.9% and 2.2% of all incident infections, depending on the target population, with the greatest impact when testing FSW (2.1%) or their clients (2.2%). Screening had a lower impact overall, averting up to 0.6% of all infections when targeting FSW or CFSW. The largest reduction in morbidity was achieved with diagnostic testing among pregnant women, averting 3.5% of total QALY losses. With full POCT coverage among eligible individuals, the impact of screening surpassed that of diagnostic testing across all target populations, although it remained less efficient. Diagnostic testing and screening were most impactful when targeting men (15.3% and 45.8% of infections averted) and CFSW (11.6% and 36.3%), and averted more QALY losses than targeting pregnant women. Both testing strategies were more efficient among higher-risk than lower-risk populations.

A key strength of my analysis was the direct comparison of diagnostic testing and screening strategies for POCT deployment across multiple potential target populations. Previous modelling studies in SSA have evaluated either screening[271,272] or diagnostic interventions,[273,274] without considering constraints in test availability. Another strength lies in evaluating the impact of POCTs on both incidence and morbidity. I extended the analysis by Li *et al.*,[407] which quantified QALY losses from gonorrhoea sequelae among men and non-pregnant women, by incorporating losses related to adverse pregnancy outcomes. While prior modelling has focused primarily on transmission dynamics, several of their findings were consistent with mine. An individual-based model of high-coverage opportunistic screening

in South Africa found that targeting FSWs was most efficient, while targeting larger groups, such as youth or individuals in HIV care, achieved greater reductions in overall transmission.[271] Another South African study estimated that POCT-based screening among ANC attendees could substantially reduce adverse pregnancy outcomes.[272] Models evaluating POCTs among symptomatic FSW[273,274] and women in long-term partnerships[274] found that even moderate-sensitivity tests could outperform syndromic management in reducing infections and overtreatment.

Diagnostic testing was more impactful than screening across all target populations when POCT availability was limited, but this pattern reversed under full POCT coverage. Testing symptomatic individuals, who were more likely to be infected than those attending routine healthcare services, yielded a higher positivity rate, making diagnostic testing a more efficient use of limited resources. When POCT availability was increased to enable full coverage, screening averted more infections overall, but remained less efficient. It enabled detection and treatment of asymptomatic infections that would otherwise go unnoticed, with the highest absolute impact achieved when targeting larger population groups. However, reaching such high coverage would require substantially more tests than are likely to be available during early phases of POCT implementation. Moreover, the modelled screening scenarios did not incorporate assumptions about how screening would be delivered or the timing of implementation. For example, reaching CFSW would require targeted outreach interventions, and screening during pregnancy would typically occur at a single ANC visit rather than uniformly across gestation. These factors are likely to affect the feasibility and impact of such screening strategies in real-world settings.

Diagnostic testing averted more infections among male than female populations, despite the already high sensitivity of syndromic management for urethral discharge (80–90%)[125] compared to POCTs (95%) for detecting gonorrhoea. This outcome was unexpected, given the poor sensitivity of the vaginal discharge syndrome (40-50%).[116] It likely reflects the higher likelihood of symptom presentation and care-seeking among males, as well as the lower proportion of syndromic cases attributed to gonorrhoea in females. My findings suggest that the WHO Minimal TPP for symptomatic testing (80% sensitivity)[175] may not offer substantial benefit over syndromic management for males, and that prioritising female populations may yield greater marginal gains for lower sensitivity tests. In this analysis, I assumed POCTs met the WHO Optimal TPP (95% sensitivity),[175] which may not reflect the performance of forthcoming tests. As new POCTs approach market introduction, their performance and optimal use-cases by sex will require re-evaluation. Moreover, since vaginal discharge syndrome is caused

by several STIs, multiplexed POCTs may offer greater diagnostic utility among female populations. Future studies should assess their comparative effectiveness and the implications for prioritising across target populations.

Programmes aiming to maximise reductions in incidence with limited POCTs should prioritise diagnostic testing among symptomatic individuals in higher-risk populations. Testing FSW and their clients was more efficient at averting new infections than testing lower-risk groups, due to higher prevalence and the greater indirect benefits of reducing onward transmission. As POCT availability increases and coverage among higher-risk groups approaches saturation, diagnostic testing should be expanded to include lower-risk symptomatic males to further reduce incident infections across the broader population, contingent on the performance of available diagnostic tests. Simultaneously targeting FSW and their clients may yield diminishing marginal returns due to overlapping indirect effects within their shared transmission networks. However, in this analysis, I evaluated each population group in isolation. Future modelling could assess the impact of combined targeting strategies, as well as dynamic prioritisation in response to changing epidemiologic conditions and POCT availability.

Programmes aiming to maximise reductions in morbidity with limited POCTs should prioritise diagnostic testing among symptomatic pregnant women. Although pregnant women had relatively low incidence, untreated infections led to disproportionately high QALY losses, making diagnostic testing among this group the most efficient strategy for reducing population-level morbidity. Since pregnant women did not contribute substantially to ongoing transmission, focusing exclusively on this group may limit opportunities for broader infection control. In contexts with increasing gonorrhoea prevalence, strategies that maximise incidence reduction may therefore take precedence and help mitigate morbidity over the longer term. Conversely, in settings with stable or declining prevalence, targeting high-burden populations may yield more immediate and impactful health gains.

Screening among asymptomatic populations offered limited population-level benefit, particularly when implemented among populations with low gonorrhoea prevalence. Even highly sensitive and specific POCTs can yield low positive predictive values in such contexts, resulting in a high proportion of false positive results. This increases the likelihood of unnecessary treatment and associated healthcare expenditure, and may contribute to the emergence and spread of AMR. Although my model did not simulate treatment of false positive cases or resistance dynamics, modelling from high-income settings suggests that intensified screening of asymptomatic individuals, particularly among high-risk populations such as MSM, may drive resistance by increasing antibiotic exposure.[423] In contrast,

diagnostic testing is likely to reduce unnecessary treatment compared to syndromic management,[273,274] which has poor specificity for both urethral (60-70%)[125] and vaginal discharge (55–60%).[116] These considerations reinforce the benefit of prioritising POCTs for diagnostic use, particularly among populations with frequent antibiotic exposure and high rates of over treatment, such as higher-risk and female populations. Looking ahead, WHO has developed TPPs for POCTs capable of detecting genetic markers of gonorrhoea resistance, which could support more targeted treatment and improved antimicrobial stewardship.[175] Integrating resistance dynamics into future models and prioritisation frameworks will be critical for aligning gonorrhoea control efforts with antimicrobial stewardship.

My analysis was constrained by data limitations affecting the transmission model. Population-representative studies of gonorrhoea prevalence were limited. Most prevalence estimates for FSW were derived from enrolment testing in urban cohort studies, which may not be nationally representative. Prevalence data were sparse for men and absent for CFSW, so calibration targets relied on male-to-female ratios for both prevalence and symptomatic case rates. Similar gaps existed for AGYW and pregnant women, preventing the inclusion of calibration targets for these populations. The combination of limited prevalence data and absence of national syndromic case reporting data resulted in poor identifiability of most parameters during model calibration. Data to inform sexual behaviour and care-seeking parameters for the general population relied on self-reporting in household surveys, which is subject to reporting biases,[351] while inputs for FSW were drawn from urban cohort studies. Behavioural, care-seeking, and population size parameters for CFSW and intermediate risk groups were informed by sparse data or based on assumptions.

Estimates of QALY losses were also affected by data limitations and input assumptions. For men and non-pregnant women, I relied on sequelae probabilities derived from chlamydia studies, which may misrepresent risks associated with gonorrhoea. Several pregnancy-related outcomes, such as premature rupture of membranes, preterm birth, and neonatal death, were not included due to insufficient evidence, likely underestimating the full burden of morbidity among pregnant women and their infants.[71,369] All parameters for QALY loss estimation were treated as fixed values, without incorporating uncertainty, limiting assessment of how sensitive target population prioritisation was to variation in input assumptions. The inclusion of stillbirth in QALY estimation varies across studies and remains an area of methodological debate. I assigned a utility loss equivalent to neonatal death,[424] although some studies exclude stillbirths or apply lower utility losses to reflect the gradual accrual of life potential in early childhood.[418,424] My sensitivity analyses using these approaches did not however

change the prioritisation of pregnant women for morbidity reduction.

Structural assumptions in the transmission model may have influenced intervention impact estimates. I assumed proportionate sexual mixing, which may overestimate transmission in lower-risk groups and underestimate infection concentration in higher-risk groups,[425] potentially under-representing differences in intervention impact and prioritisation between target populations. The model also excluded acquired or partial immunity after gonorrhoea infection due to inconsistent evidence,[45,52,54] which allowed rapid reinfection and may have overestimated the impact of POCT in averting transmission.[52] I assumed universal access to effective treatment following diagnosis through syndromic management or POCT, which may not reflect real-world constraints such as stock-outs. Treatment under syndromic management was modelled as dependent on its diagnostic sensitivity, but in practice, clinicians may overtreat or individuals may access antibiotics without prescription.[426] These simplifications may have overestimated the incremental benefit of POCT-based diagnostic testing over syndromic management. Furthermore, I did not distinguish between healthcare sectors, where differences in care-seeking, syndromic management quality, and antibiotic availability could have influenced intervention impact estimates. I assumed stable gonorrhoea prevalence at baseline, once equilibrium was reached, which may have oversimplified longer-term trends when evaluating intervention impact. However, estimates suggest relatively stable prevalence over time in Eastern Africa (as shown in Chapter 2). In addition, I assumed a six-year POCT implementation period, which may have underestimated cumulative impact, particularly the longer-term indirect effects of reduced transmission. My model did not differentiate prevalence by age among women, due to limited data and mixed evidence in other contexts.[230,317,318] However, the impact of targeting AGYW may have been underestimated if their prevalence is higher than that of older women in Kenya. MSM were not included in the model, despite their high gonorrhoea prevalence.[427] While my findings among other high-risk populations support prioritising MSM for POCT, future analyses should directly assess impact in this group. Finally, I did not consider behavioural and psychosocial dimensions of testing, such as the effects on partner notification and treatment, care-seeking, and stigma, which may influence POCT uptake and consequent impact.

In conclusion, I demonstrated that gonorrhoea POCTs meeting the WHO Optimal TPP would be most impactful in reducing both morbidity and infections if deployed for diagnostic testing rather than screening when availability is limited in Kenya. This supports WHO guidance to strengthen aetiologic diagnosis within syndromic management.[117] However, deployment of POCTs would be more

efficient for informing treatment of urethral rather than vaginal discharge, which was unexpected given the poorer performance of syndromic management for vaginal discharge in diagnosing and treating gonorrhoea. A limited POCT supply should be prioritised for diagnostic testing among pregnant women to maximise reductions in total QALY losses, or among higher-risk populations, such as FSW or their clients, to maximise reductions in population-level incidence. As POCTs approach market introduction, particularly those with multiplex or resistance detection capabilities, prioritisation strategies should be re-evaluated to account for diagnostic performance, consider optimal combined allocation across testing strategies and target populations, and align with evolving programmatic goals. Implementing POCTs would not only reduce the burden of disease, but could also address critical gaps in aetiologic prevalence data, strengthening efforts to improve STI surveillance.

Chapter 7

Discussion

This chapter summarises key findings from my research, reflects on their broader implications, and identifies priorities for extending my work.

7.1 Summary of findings

In this thesis, I aimed to use available data and mathematical modelling to generate evidence that strengthens STI surveillance and control in SSA. This was largely successful: I updated regional prevalence estimates for curable STIs, addressing key limitations in existing global estimates; characterised the aetiologic profiles of common symptoms to inform regional treatment guidelines; and prioritised strategies for future POCT deployment. However, substantial gaps and inconsistencies in available surveillance data made it challenging to triangulate between data sources or generate spatially granular prevalence estimates, ultimately constraining the depth of evidence available to guide STI control strategies.

In Chapter 1, I reviewed the epidemiology, natural history, and transmission dynamics of five major STIs in SSA. I summarised published evidence on current prevention, diagnostic, and treatment strategies, and highlighted persistent challenges to STI control in the region, including widespread asymptomatic infection, limited diagnostic capacity, and weak surveillance systems. Review of previous mathematical modelling approaches to evaluate diagnostic testing and screening interventions identified considerable evidence from high-income settings, which may not adequately capture the epidemiologic patterns, care-seeking behaviours, or health system constraints typical of SSA.

In Chapter 2, I estimated the prevalence of chlamydia, gonorrhoea, and trichomoniasis among the

general population in SSA from 2000 to 2024 using a systematic review and meta-regressions, which updated previous estimates by incorporating variation by sex, sub-region, and over time. All three infections were found to be highly prevalent. Geographic variation was substantial; prevalence of chlamydia and gonorrhoea was highest in Southern Africa, followed by Eastern Africa and then Western and Central Africa, mirroring the distribution of HIV in the region. Trichomoniasis prevalence, by contrast, was relatively consistent across sub-regions for females and highest in Eastern Africa for males. Over time, chlamydia prevalence has increased considerably in Eastern and Southern Africa, which is particularly concerning given its asymptomatic nature and potential severe sequelae if untreated. Gonorrhoea prevalence remained stable over time; although it is well addressed via the urethral discharge syndrome algorithm among males, it frequently presents asymptomatically among females and remains untreated. Trichomoniasis prevalence declined in Southern Africa, though this trend may reflect changes in diagnostic methods over time rather than true reductions in burden. Across all three infections, prevalence was higher among females than males, with the smallest sex difference observed for gonorrhoea and the largest for trichomoniasis, reflecting a combination of biological, behavioural, and structural factors. The sex-prevalence ratios for SSA differed from existing global ratios, and should be used in future burden estimates to more accurately reflect underlying regional epidemiology.

In Chapter 3, I shifted focus from the general population to individuals with genital symptoms, examining the underlying aetiologies of vaginal discharge, urethral discharge, and genital ulcer in SSA from 1970 to 2023 using a systematic review and meta-regressions. Most symptomatic cases were attributable to pathogens targeted in current WHO global syndromic management guidelines, though a substantial proportion - particularly for vaginal discharge and genital ulcer - lacked an identifiable cause. Vaginal discharge was most frequently caused by candidiasis, bacterial vaginosis, and trichomoniasis, while chlamydia and gonorrhoea together accounted for less than 25% of cases. Urethral discharge was primarily due to gonorrhoea, followed by chlamydia, with aetiologic proportions remaining stable over time for both pathogens. Genital ulcer aetiology shifted markedly over time; the leading cause shifted from chancroid in 1990 to HSV-2 by 2010. The substantial decline in chancroid, a historically highly prevalent infection, provides evidence of the successful scale-up of syndromic management in SSA, where widespread empiric antibiotic treatment has been particularly effective against a typically symptomatic and drug-susceptible pathogen. The distribution of pathogens causing each symptom was broadly consistent across sub-regions and population strata. Overall, these findings support the continued applicability of WHO-recommended syndromic algorithms in SSA, while reinforcing the need for regular national aetiologic assessments to ensure these algorithms remain aligned with evolving

epidemiology.

Chapters 2 and 3 identified substantial gaps in available aetiologic data. In response, Chapter 4 evaluated whether self-reported genital symptoms from population-representative surveys, which have been underutilised for STI surveillance, could support quantifying relative STI burdens and trends in SSA. Drawing on nationally-representative DHS and AIS between 2000 and 2024, I used regression models to examine geographic and temporal patterns in symptom history and treatment-seeking behaviours. Reported symptom prevalence varied across countries and sub-regions and increased markedly over time, but neither pattern aligned with estimates from studies directly measuring aetiologic prevalence in the typically non-representative samples analysed in Chapter 2. Vaginal discharge and genital ulcer were most frequently reported by females in Western Africa, despite the low prevalence of their primary aetiologies in that sub-region. In contrast, reported urethral discharge was more uniformly distributed across the region and aligned more closely with gonorrhoea prevalence patterns among males. Less than 60% of symptomatic individuals reported seeking treatment, with coverage highest in Southern Africa. Reported treatment-seeking was often fragmented, spanning public, private, and informal providers, with notable increases in pharmacy use over time, particularly in Western and Central Africa. These findings suggest that self-reported symptoms may be more informative for monitoring patterns of care-seeking and health system engagement than for estimating aetiologic STI burden.

In Chapter 5, I further evaluated symptomatic STI surveillance by comparing self-reported genital symptoms and treatment-seeking from population-representative surveys between 2000 and 2015 with syndromic case reports from health facility surveillance between 2014 and 2021 in Malawi. Using descriptive and regression analyses, I examined the distribution of vaginal discharge, urethral discharge, and genital ulcer across both data sources. Survey-reported symptoms and facility-recorded cases had similar spatial and demographic patterns, indicating a higher burden of symptoms among women, individuals above 25 years, and in Southern Malawi. Specifically, self-reported genital ulcer in surveys was highest among women 35-39 years old and in the Southern region, aligning with prior studies on the prevalence of HSV-2 and HIV. However, the magnitude and relative distribution of symptoms differed between data sources, which could affect how programme priorities are determined. Genital ulcer and vaginal discharge were the most commonly reported symptoms in surveys, while facility case diagnoses were highest for vaginal and urethral discharge syndromes, with much lower rates of genital ulcer. These discrepancies may partially reflect different care-seeking patterns, such as greater reliance

on private or traditional providers for genital ulcer management. Facility case diagnoses increased between 2015 and 2020, whereas surveys indicated no significant change in symptom reporting and a decline in treatment-seeking between 2000 and 2015. My findings indicate that neither data source alone is sufficient for surveillance. Aetiologic studies are needed to reconcile the discrepancies and inform programmatic responses, particularly in settings with fragmented care-seeking and incomplete case reporting.

Finally, in Chapter 6, I used mathematical modelling to assess how a limited supply of gonorrhoea POCTs could be optimally allocated across testing strategies and target populations in Kenya. POCTs offer an opportunity to improve case detection and treatment outcomes, given the limitations of syndromic management identified in earlier chapters, but widespread deployment is likely to be constrained by supply and cost. I found that diagnostic testing among symptomatic populations was a more efficient use of POCTs than asymptomatic screening. Testing FSW or their clients achieved the largest reduction in total incident infections, while testing pregnant women achieved the largest gains in total QALYs, primarily by preventing adverse neonatal outcomes. Diagnostic testing was more impactful among male than female populations, despite the already high sensitivity of syndromic management for urethral discharge compared to POCTs for detecting gonorrhoea. This reflects the lower proportion of syndromic cases attributed to gonorrhoea in females, as well as the higher likelihood of symptom presentation and care-seeking among infected males. My findings support the WHO's recommendation to incorporate aetiologic diagnosis within syndromic management, and further suggest that POCTs may be more efficiently deployed for diagnosing urethral rather than vaginal discharge syndrome.

7.2 Implications and limitations

Weak surveillance undermines burden estimation and progress monitoring

Challenges with STI surveillance have long been recognised as a barrier to effective control in SSA.[224] My work demonstrates the extent to which surveillance remains constrained across the region. Routine syndromic case reporting data were not widely accessible or included in publicly available national STI strategic plans, treatment guidelines, or programme reports that I reviewed during grey literature searches (Chapter 3). South Africa and Malawi stand out as key exceptions. In South Africa, case reports have been used to estimate syndromic case incidence,[239,428] assess health facility reporting compliance,[210,239,428] and inform national gonorrhoea prevalence estimates.[239] In Malawi, syndromic

case reports are publicly accessible via an online dashboard,[429] and are summarised in quarterly programme reports.[430] My analysis in collaboration with the Directorate of HIV & AIDS, STIs, and Viral Hepatitis within the Malawi Ministry of Health provided a more in-depth examination of these data than is routinely conducted (Chapter 5). To my knowledge, these are the only two countries in SSA where national routine syndromic case reports have been systematically analysed and disseminated in the peer-reviewed literature.

Infection prevalence studies and aetiologic assessments are geographically and demographically limited across the region. I identified prevalence studies of curable STIs from only 58% of countries between 2000 and 2024 (Chapter 2), and aetiologic assessments from 67% of countries between 1969 and 2022 (Chapter 3). Studies seldom used population-representative samples or included men, and were often geographically concentrated. South Africa was the only country identified with regular and coordinated aetiologic surveillance among symptomatic individuals attending sentinel clinics. Many studies, particularly older ones, relied on diagnostic tests with poor sensitivity and specificity, requiring statistical adjustments that introduced additional uncertainty. Analyses of these data were further constrained by inconsistent reporting of key covariates such as age, HIV status, or urban and rural location.

These surveillance gaps undermine the ability of national programmes to adequately target interventions, allocate resources efficiently, and monitor progress toward WHO 2030 STI incidence reduction targets.[222] The scarcity of aetiologic prevalence data impedes generating spatially granular burden estimates and obscures shifts in the distribution of pathogens causing each syndrome, potentially leading to ineffective treatment or overtreatment.[116] Inadequate routine syndromic surveillance delays timely detection of emerging epidemiologic patterns, such as localised incidence increases, demographic shifts in infection burden, or declining treatment efficacy that may warrant AMR investigation.[222] As a result, strategic decisions are often based on outdated or incomplete information, or rely on global or regional averages that do not necessarily reflect local epidemiologic realities.

Addressing these challenges requires coordinated regional efforts and sustained investment, which is challenging amidst disruptions to US foreign aid.[221] The WHO Global Health Sector Strategy recommends strengthening STI health information systems, expanding data collection and analysis, and capturing disaggregated data to better characterise infection patterns and monitor equity in service access and health outcomes.[1] Investing in laboratory capacity, and POCTs as they become available, is critical for strengthening aetiologic surveillance.[1] In settings where pharmacies and the private sector

are major providers of STI services (Chapter 4), countries should also establish systems to promote integrated multi-sectoral reporting, including provider registries, standardised reporting tools, and mechanisms to ensure data quality, confidentiality, and coordination.[1] Without such investments, STI control efforts will continue to be undermined.

Symptom reporting is not reliable for infection surveillance

Given limitations in the coverage and representativeness of aetiologic STI surveillance across SSA, I explored the potential of population-representative surveys that collect data on self-reported STI symptoms and treatment-seeking from nationally-representative samples of adults as a complementary data source to quantify relative STI burdens and trends. However, self-reported symptom data proved unreliable for infection surveillance. Reported symptom prevalence varied between countries (Chapter 4) and sub-nationally within Malawi (Chapter 5), but did not correlate with either aetiologic prevalence estimates (Chapter 4) or syndromic case reports (Chapter 5). High rates of asymptomatic infection, poor symptom recognition, and the non-specific nature of genital symptoms are recognised limitations of these self-reported data, which my analyses confirmed to substantially limit the utility of symptom reporting for infection burden estimation. However, my ability to validate symptom reporting was constrained by weaknesses in the comparator data sources for each analysis. Aetiologic prevalence studies were methodologically heterogeneous, geographically sparse, and seldom included men, among whom symptom-based measures might have been more reliable. Syndromic case reporting captured only public sector diagnoses, lacked age and sex stratification, and could not distinguish repeat diagnoses for the same individual.

Despite these challenges, household surveys remain critical for understanding health system engagement and treatment for STIs. Reported treatment-seeking among individuals with symptoms can highlight service access gaps, such as low public sector utilisation or reliance on informal providers (Chapter 4), and broader inequities in treatment by gender, age, or socioeconomic status (Chapters 4 and 5). No other surveillance data provide comparable population-level insights on care-seeking for symptomatic STIs. Symptom reporting may also help guide targeted surveillance by identifying populations or geographies with unusually high symptom prevalence that warrant aetiologic investigation, particularly in resource-constrained settings. The recent termination of the DHS Programme is therefore deeply concerning, given its potential to improve intervention targeting and coverage for STIs, along with its broader role in monitoring health and development indicators to inform policy, investment, and progress tracking towards the Sustainable Development Goals.[431]

Epidemiologic heterogeneity calls for more targeted programme strategies

I identified considerable geographic, temporal, and demographic heterogeneity in the STI burden across SSA, despite the limitations of available surveillance data. Curable STI prevalence varied across sub-regions in SSA (Chapter 2), and syndromic diagnoses varied sub-nationally within Malawi (Chapter 5), with geographic patterns in STI burden closely mirroring those of HIV (Chapters 2 and 5). Over time, chlamydia prevalence increased in Eastern and Southern Africa, while trichomoniasis declined in Southern Africa (Chapter 2). The aetiologies of common STI symptoms also evolved substantially (Chapter 3). Women consistently had higher STI prevalence than men (Chapter 2), with symptomatic AGYW and people living with HIV at higher risk of diagnosis with several infections compared to older women and individuals without HIV, respectively (Chapter 3). Gonorrhoea prevalence and associated morbidity also differed across population groups in Kenya (Chapter 6). Moreover, access to treatment among individuals with symptoms was highly variable across the region and has changed over time (Chapter 4).

However, existing STI programmes have largely failed to account for this epidemiologic heterogeneity. Regional and national responses seldom incorporate geographic or population-specific prioritisation,[1,139,219] resulting in largely uniform service delivery models and programmatic approaches. Syndromic management algorithms have not changed over the past two decades, with WHO flowcharts from 2003 updated only in 2021.[117] STI case detection remains predominantly passive, relying on symptomatic individuals presenting to health facilities for care, with minimal active case-finding through community-based or peer-led outreach.[432] Although provider-initiated symptom screening is recommended within HIV-related services, this approach does not address the high burden of asymptomatic infections among key populations (e.g., cervical infections in FSW, rectal infections in MSM),[1,117] limiting its effectiveness in curbing transmission among those at greatest risk.

The HIV response offers a strong precedent for addressing epidemic heterogeneity. The UNAIDS principle of "know your epidemic, know your response" has been central to adapting programming to align with local epidemiology and driving declines in HIV incidence across much of SSA.[433,434] Progress towards national and regional targets is monitored by Ministries of Health and UNAIDS, underpinned by coordinated surveillance and modelling processes that also translate national goals into sub-national targets.[435] These targets guide district-level planning, resource allocation, and service delivery, enabling countries to respond more dynamically to their evolving epidemics. Differentiated service delivery models, community-led approaches, and targeted interventions for key populations

have been introduced to better address the needs of diverse populations.[434] Although considerable progress has been made, persistent challenges in data quality, programme coordination, and reaching underserved populations must not be overlooked.[434]

In line with WHO guidance, STI control efforts should better leverage available surveillance data to set and monitor targets, prioritise high-burden populations and geographies, and adapt to changing epidemic patterns.[1] While resource constraints limit the scale of programme transformation, strategic prioritisation would enable more effective and equitable STI control responses across the region.

Renewed innovation offers opportunities to strengthen control

Strategies for STI prevention and management have remained largely unchanged for much of the last two decades, limiting the effectiveness of control efforts in SSA. However, in high-income countries, increasing STI incidence, resurgent congenital syphilis, and growing antimicrobial resistance have driven renewed global attention to STI control.[436] This momentum has catalysed new research investment, policy initiatives, and innovation aimed at addressing long-standing gaps.[437,438] As part of this effort, WHO has outlined a global roadmap to accelerate vaccine development for gonorrhoea, chlamydia, syphilis, and HSV, including preferred product characteristics to guide research and development.[439]

Several promising interventions are currently being evaluated. These include doxycycline as post- and pre-exposure prophylaxis (DoxyPEP and DoxyPrEP) to prevent bacterial STI acquisition, repurposing the meningococcal group B vaccine (4CMenB) to provide partial protection against gonorrhoea, and advancements in POCT technologies, such as the recently approved self-test for chlamydia, gonorrhoea, and trichomoniasis in women. While these innovations show promise, most evidence to date comes from high-income settings. For example, DoxyPEP has demonstrated high efficacy among MSM and transgender women in the United States and France,[440–443] with studies reporting moderate acceptability of DoxyPEP use among these populations.[444–446] However, a DoxyPEP trial among cisgender women in Kenya found no significant effect on STI incidence, likely due to challenges with adherence,[447] emphasising the influence of contextual factors on intervention effectiveness. 4CMenB has demonstrated partial protection against gonorrhoea in observational studies across the US, Australia, and Europe,[448] and modelling work suggests that 4CMenB vaccination among MSM or adolescents could reduce gonorrhoea incidence in England.[449–451] However, its potential impact remains largely unexplored in SSA. More encouragingly, several studies have assessed the feasibility, acceptability, or uptake of either self-testing or POCTs for STIs in SSA, demonstrating demand across diverse

settings.[452–460]

Given ongoing evidence gaps, my research provides insight into evaluating and optimising the deployment of emerging STI interventions in SSA. First, even incremental changes, such as introducing targeted gonorrhoea POCTs within existing syndromic management frameworks, can improve treatment coverage and reduce STI burden (Chapter 6). Second, resource constraints will require prioritising between interventions, target populations, and service delivery settings - factors I showed to substantially influence population-level impact (Chapter 6). Third, given fragmented care pathways across public, private, and informal sectors in most countries (Chapter 4), successful implementation will likely require multi-sectoral delivery to maximise coverage and effectiveness. Finally, equitable access must be central to implementation strategies, as care-seeking behaviour varies across socio-demographic groups and geographies (Chapters 4 and 5).

Renewed innovation offers a critical opportunity to strengthen STI control in SSA. Realising this potential will require evidence-based strategic prioritisation, and a commitment to ensuring that new advances do not deepen global inequities in STI care.

7.3 Future directions

Future work should support a more strategic and coordinated regional response toward the WHO 2030 STI incidence reduction targets, while also laying the foundation for long-term improvements in STI control beyond 2030. Building on the findings of this thesis, there are two key priorities in SSA: (1) strengthen surveillance systems to generate higher-quality and more actionable data, and (2) optimise the implementation of emerging diagnostic and preventive interventions.

Strengthen surveillance for more effective national and regional control

Further progress in burden estimation, national planning, and timely programmatic response requires strengthening and expanding data collection to meet these explicit surveillance goals, while remaining feasible under constrained resources. Below, I outline interrelated priorities to improve STI surveillance in SSA. These draw on the WHO STI surveillance framework,[222] but are adapted to the data limitations and operational challenges identified in this thesis.

- **Establish sentinel sites for enhanced case reporting:** Countries should establish sentinel surveillance sites at high-volume health facilities selected to ensure broad geographic coverage and

representation beyond capital cities. These sites should receive targeted investment to support standardised and complete case reporting through staff training and routine quality assurance. Aetiologic assessments among symptomatic clients should be periodically conducted to detect shifts in pathogen distributions and inform treatment protocols. Sentinel sites could also support other research activities, including AMR monitoring or diagnostic intervention studies.

- **Adapt existing surveys to improve burden estimates:** National household surveys (e.g. DHS, AIS) and key population surveys (e.g. Integrated Biobehavioural Surveys) can be adapted to better support STI surveillance. Symptom-related questions should be refined to improve specificity and reduce misclassification, such as by collecting data on additional symptoms (e.g. dysuria, itching, and pelvic pain) and on symptom timing, severity, recurrence, and duration. Aligning symptom questions with syndromic case definitions may also improve consistency with facility case reporting. Surveys should also incorporate nested sub-studies with aetiologic testing for key pathogens, particularly in countries where prevalence data are currently sparse. This would yield more representative prevalence estimates, and support evaluations of asymptomatic infection and the reliability of symptom-based indicators.

- **Collect and report stratified data to monitor disparities:** All surveillance modalities should collect and report outcomes stratified by key variables such as sex, age, HIV status, and geographic location. Prevalence surveys should also include social and behavioural variables such as income, education, urban or rural residence, condom use, and number of sexual partners, to enable more nuanced analyses of infection risk. Many of these variables are already included in existing biobehavioural surveys and, if combined with STI testing, could help identify disparities in burden to inform more targeted responses.

- **Design surveillance to support triangulation and modelling:** Surveillance activities should be designed to enable integration across data sources. This requires consistent case definitions, reporting formats, and standardised stratification categories across syndromic, aetiologic, and behavioural data sources, as well as alignment in the coverage of population groups and geographies. Clear documentation of methods, sampling frames, and data collection periods is essential to support comparability. These design features would facilitate data triangulation, strengthen the use of routine data for epidemiological modelling, and support prevalence estimation in settings or populations with limited direct aetiologic data. Modelling should be used iteratively to prioritise future surveillance efforts by identifying settings or sub-populations where missing or inconsistent

data contribute to the greatest uncertainty.

- **Promote routine data use at all levels:** Surveillance data are often underutilised due to weak integration into routine programme planning. National systems should prioritise regular dissemination of data summaries tailored to clinical staff, district health officers, and national programme managers. Embedding these feedback loops would not only support timely responses to changing epidemiology, but also incentivise more complete and accurate reporting. Investments in local capacity through training and technical assistance would support embedding data-driven decision-making in STI programmes.

Optimise intervention strategies across pathogens, populations, and settings

Alongside strengthening surveillance, future research should focus on optimising the implementation of emerging STI interventions. The gonorrhoea transmission model developed in this thesis can be extended to inform strategic prioritisation of intervention packages across populations and delivery settings. Below, I outline key questions to guide this work.

- **How should POCTs be allocated among multiple target populations in combination to maximise population-level impact?** As POCT availability increases, several allocation strategies could be explored: distributing tests among multiple high-risk groups (e.g., FSW and their clients), retesting within a single high-risk group, extending testing to include pregnant women, men, or both, or implementing mixed strategies combining these approaches.
- **How does considering MSM as a target population affect POCT allocation strategies?** Given the high incidence of gonorrhoea but relatively lower associated morbidity among MSM, explicitly modelling this group may shift prioritisation strategies, particularly when balancing infection prevention against gains in health outcomes.
- **How should diagnostic and preventive interventions be prioritised individually and in combination to maximise population-level impact?** The model could be extended to incorporate vaccination (e.g., 4CMenB) and biomedical prophylaxis (e.g., DoxyPEP) to assess their individual and combined impact relative to POCTs. This could help inform optimal sequencing, integration, and targeting strategies.
- **Does calibrating the model to a different epidemiologic context influence intervention prioritisation across target populations?** Calibrating the model to a setting with more

extensive aetiologic and syndromic surveillance data, such as South Africa, may help reduce parameter uncertainty and improve the precision of projected intervention impacts. It would also enable assessment of whether variations in infection burden and service access across target populations alter prioritisation strategies in different epidemiologic contexts.

- **How should intervention strategies be prioritised when considering multiple STIs, such as gonorrhoea and chlamydia?** The model could be expanded to include chlamydia, which shares a similar natural history with gonorrhoea. This would enable the evaluation of dual POCTs, which may be particularly important for increasing the proportion of symptomatic women with a diagnosed aetiology. Further expansions could incorporate biomedical prevention strategies to support integrated optimisation of diagnostic and preventive interventions across pathogens and populations.

Together, addressing these questions would enable more strategic deployment of diagnostic and preventive interventions as they become more widely available, and help to optimise the use of limited resources. In doing so, this work would support short-term improvements while laying the foundation for sustainable, longer-term programmes to enhance STI control across SSA.

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Appendix A

Supplement to Chapter 2

A.1 Supplementary methods

A.1.1 Search strategy and data extraction

Table A1: Search strategy for systematic review

EMBASE: Search conducted 17 September 2024 with 3794 articles retrieved.
STI domain: exp gonorrhea/ or exp Neisseria gonorrhoeae/ or exp Chlamydia trachomatis/ or exp Chlamydia trachomatis infection/ or exp vaginal trichomoniasis/ or exp trichomoniasis/ or exp Trichomonas vaginalis/ or (gonorrhea* or gonorrhoea* or gonorrhoeae* or chlamydia* or trichomonas or trichomoniasis).ab,ti,kw.
AND sub-Saharan Africa domain: exp Africa south of the Sahara/ or exp africa, eastern/ or exp africa, western/ or exp africa, central/ or exp africa, southern/ or (Africa or Angola or Benin or Botswana or Motswana or Batswana or Burkina or Burundi or "Cabo Verde" or "Cape verde" or Cameroon or "Central African Republic" or "Republique centrafricaine" or Chad or Comoros or Congo or "Democratic Republic of Congo" or "Republique democratique du Congo" or DRC or "Cote d'Ivoire" or "Ivory Coast" or Djibouti or Guinea or Eritrea or Erythree or Ethiopia or Gabon or Gambia or Ghana or Guinea or "Equatorial Guinea" or "Guinee Equatoriale" or "Equatoguinean" or "Guinea-Bissau" or Kenya or Lesotho or Basotho or Liberia or Madagascar or Malawi or Mali or Mauritania or Mozambique or Namibia or Niger or Nigeria or Rwanda or Rouanda or Ruanda or "Sao Tome" or Senegal or Seychelles or "Sierra Leone" or Somalia or Somali or "South Africa" or "Afrique du Sud" or "South Sudan" or "Soudan de sud" or Sudan or Swaziland or eSwatini or Tanzania or Togo or Uganda or Ouganda or Zambia or Zimbabwe).mp.
AND Limit publication year = "January 1, 2000 - Current"
MEDLINE: Search conducted 17 September 2024 with 2231 articles retrieved.
STI domain: exp Neisseria gonorrhoeae/ or exp Gonorrhea/ or exp Chlamydia Infections/ or exp Chlamydia/ or exp Chlamydia trachomatis/ or exp Trichomonas vaginalis/ or exp Trichomonas Infections/ or exp Trichomonas/ or exp Trichomonas Vaginitis/ or (gonorrhea* or gonorrhoea* or gonorrhoeae* or chlamydia* or trichomonas* or trichomoniasis*).ab,ti,kw.
AND sub-Saharan Africa domain: exp Africa, Western/ or exp Africa, Central/ or exp "Africa South of the Sahara"/ or exp Africa/ or exp Africa, Eastern/ or exp Africa, Southern/ or (Africa or Angola or Benin or Botswana or Motswana or Batswana or Burkina or Burundi or "Cabo Verde" or "Cape verde" or Cameroon or "Central African Republic" or "Republique centrafricaine" or Chad or Comoros or Congo or "Democratic Republic of Congo" or "Republique democratique du Congo" or DRC or "Cote d'Ivoire" or "Ivory Coast" or Djibouti or Guinea or Eritrea or Erythree or Ethiopia or Gabon or Gambia or Ghana or Guinea or "Equatorial Guinea" or "Guinee Equatoriale" or "Equatoguinean" or "Guinea-Bissau" or Kenya or Lesotho or Basotho or Liberia or Madagascar or Malawi or Mali or Mauritania or Mozambique or Namibia or Niger or Nigeria or Rwanda or Rouanda or Ruanda or "Sao Tome" or Senegal or Seychelles or "Sierra Leone" or Somalia or Somali or "South Africa" or "Afrique du Sud" or "South Sudan" or "Soudan de sud" or Sudan or Swaziland or eSwatini or Tanzania or Togo or Uganda or Ouganda or Zambia or Zimbabwe).mp.
AND Limit publication year = "January 1, 2000 - Current"
Global Health: Search conducted 17 September 2024 with 2364 articles retrieved.
STI domain: exp gonorrhoea/ or exp Neisseria gonorrhoeae/ or exp Chlamydia trachomatis/ or exp Chlamydia/ or exp trichomoniasis/ or exp Trichomonas vaginalis/ or (gonorrhea* or gonorrhoea* or gonorrhoeae* or chlamydia* or trichomonas or trichomoniasis).ab,ti,mp.

AND sub-Saharan Africa domain: exp "Africa South of Sahara"/ or exp East Africa/ or exp Africa/ or exp Central Africa/ or exp Southern Africa/ or exp West Africa/ or (Africa or Angola or Benin or Botswana or Motswana or Batswana or Burkina or Burundi or "Cabo Verde" or "Cape verde" or Cameroon or "Central African Republic" or "Republique centrafricaine" or Chad or Comoros or Congo or "Democratic Republic of Congo" or "Republique democratique du Congo" or DRC or "Cote d'Ivoire" or "Ivory Coast" or Djibouti or Guinea or Eritrea or Erythree or Ethiopia or Gabon or Gambia or Ghana or Guinea or "Equatorial Guinea" or "Guinee Equatoriale" or "Equatoguinean" or "Guinea-Bissau" or Kenya or Lesotho or Basotho or Liberia or Madagascar or Malawi or Mali or Mauritania or Mozambique or Namibia or Niger or Nigeria or Rwanda or Rouanda or Ruanda or "Sao Tome" or Senegal or Seychelles or "Sierra Leone" or Somalia or Somali or "South Africa" or "Afrique du Sud" or "South Sudan" or "Soudan de sud" or Sudan or Swaziland or eSwatini or Tanzania or Togo or Uganda or Ouganda or Zambia or Zimbabwe).mp.

AND Limit publication year = "January 1, 2000 - Current"

PubMed: Search conducted 17 September 2024 with 2663 articles retrieved.

STI domain: "Neisseria gonorrhoeae"[MeSH Terms] or "Gonorrhea"[MeSH Terms] or "Chlamydia Infections"[MeSH Terms] or "Chlamydia"[MeSH Terms] or "Chlamydia trachomatis"[MeSH Terms] or "Trichomonas vaginalis"[MeSH Terms] or "Trichomonas Infections"[MeSH Terms] or "Trichomonas"[MeSH Terms] or "Trichomonas Vaginitis"[MeSH Terms] or gonorrhea*[Title/Abstract] OR gonorrhoea*[Title/Abstract] OR gonorrhoeae*[Title/Abstract] OR chlamydia*[Title/Abstract] OR trichomonas[Title/Abstract] OR trichomoniasis[Title/Abstract]

AND sub-Saharan Africa domain: "Africa south of the Sahara"[MeSH Terms] or "africa, eastern"[MeSH Terms] or "africa, western"[MeSH Terms] or "africa, central"[MeSH Terms] or "africa, southern"[MeSH Terms] or "Africa"[All fields] or "Angola"[All fields] or "Benin"[All fields] or "Botswana"[All fields] or "Motswana"[All fields] or "Batswana"[All fields] or "Burkina"[All fields] or "Burundi"[All fields] or "Cabo Verde"[All fields] or "Cape verde"[All fields] or "Cameroon"[All fields] or "Central African Republic"[All fields] or "Republique centrafricaine"[All fields] or "Chad"[All fields] or "Comoros"[All fields] or "Congo"[All fields] or "Democratic Republic of Congo"[All fields] or "Republique democratique du Congo"[All fields] or "DRC"[All fields] or "Cote d'Ivoire"[All fields] or "Ivory Coast"[All fields] or "Djibouti"[All fields] or "Guinea"[All fields] or "Eritrea"[All fields] or "Ethiopia"[All fields] or "Gabon"[All fields] or "Gambia"[All fields] or "Ghana"[All fields] or "Guinea"[All fields] or "Equatorial Guinea"[All fields] or "Equatoguinean"[All fields] or "Guinea-Bissau"[All fields] or "Kenya"[All fields] or "Lesotho"[All fields] or "Basotho"[All fields] or "Liberia"[All fields] or "Madagascar"[All fields] or "Malawi"[All fields] or "Mali"[All fields] or "Mauritania"[All fields] or "Mozambique"[All fields] or "Namibia"[All fields] or "Niger"[All fields] or "Nigeria"[All fields] or "Rwanda"[All fields] or "Rouanda"[All fields] or "Ruanda"[All fields] or "Sao Tome"[All fields] or "Senegal"[All fields] or "Seychelles"[All fields] or "Sierra Leone"[All fields] or "Somalia"[All fields] or "Somali"[All fields] or "South Africa"[All fields] or "Afrique du Sud"[All fields] or "South Sudan"[All fields] or "Sudan"[All fields] or "Swaziland"[All fields] or "eSwatini"[All fields] or "Tanzania"[All fields] or "Togo"[All fields] or "Uganda"[All fields] or "Ouganda"[All fields] or "Zambia"[All fields] or "Zimbabwe"[All fields]

AND Limit publication year = "January 1, 2000 - Current"

African Index Medicus: Search conducted 17 September 2024 with 84 articles retrieved.

STI domain: mh:("Chlamydia Infection" or "Chlamydia trachomatis" OR "Chlamydia", or "Gonorrhoea" OR "Neisseria gonorrhoeae" OR "Trichomonas" OR "Trichomoniasis" OR "Trichomonas vaginalis" OR "Trichomonas infections" OR "Trichomonas infection") or tw:(gonorrhea* or gonorrhoea* or gonorrhoeae* or chlamydia* or trichomonas or trichomoniasis)
AND Limit publication year = "January 1, 2000 - Current"

Table A2: Classification of included countries by sub-region in SSA

Sub-region	Country
Central Africa	Angola, Cameroon, Central African Republic, Chad, Congo, Democratic Republic of Congo, Equatorial Guinea, Gabon, Sao Tome and Principe
Western Africa	Benin, Burkina Faso, Cabo Verde, Cote d'Ivoire, Gambia, Ghana, Guinea-Bissau, Guinea, Liberia, Mali, Mauritania, Niger, Nigeria, Senegal, Sierra Leone, Togo
Eastern Africa	Burundi, Comoros, Djibouti, Eritrea, Ethiopia, Kenya, Madagascar, Malawi, Mauritius, Mozambique, Rwanda, Seychelles, Somalia, South Sudan, Uganda, United Republic of Tanzania, Zambia, Zimbabwe
Southern Africa	Botswana, Eswatini, Lesotho, Namibia, South Africa

Countries classified according to UN M49 Standard.[282]

Table A3: Variables extracted from included studies

Category	Variable
Study characteristics	• Authors • Publication title • Publication year • Dates of data collection • Country of study • Sub-national region or city of study • Study name (as applicable)
Participant characteristics	• Study population category • Study population age (mean, SD, median, IQR, range) • Study population HIV status • Study population HIV prevalence
Diagnostic methodology	• Diagnostic test per infection • Diagnostic specimen per infection • Number tested per infection • Number positive per infection • Prevalence of each infection

A.1.2 Adjustment for diagnostic test performance

Diagnostic tests from included studies were classified into broad categories. Antibody tests included enzyme immunoassays or enzyme-linked immunosorbent assays. Culture included any culture test with a valid medium. NAAT included polymerase chain reaction, strand displacement assay, transcription mediated assay, ligase chain reaction, and GeneXpert. Treponemal tests for syphilis included *Treponema pallidum* Particle Agglutination Assay, *Treponema pallidum* Hemagglutination Assay, Fluorescent Treponemal Antibody Absorption, Microhemagglutination Assay for *Treponema pallidum*, and point of care rapid tests. Non-treponemal tests for syphilis included the Venereal Disease Research Laboratory test and Rapid Plasma Reagin test. Syphilis tests were categorised regardless of titre due to variations in reporting.

Point value sensitivity and specificity estimates for each diagnostic test category were compiled from literature, with priority given to characteristics published by WHO (Table A4).[145,230] For *T. vaginalis*, the accuracy of culture and wet mount using urine samples was assumed to be equivalent to that of genital fluid samples.[142,230,461] In studies using two diagnostic tests to identify a pathogen, combined sensitivity and specificity was determined: (i) if either test could be positive, the lower sensitivity and lower specificity of the two tests was used, and (ii) if both tests had to be positive, the lower sensitivity and higher specificity of the two tests was used.

To correct for imperfect diagnostic test performance, a Bayesian approach was used. The Rogan-Gladen estimator (Equation A.1) estimates true prevalence (p^{true}) of infection from observed prevalence (p^{observed}) measured by an imperfect diagnostic test.[462] However, direct application can yield negative values for p^{true} when p^{observed} is lower than the false positive probability (1 - specificity). Therefore, a Bayesian approach was used to estimate p^{true} by accounting for the observed sample size (N) and number of positive test results (n) (Equation A.2), with a uniform prior probability for p^{true} between 0 and 1 (Equation A.3).[284,285]

$$p^{\text{observed}} = p^{\text{true}} \cdot \text{sensitivity} + (1 - p^{\text{true}})(1 - \text{specificity}) \tag{A.1}$$

$$n \sim \text{binomial}(N, p^{\text{observed}}) \tag{A.2}$$

$$p^{\text{true}} \sim \text{uniform}(0, 1) \tag{A.3}$$

The model was fit using Markov Chain Monte Carlo with the NUTS (No-U-Turn Sampler) algorithm, using four chains of 2000 iterations each. Convergence was assessed qualitatively using trace plots

and quantitatively using the Gelman-Rubin statistic (R-hat). The posterior mean estimate ($\hat{p}^{\text{true}}$) and standard error (se^{true}) were used to calculate adjusted numerators (n^{adjusted}) and denominators (N^{adjusted}) for each observation (Equations A.4 and A.5), which were then used for pooling in generalized linear mixed-effects models.

$$N^{\text{adjusted}} = \frac{\hat{p}^{\text{true}}(1-\hat{p}^{\text{true}})}{(se^{\text{true}})^2} \tag{A.4}$$

$$n^{\text{adjusted}} = \hat{p}^{\text{true}} \cdot N^{\text{adjusted}} \tag{A.5}$$

Table A4: Performance of diagnostic tests

STI	Test	Specimen	N	Sensitivity (%)	Specificity (%)	Source
Female						
CT	DFA	Genital fluid	2	82.0	98.5	WHO 1999[145]
	DFA	Urine	1	82.0	98.5	WHO 1999[145]
	ELISA	Genital fluid	2	65.0	100.0	WHO 2011[230]
	NAAT	Genital fluid	105	88.6	99.6	WHO 2011[230]
	NAAT	Urine	41	87.0	99.8	WHO 2011[230]
	NAAT	Genital fluid or urine	9	87.0	99.6	Combined estimate[230,i]
	Rapid antigen test	Genital fluid	8	56.0	99.0	Grillo-Ardila 2020,[463] Zhou 2021[464]
	DFA and NAAT	genital fluid	1	82.0	99.6	Combined estimate[145, 230,ii]
NG	Culture	Genital fluid	23	75.7	100.0	WHO 2011[230]
	NAAT	Genital fluid	98	93.3	99.2	WHO 2011[230]
	NAAT	Urine	40	91.6	100.0	WHO 2011[230]
	NAAT	Genital fluid or urine	8	91.6	99.2	Combined estimate[230,i]
	Rapid antigen test	Genital fluid	1	70.0	96.0	Watchirs Smith 2013[172]
	Culture or NAAT	Genital fluid or urine	1	75.7	99.2	Combined estimate[230,i]
TV	Culture	Genital fluid	32	68.8	100.0	WHO 2011[230]
	Culture	Urine	1	68.8	100.0	Assumed equivalent to genital fluid test.
	NAAT	Genital fluid	47	95.0	98.0	WHO 2011[230]
	NAAT	Urine	7	66.9	98.3	WHO 2011[230]
	NAAT	Genital fluid or urine	1	66.9	98.0	Combined estimate[230,i]
	Rapid antigen test	Genital fluid	17	83.3	98.8	Gaydos 2017[148]
	Wet mount	Genital fluid	71	52.0	100.0	WHO 2011[230]
	Wet mount	Urine	2	52.0	100.0	Assumed equivalent to genital fluid test.
	Wet mount	Genital fluid and urine	4	52.0	100.0	Assumed equivalent to genital fluid test.
	Culture and wet mount	Genital fluid	5	52.0	100.0	Combined estimate[230,ii]
	Culture or NAAT	Genital fluid or urine	1	66.9	98.0	Combined estimate[230,i]
	Wet mount and NAAT	Genital fluid	1	52.0	100.0	Combined estimate[230,ii]
Male						
CT	NAAT	Genital fluid	1	87.5	99.2	WHO 2011[230]
	NAAT	Urine	32	87.8	99.3	WHO 2011[230]
	DFA and NAAT	Genital fluid	1	82.0	99.2	Combined estimate[145, 230,ii]
NG	Culture	Genital fluid	1	87.6	100.0	WHO 2011[230]
	NAAT	Genital fluid	1	96.1	99.0	WHO 2011[230]
	NAAT	Urine	29	80.9	99.9	WHO 2011[230]
	Culture or NAAT	Urine	1	80.9	99.9	Combined estimate[230,i]

Continued...

STI	Test	Specimen	N	Sensitivity (%)	Specificity (%)	Source
	Culture	Urine	3	87.6	100.0	WHO 2011[230]
	Culture	Genital fluid or urine	1	87.6	100.0	Combined estimate[230,i]
	NAAT	Urine	13	96.0	97.7	WHO 2011[230]
TV	Rapid antigen test	Genital fluid	1	68.5	97.4	Assumed equivalent to test for females.
	Wet mount	Urine	1	44.0	100.0	WHO 2011[230]
	Culture or NAAT	Urine	1	87.6	97.7	Combined estimate[230,i]

Combined performance characteristics estimated by: (i) using lower sensitivity and lower specificity values when at least one of two diagnostic approaches identified a positive case, and (ii) using lower sensitivity and higher specificity values when both diagnostic approaches needed to be positive. N: Number of observations. CT: *Chlamydia trachomatis*, NG: *Neisseria gonorrhoeae*, TV: *Trichomonas vaginalis*. DFA: Direct fluorescent antibody, ELISA: Enzyme-linked immunosorbent assay, NAAT: Nucleic acid amplification test.

A.1.3 Risk of bias

Table A5: Risk of bias criteria

Category	Criteria	Definition of lower risk	Definition of higher risk
Selection bias	Participant sampling	Probability-based sampling with a stated randomisation method.	Non-probability based sampling, such as consecutive or convenient enrolment, or recruitment method not reported.
Selection bias	Participant characterisation	Participants described in sufficient detail to assess representativeness, with complete reporting on HIV prevalence, age range, mean or median age, and study location.	Participants not described in sufficient detail, with incomplete reporting on one or more key characteristics.
Measurement bias	Diagnostic method consistency	Standardised mode of sample collection and a single diagnostic technique consistently applied to all participants.	Variation in sample collection methods or use of multiple diagnostic techniques within the same study.
Study precision	Sample size adequacy	Study tested ≥ 100 participants.	Study tested < 100 participants.

Criteria adapted from the Joanna Briggs Institute critical appraisal tool for prevalence studies.[289]

Table A6: Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) guideline checklist

Topic	Item	Checklist item	Location
Title			
Title	1	Identify the report as a systematic review.	Title
Abstract			
Abstract	2	See the PRISMA 2020 for Abstracts checklist.	Abstract
Introduction			
Rationale	3	Describe the rationale for the review in the context of existing knowledge.	Introduction
Objectives	4	Provide an explicit statement of the objective(s) or question(s) the review addresses.	Introduction
Methods			
Eligibility criteria	5	Specify the inclusion and exclusion criteria for the review and how studies were grouped for the syntheses.	Methods
Information sources	6	Specify all databases, registers, websites, organisations, reference lists and other sources searched or consulted to identify studies. Specify the date when each source was last searched or consulted.	Methods
Search strategy	7	Present the full search strategies for all databases, registers and websites, including any filters and limits used.	Methods, Tables A1, A2
Selection process	8	Specify the methods used to decide whether a study met the inclusion criteria of the review, including how many reviewers screened each record and each report retrieved, whether they worked independently, and if applicable, details of automation tools used in the process.	Methods
Data collection process	9	Specify the methods used to collect data from reports, including how many reviewers collected data from each report, whether they worked independently, any processes for obtaining or confirming data from study investigators, and if applicable, details of automation tools used in the process.	Methods
Data items	10a	List and define all outcomes for which data were sought. Specify whether all results that were compatible with each outcome domain in each study were sought (e.g. for all measures, time points, analyses), and if not, the methods used to decide which results to collect.	Methods, Table A3
	10b	List and define all other variables for which data were sought (e.g. participant and intervention characteristics, funding sources). Describe any assumptions made about any missing or unclear information.	Methods, Tables A2 and A3
Study risk of bias assessment	11	Specify the methods used to assess risk of bias in the included studies, including details of the tool(s) used, how many reviewers assessed each study and whether they worked independently, and if applicable, details of automation tools used in the process.	Methods, Table A5
Effect measures	12	Specify for each outcome the effect measure(s) (e.g. risk ratio, mean difference) used in the synthesis or presentation of results.	Methods

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Topic	Item	Checklist item	Location
Synthesis methods	13a	Describe the processes used to decide which studies were eligible for each synthesis (e.g. tabulating the study intervention characteristics and comparing against the planned groups for each synthesis (item #5)).	Methods
	13b	Describe any methods required to prepare the data for presentation or synthesis, such as handling of missing summary statistics, or data conversions.	Methods, Section A.1.2, Table A4
	13c	Describe any methods used to tabulate or visually display results of individual studies and syntheses.	Methods
	13d	Describe any methods used to synthesize results and provide a rationale for the choice(s). If meta-analysis was performed, describe the model(s), method(s) to identify the presence and extent of statistical heterogeneity, and software package(s) used.	Methods
	13e	Describe any methods used to explore possible causes of heterogeneity among study results (e.g. subgroup analysis, meta-regression).	Methods
	13f	Describe any sensitivity analyses conducted to assess robustness of the synthesized results.	Methods
Reporting bias assessment	14	Describe any methods used to assess risk of bias due to missing results in a synthesis (arising from reporting biases).	Not applicable
Certainty assessment	15	Describe any methods used to assess certainty (or confidence) in the body of evidence for an outcome.	Not applicable
Results			
Study selection	16a	Describe the results of the search and selection process, from the number of records identified in the search to the number of studies included in the review, ideally using a flow diagram.	Results, Figures 2.1 and A1
	16b	Cite studies that might appear to meet the inclusion criteria, but which were excluded, and explain why they were excluded.	Results, Figure 2.1
Study characteristics	17	Cite each included study and present its characteristics.	Table A7, Online supplementary data
Risk of bias in studies	18	Present assessments of risk of bias for each included study.	Online supplementary data
Results of individual studies	19	For all outcomes, present, for each study: (a) summary statistics for each group (where appropriate) and (b) an effect estimate and its precision (e.g. confidence/credible interval), ideally using structured tables or plots.	Online supplementary data
Results of syntheses	20a	For each synthesis, briefly summarise the characteristics and risk of bias among contributing studies.	Results, Tables 2.1
	20b	Present results of all statistical syntheses conducted. If meta-analysis was done, present for each the summary estimate and its precision (e.g. confidence/credible interval) and measures of statistical heterogeneity. If comparing groups, describe the direction of the effect.	Results, Tables 2.2, A8 - A15, Figures 2.2 - 2.4 and A2 - A4
	20c	Present results of all investigations of possible causes of heterogeneity among study results.	Results, Tables 2.2, A9 - A15, Figures 2.2 - 2.4 and A2 - A4

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Topic	Item	Checklist item	Location
	20d	Present results of all sensitivity analyses conducted to assess the robustness of the synthesized results.	Results, Tables A16 and A17,
Reporting biases	21	Present assessments of risk of bias due to missing results (arising from reporting biases) for each synthesis assessed.	Not applicable
Certainty of evidence	22	Present assessments of certainty (or confidence) in the body of evidence for each outcome assessed.	Not applicable
Discussion			
Discussion	23a	Provide a general interpretation of the results in the context of other evidence.	Discussion
	23b	Discuss any limitations of the evidence included in the review.	Discussion
	23c	Discuss any limitations of the review processes used.	Discussion
	23d	Discuss implications of the results for practice, policy, and future research.	Discussion
Other information			
Registration and protocol	24a	Provide registration information for the review, including register name and registration number, or state that the review was not registered.	Methods
	24b	Indicate where the review protocol can be accessed, or state that a protocol was not prepared.	Methods
	24c	Describe and explain any amendments to information provided at registration or in the protocol.	Not applicable
Support	25	Describe sources of financial or non-financial support for the review, and the role of the funders or sponsors in the review.	Online publication
Competing interests	26	Declare any competing interests of review authors.	Online publication
Availability of data, code, other materials	27	Report which of the following are publicly available and where they can be found: template data collection forms, data extracted from included studies, data used for all analyses, analytic code, any other materials used in the review.	Online publication

A.2 Supplementary results

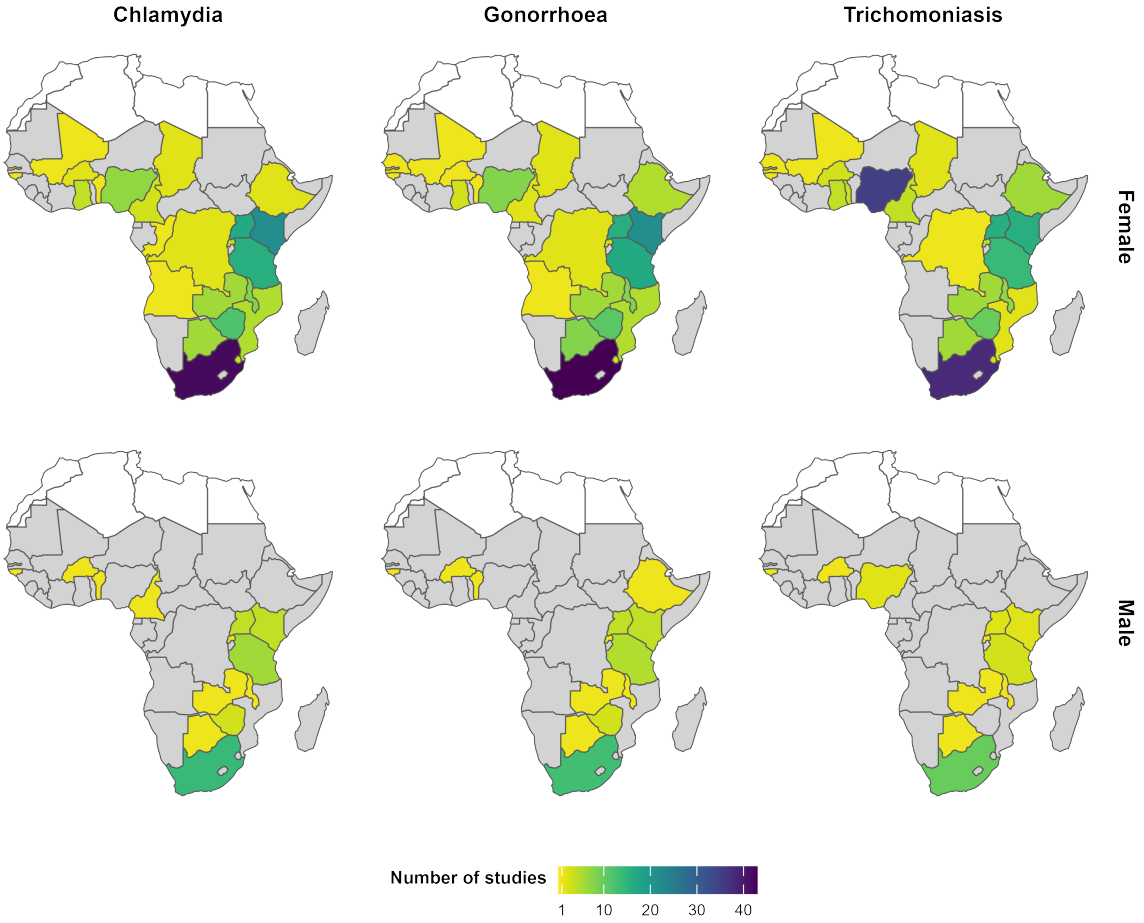


Figure A1: Number of studies included per country in SSA.

Number of studies assessing the prevalence of chlamydia, gonorrhoea, and trichomoniasis among females and males in SSA. Grey shading represents countries with no identified studies. White shading represents countries outside of SSA, according to the UN M49 Standard.[282] Total included studies were 139 for chlamydia, 140 for gonorrhoea, and 162 for trichomoniasis. Base map source is Natural Earth.[465]

Table A7: Overview of studies included in the systematic review

Region	Reference	Year	Country	Population	Sex	Age	Age range	HIV status	Infections
WA	Lagarde 2004[466]	2000	Burkina Faso	Population-representative survey participants	F, M	Adult	13 to 49	Non-stratified	CT, NG, TV
WA	Lafort 2003[467]	2000	Côte d'Ivoire	FP attendees	F	Adult	18 to 53	Non-stratified	CT, NG, TV
WA	Aboyeji 2003[468]	2000	Nigeria	ANC attendees	F	Adult	19 to 43	Non-stratified	NG, TV
WA	Donbraye 2010[469]	2000	Nigeria	ANC attendees	F	Adult	NR	Non-stratified	TV
WA	Apea-Kubi 2004[470]	2001	Ghana	ANC attendees	F	Adult	16+	Non-stratified	CT, NG
WA	Apea-Kubi 2005[471]	2002	Ghana	ANC attendees	F	Adult	16+	Non-stratified	TV
WA	Adejuwon 2005[472]	2002*	Nigeria	FP attendees	F	Adult	NR	Non-stratified	NG, TV
WA	Obiajuru 2005[473]	2002	Nigeria	Community members	F	Adult	NR	Non-stratified	NG, TV
WA	Tukur 2006[474]	2002	Nigeria	FP attendees	F	Adult	NR	Non-stratified	CT
WA	Balaka 2005[475]	2002	Togo	ANC attendees	F	Adult	16 to 42	Non-stratified	TV
WA	Kirakoya-Samadoulougou 2008[476]	2003	Burkina Faso	ANC attendees	F	Adult	14 to 49	Non-stratified	TV
WA	Siemer 2008[477]	2003	Ghana	ANC attendees	F	Adult	NR	Non-stratified	CT
WA	Chigbu 2006[478]	2003*	Nigeria	ANC attendees, GYN attendees	F	Adult	15 to 65	Non-stratified	NG, TV
WA	Inabo 2006[479]	2003*	Nigeria	ANC attendees	F	Adult	18 to 47	Non-stratified	TV
WA	Jatau 2006[480]	2003*	Nigeria	ANC attendees	F	Adult	16+	Non-stratified	TV
WA	Sagay 2005[481]	2003	Nigeria	ANC attendees	F	Adult	NR	Non-stratified	TV
WA	Yirenya-Tawiah 2014[482]	2006	Ghana	Community members	F	Adult	15 to 49	Non-stratified	CT, NG
WA	Fayemiwo 2018[483]	2006	Nigeria	FP attendees	F	Adult	19 to 54	Non-stratified	CT, NG, TV
WA	Omoregie 2009[484]	2006*	Nigeria	PHC/OPD attendees	F, M	Adult	NR	HIV negative	TV
WA	Kengne 2010[485]	2007*	Côte d'Ivoire	ANC attendees	F	Adult	NR	Non-stratified	CT, NG, TV
WA	Chinyere 2012[486]	2007	Nigeria	ANC attendees	F	Adult	15 to 40	Non-stratified	TV
WA	Niemogha 2010[487]	2007*	Nigeria	FP attendees, GYN attendees, Students	F	Adult	NR	Non-stratified	TV
WA	Behanzin 2012[488]	2008	Benin	Population-representative survey participants	F, M	Adult	15 to 49	Non-stratified	CT, NG
WA	Usanga 2011[489]	2008	Nigeria	ANC attendees	F	Adult	15 to 49	Non-stratified	NG, TV

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Region	Reference	Year	Country	Population	Sex	Age	Age range	HIV status	Infections
WA	Sam-Wobo 2012[490]	2009*	Nigeria	ANC attendees	F	Adult	16 to 50	Non-stratified	TV
WA	Arinze 2014[491]	2011*	Nigeria	Students	F	Adult	15 to 30	Non-stratified	CT
WA	Bolaji 2013[492]	2011	Nigeria	ANC attendees	F	Adult	20 to 40	Non-stratified	TV
WA	Tchelougou 2013[493]	2011	Togo	ANC attendees	F	Adult	NR	Non-stratified	TV
WA	Volker 2017[494]	2012	Ghana	ANC attendees	F	Adult	14 to 48	Non-stratified	CT, NG
WA	Adesiji 2015[495]	2012*	Nigeria	FP attendees	F	Adult	20+	Non-stratified	CT
WA	Olowe 2014[496]	2012	Nigeria	ANC attendees	F	Adult	21 to 40	Non-stratified	TV
WA	Samuel 2015[497]	2012*	Nigeria	ANC attendees	F	Adult	21 to 50	Non-stratified	TV
WA	Etuketu 2015[498]	2013	Nigeria	ANC attendees	F	Adult	15 to 44	Non-stratified	TV
WA	Nnaemeka 2016[499]	2013*	Nigeria	Population-representative survey participants	F	Adult	22 to 42	Non-stratified	TV
WA	Olusegun-Joseph 2016[500]	2013*	Nigeria	PHC/OPD attendees, Students	F	Adult	16 to 55	Non-stratified	TV
WA	Akinbo 2017[501]	2014*	Nigeria	Students	F	Youth	13 to 17	Non-stratified	TV
WA	Oyeyemi 2016[502]	2014	Nigeria	ANC attendees	F	Adult	21+	Non-stratified	TV
WA	Wokem 2015[503]	2014	Nigeria	ANC attendees	F	Adult	11 to 60	Non-stratified	TV
WA	Sangare 2021[504]	2015	Burkina Faso	ANC attendees	F	Adult	15 to 49	Non-stratified	TV
WA	Konadu 2019[505]	2015	Ghana	ANC attendees	F	Adult	12 to 54	Non-stratified	TV
WA	Alexander 2018[506]	2015	Nigeria	ANC attendees	F	Adult	15 to 60	Non-stratified	TV
WA	Ebhodaghe 2017[507]	2015	Nigeria	ANC attendees	F	Adult	19 to 43	HIV negative	CT, NG, TV
WA	Asmah 2017[508]	2016	Ghana	ANC attendees	F	Adult	NR	Non-stratified	TV
WA	Squire 2019[509]	2016	Ghana	GYN attendees	F	Adult	16+	Non-stratified	TV
WA	Cowley 2021[510]	2016	Guinea-Bissau	Population-representative survey participants	F, M	Adult	16 to 49	Non-stratified	CT, NG, TV
WA	Ezeanya 2019[511]	2016*	Nigeria	Students	F	Adult	15 to 39	Non-stratified	CT, TV
WA	Ukatu 2019[512]	2016*	Nigeria	ANC attendees	F	Adult	18 to 45	Non-stratified	TV
WA	Kashibu 2018[513]	2017	Nigeria	ANC attendees	F	Adult	15 to 39	Non-stratified	TV
WA	Odaranle 2020[514]	2017	Nigeria	FP attendees	F	Adult	20 to 45	Non-stratified	TV
WA	Isara 2021[515]	2017	Gambia	ANC attendees	F	Adult	15 to 44	Non-stratified	CT, NG, TV
WA	Jary 2021[516]	2018	Mali	PHC/OPD attendees	F	Adult	18+	Non-stratified	CT, NG, TV
WA	Rasheed 2021[517]	2018	Nigeria	ANC attendees	F	Adult	18+	Non-stratified	TV

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Region	Reference	Year	Country	Population	Sex	Age	Age range	HIV status	Infections
WA	Ajani 2022[518]	2019	Nigeria	Students	F, M	Adult	15 to 30	Non-stratified	TV
WA	Auta 2020[519]	2019	Nigeria	ANC attendees	F	Adult	15+	Non-stratified	TV
WA	Maureen 2022[520]	2019*	Nigeria	ANC attendees	F	Adult	18+	Non-stratified	CT, NG, TV
WA	Lingani 2021[521]	2020	Burkina Faso	ANC attendees	F	Adult	16 to 45	Non-stratified	CT
WA	Agabi 2023[522]	2020*	Nigeria	GYN attendees	F	Adult	16 to 57	Non-stratified	TV
WA	Enwuru 2024[523]	2020	Nigeria	ANC attendees	F	Adult	15+	HIV negative	TV
WA	Butcher 2023[524]	2020	Gambia	Prevention trial participants	F	Adult	15 to 69	Non-stratified	CT, NG, TV
WA	Ngom 2023[525]	2021	Senegal	ANC attendees	F	Adult	16 to 46	Non-stratified	NG, TV
CA	Ngandjio 2003[526]	2001	Cameroon	Students	F, M	Youth	NR	Non-stratified	CT
CA	Kinoshita-Moleka 2008[527]	2004	Democratic Republic of the Congo	ANC attendees	F	Adult	15 to 45	Non-stratified	CT, NG
CA	Mbu 2008[528]	2006	Cameroon	ANC attendees	F	Adult	NR	Non-stratified	CT, NG, TV
CA	Alexandre 2015[529]	2012	Angola	PHC/OPD attendees	F	Adult	14 to 52	Non-stratified	CT, NG
CA	Vieira-Baptista 2017[530]	2015	Sao Tome and Principe	GYN attendees	F	Adult	21 to 60	Non-stratified	CT, NG, TV
CA	Compain 2019[531]	2017	Chad	Community members	F	Adult	20 to 65	Non-stratified	CT, NG, TV
CA	Nodjikouambaye 2019[532]	2017	Chad	GYN attendees	F	Adult	18+	Non-stratified	CT, NG, TV
CA	Gadoth 2019[533]	2017	Democratic Republic of the Congo	ANC attendees	F	Adult	18+	HIV negative	CT, NG, TV
CA	Mbah 2022[534]	2018	Cameroon	ANC attendees	F	Adult	15 to 46	Non-stratified	CT, NG, TV
CA	Payne 2020[535]	2018	Cameroon	PHC/OPD attendees	F	Adult	15 to 55	Non-stratified	TV
CA	Ngombe Mouabata 2024[536]	2021	Congo	GYN attendees	F	Adult	21 to 71	Non-stratified	CT
CA	Eyong 2023[537]	2022	Cameroon	PHC/OPD attendees	F	Adult	17 to 53	Non-stratified	TV
EA	Hawken 2002[538]	2000	Kenya	Population-representative survey participants	F, M	Adult	15 to 49	Non-stratified	CT, NG
EA	Kaydos-Daniels 2003[539]	2000	Malawi	PHC/OPD attendees	M	Adult	18+	Non-stratified	TV
EA	Paz-Soldan 2012[540]	2000	Malawi	Population-representative survey participants	F, M	Adult	15-44	Non-stratified	CT, NG
EA	Menendez 2010[541]	2000	Mozambique	ANC attendees	F	Adult	14 to 61	Non-stratified	CT, NG, TV

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Region	Reference	Year	Country	Population	Sex	Age	Age range	HIV status	Infections
EA	Clift 2003[542]	2000	United Republic of Tanzania	Community members	F, M	Adult	16 to 54	Non-stratified	CT, NG
EA	Cowan 2002[543]	2000	Zimbabwe	Community members	F, M	Youth	16 to 19	Non-stratified	CT, NG
EA	van de Wijgert 2009[544]	2002	Uganda, Zimbabwe	FP attendees	F	Adult	18 to 35	HIV negative	CT, NG, TV
EA	Munjoma 2010[545]	2002	Zimbabwe	ANC attendees	F	Adult	NR	Non-stratified	TV
EA	Bailey 2007[546]	2003	Kenya	Prevention trial participants	M	Youth	18 to 24	HIV negative	CT, NG, TV
EA	Ghebremichael 2009[547]	2003	United Republic of Tanzania	Community members	F	Adult	20 to 44	Non-stratified	CT, NG, TV
EA	Ghebremichael 2011[548]	2003	United Republic of Tanzania	Community members	M	Adult	20+	Non-stratified	CT, TV
EA	Mapingure 2010[549]	2003	United Republic of Tanzania, Zimbabwe	ANC attendees	F	Adult	14 to 43	Non-stratified	TV
EA	Msuya 2009[550]	2003	United Republic of Tanzania	ANC attendees	F	Adult	14 to 43	Non-stratified	NG, TV
EA	Celentano 2010[551]	2003	Zimbabwe	Community members	F, M	Adult	18 to 30	Non-stratified	CT, NG, TV
EA	Mensch 2008[552]	2004	Malawi	Population-representative survey participants	F	Youth	15 to 21	Non-stratified	CT, NG, TV
EA	Lujan 2008[553]	2004	Mozambique	ANC attendees	F	Adult	15 to 45	Non-stratified	CT, NG
EA	Kamali 2010[554]	2004	Uganda	Prevention trial participants	F	Adult	18 to 45	Non-stratified	CT, NG, TV
EA	Tann 2006[555]	2004	Uganda	ANC attendees	F	Adult	15 to 40	Non-stratified	CT, NG, TV
EA	Ramjee 2008[556]	2004	Zambia	Prevention trial participants	F	Adult	18+	Non-stratified	CT, NG, TV
EA	Venkatesh 2011[557]	2004	Zimbabwe	Prevention trial participants	F	Adult	18 to 49	HIV negative	CT, NG, TV
EA	Gray 2009[558]	2005	Uganda	Community members	F	Adult	15 to 49	HIV negative	TV
EA	Lingappa 2009[559]	2006	Botswana, Kenya, Rwanda, South Africa, United Republic of Tanzania, Uganda, Zambia	Prevention trial participants	F, M	Adult	18+	HIV negative	CT, NG, TV
EA	Chersich 2009[560]	2006	Kenya	PHC/OPD attendees	F	Adult	16 to 45	Non-stratified	TV
EA	Guffey 2014[561]	2006	Malawi, Zambia, Zimbabwe	Prevention trial participants	F	Adult	18+	HIV negative	CT, NG, TV

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Region	Reference	Year	Country	Population	Sex	Age	Age range	HIV status	Infections
EA	McCormack 2010[562]	2007	United Republic of Tanzania, Uganda, Zambia	Prevention trial participants	F	Adult	16+	HIV negative	CT, NG, TV
EA	Crucitti 2010[563]	2007*	Zambia	ANC attendees, Students	F	Adult	15 to 42	Non-stratified	TV
EA	Otieno 2015[564]	2008	Kenya	Community members	F, M	Adult	18 to 34	HIV negative	CT, NG
EA	Mocumbi 2017[565]	2008	Mozambique	Prevention trial participants	F	Adult	17 to 59	HIV negative	CT, NG, TV
EA	Lemme 2013[566]	2008	United Republic of Tanzania	Community members	F, M	Youth	15 to 30	Non-stratified	CT, NG
EA	Muvunyi 2011[567]	2009	Rwanda	Community members	F	Adult	21 to 45	Non-stratified	CT, NG
EA	Rutherford 2014[568]	2009	Uganda	Students	F, M	Youth	19 to 25	Non-stratified	CT, NG, TV
EA	Ademe 2013[569]	2010	Ethiopia	ANC attendees	F	Adult	15 to 49	Non-stratified	TV
EA	Chiduo 2012[570]	2010	United Republic of Tanzania	ANC attendees	F	Adult	18 to 44	Non-stratified	CT, NG, TV
EA	Downs 2012[571]	2010	United Republic of Tanzania	PHC/OPD attendees	F	Adult	18 to 50	Non-stratified	CT, NG
EA	Jespers 2014[572]	2011	Kenya	ANC attendees, FP attendees	F	Adult	18 to 35	Non-stratified	CT, NG, TV
EA	de Walque 2012[573]	2011	United Republic of Tanzania	Prevention trial participants	F, M	Adult	18 to 30	Non-stratified	CT, NG, TV
EA	Lazenby 2014[574]	2011*	United Republic of Tanzania	GYN attendees	F	Adult	30 to 60	Non-stratified	CT, NG, TV
EA	Kiene 2017[575]	2011	Uganda	PHC/OPD attendees	F, M	Adult	18+	Non-stratified	CT, NG
EA	Ogilvie 2013[576]	2011	Uganda	Community members	F	Adult	26 to 69	Non-stratified	CT, NG
EA	Eshete 2013[577]	2012	Ethiopia	ANC attendees	F	Adult	15 to 36	Non-stratified	TV
EA	Kerubo 2016[578]	2012	Kenya	Students	F	Youth	14 to 17	Non-stratified	CT, NG, TV
EA	Kinuthia 2015[579]	2012	Kenya	ANC attendees	F	Adult	14+	HIV negative	CT, NG, TV
EA	Ravindran 2021[580]	2012	Kenya	ANC attendees	F	Adult	14+	HIV negative	CT, NG, TV
EA	Nkhoma 2017[581]	2012	Malawi	ANC attendees	F	Adult	15+	Non-stratified	TV
EA	Hokororo 2015[582]	2012	United Republic of Tanzania	ANC attendees	F	Youth	14 to 20	Non-stratified	CT, NG, TV
EA	Stephen 2017[583]	2012	Zimbabwe	ANC attendees	F	Adult	18+	Non-stratified	CT
EA	Mulu 2015[584]	2013	Ethiopia	ANC attendees	F	Adult	15 to 49	Non-stratified	NG, TV

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Region	Reference	Year	Country	Population	Sex	Age	Age range	HIV status	Infections
EA	Maina 2016[585]	2013	Kenya	FP attendees	F	Adult	20 to 49	Non-stratified	CT, NG, TV
EA	Palanee-Phillips 2015[586]	2013	Malawi, Uganda, Zimbabwe	Prevention trial participants	F	Adult	18 to 45	HIV negative	CT, NG, TV
EA	Kestelyn 2018[587]	2013	Rwanda	Prevention trial participants	F	Adult	18 to 35	Non-stratified	CT, NG, TV
EA	Donders 2016[588]	2013*	Uganda	PHC/OPD attendees	F	Adult	NR	Non-stratified	CT, NG, TV
EA	Nakubulwa 2015[589]	2013	Uganda	ANC attendees	F	Adult	18+	Non-stratified	CT, TV
EA	Schonfeld 2018	2014	Ethiopia	ANC attendees	F	Adult	NR	Non-stratified	CT, NG, TV
EA	Kanyina 2017[590]	2014	Kenya	GYN attendees	F	Adult	15+	Non-stratified	TV
EA	Oliver 2018[591]	2014	Kenya	Community members	F	Adult	18 to 34	Non-stratified	CT, NG
EA	Franceschi 2016[592]	2014	Rwanda	Students	F	Youth	18 to 20	Non-stratified	CT
EA	Francis 2019[593]	2014	United Republic of Tanzania	Students	F	Youth	17 to 18	Non-stratified	CT, NG, TV
EA	Homsy 2019[594]	2014	Uganda	ANC attendees	F	Adult	18 to 49	HIV negative	TV
EA	Moses 2015[595]	2014	Uganda	Community members	F	Adult	30 to 65	Non-stratified	CT, NG
EA	Chaponda 2021[596]	2014	Zambia	ANC attendees	F	Adult	NR	Non-stratified	CT, NG, TV
EA	Tadesse 2016[597]	2015	Ethiopia	GYN attendees	F	Adult	15 to 49	Non-stratified	NG
EA	Masese 2017[598]	2015	Kenya	Students	F	Youth	15 to 24	Non-stratified	CT, NG, TV
EA	Masha 2017[599]	2015	Kenya	ANC attendees	F	Adult	18 to 45	Non-stratified	CT, NG, TV
EA	Yuh 2020[600]	2015	Kenya	Students	F	Youth	16 to 20	HIV negative	CT, NG, TV
EA	Mukanyangezi 2018[601]	2015	Rwanda	GYN attendees	F	Adult	18+	HIV negative	TV
EA	Maufi 2016[602]	2015	United Republic of Tanzania	ANC attendees	F	Adult	17 to 46	Non-stratified	TV
EA	Yegorov 2018[603]	2015	Uganda	PHC/OPD attendees	F	Adult	18 to 45	HIV negative	CT, NG, TV
EA	Deese 2021[604]	2016	Kenya, Zambia	FP attendees	F	Adult	16 to 35	HIV negative	CT, NG
EA	Mgodi 2021[605]	2017	Kenya, Malawi, Mozambique, United Republic of Tanzania, Zimbabwe	Prevention trial participants	F	Adult	18 to 50	HIV negative	CT, NG
EA	Baussano 2021[606]	2017	Rwanda	Students	F	Youth	17 to 21	Non-stratified	CT
EA	Nsereko 2020[607]	2017	Rwanda	ANC attendees	F	Adult	18 to 49	Non-stratified	TV

Continued...

Region	Reference	Year	Country	Population	Sex	Age	Age range	HIV status	Infections
EA	Masatu 2022[608]	2017	United Republic of Tanzania	FP attendees	F	Adult	18+	Non-stratified	CT
EA	Kahsay 2023[609]	2018	Ethiopia	PHC/OPD attendees	F, M	Adult	15+	Non-stratified	NG
EA	Madanitsa 2023[610]	2018	Kenya, Malawi, United Republic of Tanzania	ANC attendees	F	Adult	NR	HIV negative	CT, NG, TV
EA	Mehta 2023[611]	2018	Kenya	Students	F	Youth	14 to 22	Non-stratified	CT, NG, TV
EA	Celum 2022[612]	2019	Kenya	FP attendees	F	Youth	16 to 25	HIV negative	CT, NG
EA	Lokken 2022[613]	2019	Kenya	FP attendees	F	Adult	18 to 45	HIV negative	CT, NG, TV
EA	Juliana 2020[614]	2019	United Republic of Tanzania	ANC attendees	F	Adult	16 to 48	Non-stratified	CT, NG, TV
EA	Chitneni 2020[615]	2019	Uganda	PHC/OPD attendees	F	Adult	18 to 40	HIV negative	CT, NG, TV
EA	Grabowski 2022[317]	2019	Uganda	Population-representative survey participants	F, M	Adult	18 to 49	Non-stratified	CT, NG, TV
EA	Husen 2023[616]	2020*	Ethiopia	ANC attendees	F	Adult	17 to 37	Non-stratified	TV
EA	Zenebe 2021[617]	2020	Ethiopia	ANC attendees	F	Adult	17 to 41	Non-stratified	CT, NG, TV
EA	Heffron 2021[618]	2020	Kenya	GYN attendees	F	Adult	15 to 30	HIV negative	CT, NG
EA	Mcharo 2022[619]	2020	United Republic of Tanzania	Students	F, M	Youth	18 to 24	Non-stratified	CT, NG
EA	Nair 2023[620]	2020	Uganda, Zimbabwe	Prevention trial participants	F	Youth	16 to 21	HIV negative	CT, NG, TV
EA	Martin 2021[452]	2020	Zimbabwe	Community members	F, M	Youth	16 to 24	Non-stratified	CT, NG
EA	Nyakambi 2022[621]	2021	Kenya	PHC/OPD attendees	F	Adult	18 to 49	Non-stratified	CT
EA	Oware 2023[622]	2021	Kenya	Prevention trial participants	F	Adult	18 to 30	HIV negative	CT, NG
EA	van der Veer 2024[623]	2021	Malawi	ANC attendees	F	Adult	NR	Non-stratified	CT, NG, TV
EA	Sineque 2024[624]	2021	Mozambique	PHC/OPD attendees	F	Adult	30 to 55	Non-stratified	CT, NG
EA	Mbuvi 2024[625]	2022	Kenya	PHC/OPD attendees	F	Adult	15 to 44	Non-stratified	NG, TV
EA	Senkoro 2024[626]	2022	United Republic of Tanzania	GYN attendees	F	Adult	18 to 45	Non-stratified	NG, TV
SA	Romoren 2007[627]	2000	Botswana	ANC attendees	F	Adult	15 to 43	Non-stratified	NG, TV
SA	Kleinschmidt 2007[628]	2000	South Africa	FP attendees	F	Adult	18 to 40	HIV negative	NG, TV
SA	Sturm 2004[629]	2001*	South Africa	ANC attendees	F	Adult	NR	Non-stratified	CT, NG, TV

Continued...

Region	Reference	Year	Country	Population	Sex	Age	Age range	HIV status	Infections
SA	Paz-Bailey 2005[630]	2002	Botswana	FP attendees	F	Adult	NR	Non-stratified	NG, TV
SA	Pettifor 2005[631]	2002	South Africa	PHC/OPD attendees	F, M	Youth	15 to 24	Non-stratified	CT, NG
SA	van de Wijgert 2006[632]	2002	South Africa	PHC/OPD attendees	F	Adult	18 to 69	Non-stratified	CT, NG, TV
SA	Odendaal 2006[633]	2003	South Africa	ANC attendees	F	Adult	NR	Non-stratified	CT, NG
SA	Sobngwi-Tambekou 2009[634]	2003	South Africa	Prevention trial participants	M	Adult	18 to 24	Non-stratified	CT, NG, TV
SA	Ramjee 2008[556]	2004	South Africa	Prevention trial participants	F	Adult	18+	Non-stratified	CT, NG, TV
SA	Sebitloane 2011[635]	2004	South Africa	ANC attendees	F	Adult	18+	HIV negative	TV
SA	Venkatesh 2011[557]	2004	South Africa	Prevention trial participants	F	Adult	18 to 49	HIV negative	CT, NG, TV
SA	Black 2008[636]	2005	South Africa	PHC/OPD attendees	M	Adult	NR	Non-stratified	CT, NG, TV
SA	Guffey 2014[561]	2006	South Africa	Prevention trial participants	F	Adult	18+	HIV negative	CT, NG, TV
SA	Lewis 2008[637]	2006	South Africa	PHC/OPD attendees	M	Adult	17 to 73	Non-stratified	CT, NG, TV
SA	De Jongh 2010[638]	2007*	South Africa	GYN attendees	F	Adult	13 to 41	Non-stratified	CT, NG, TV
SA	McCormack 2010[562]	2007	South Africa	Prevention trial participants	F	Adult	18+	HIV negative	CT, NG, TV
SA	Botswana Ministry of Health 2011[639]	2008	Botswana	FP attendees	F	Adult	NR	Non-stratified	CT, NG
SA	Thigpen 2012[640]	2008	Botswana	Prevention trial participants	F	Adult	18 to 39	HIV negative	TV
SA	Moodley 2015[641]	2009	South Africa	ANC attendees	F	Adult	18+	Non-stratified	CT, NG, TV
SA	Jespers 2014[572]	2011	South Africa	ANC attendees, FP attendees	F	Adult	18 to 35	Non-stratified	CT, NG, TV
SA	Kleppa 2015[642]	2011	South Africa	Students	F	Youth	15 to 31	Non-stratified	CT, NG, TV
SA	Peters 2014[643]	2011	South Africa	PHC/OPD attendees	F	Adult	18 to 49	Non-stratified	CT, NG
SA	Galappaththi-Arachchige 2016[644]	2012	South Africa	Students	F	Youth	16 to 20	Non-stratified	CT, NG, TV
SA	Shukla 2023[645]	2012	South Africa	Students	F	Youth	16 to 22	Non-stratified	CT, NG, TV
SA	Jewanraj 2021[646]	2013	South Africa	Prevention trial participants	F	Adult	20 to 44	HIV negative	CT, NG, TV
SA	Jongen 2021[647]	2013	South Africa	PHC/OPD attendees	F	Youth	16 to 24	HIV negative	CT, NG
SA	Palanee-Phillips 2015[586]	2013	South Africa	Prevention trial participants	F	Adult	18 to 45	HIV negative	CT, NG, TV
SA	Barnabas 2018[648]	2014	South Africa	PHC/OPD attendees	F	Youth	16 to 22	HIV negative	CT, NG, TV
SA	Le Roux 2017[649]	2014*	South Africa	PHC/OPD attendees	M	Adult	17 to 65	Non-stratified	CT, NG, TV
SA	Ginindza 2017[650]	2015	Eswatini	PHC/OPD attendees	F	Adult	15 to 49	Non-stratified	CT, NG, TV

Continued...

Region	Reference	Year	Country	Population	Sex	Age	Age range	HIV status	Infections
SA	Abbai-Shaik 2016[651]	2015	South Africa	PHC/OPD attendees	M	Adult	18+	Non-stratified	CT
SA	Huyveneers 2023[652]	2015	South Africa	Prevention trial participants	F	Adult	18 to 45	HIV negative	CT, NG, TV
SA	Kaida 2018[653]	2015	South Africa	Community members	F, M	Youth	16 to 24	Non-stratified	CT, NG, TV
SA	Kharsany 2020[318]	2015	South Africa	Population-representative survey participants	F, M	Adult	15 to 49	Non-stratified	CT, NG, TV
SA	Wynn 2018[654]	2016	Botswana	ANC attendees	F	Adult	18+	Non-stratified	CT, NG, TV
SA	Deese 2021[604]	2016	Eswatini, South Africa	FP attendees	F	Adult	16 to 35	HIV negative	CT, NG
SA	Gorgens 2020[655]	2016	Eswatini	Prevention trial participants	F	Youth	15 to 22	HIV negative	TV
SA	Gill 2020[656]	2016	South Africa	Prevention trial participants	F	Youth	15 to 19	HIV negative	CT, NG, TV
SA	Hoffman 2019[657]	2016	South Africa	PHC/OPD attendees	F	Adult	18 to 75	Non-stratified	CT, NG, TV
SA	Mgodi 2021[605]	2017	Botswana, South Africa	Prevention trial participants	F	Adult	18 to 50	HIV negative	CT, NG
SA	Delany-Moretlwe 2023[658]	2017	South Africa, Zimbabwe	Prevention trial participants	F	Youth	16 to 25	HIV negative	CT, NG, TV
SA	Francis 2018[659]	2017	South Africa	Population-representative survey participants	F, M	Youth	15 to 24	Non-stratified	CT, NG, TV
SA	Dessai 2020[660]	2018	South Africa	ANC attendees	F	Adult	18 to 43	Non-stratified	TV
SA	Govender 2023[661]	2018	South Africa	ANC attendees	F	Adult	15+	HIV negative	CT, NG, TV
SA	Gray 2021[662]	2018	South Africa	Prevention trial participants	F, M	Adult	18 to 35	HIV negative	CT, NG, TV
SA	Joseph Davey 2019[663]	2018	South Africa	ANC attendees	F	Adult	18+	Non-stratified	CT, NG, TV
SA	Naicker 2021[664]	2018	South Africa	ANC attendees	F	Adult	18+	Non-stratified	TV
SA	Price 2024[665]	2018	South Africa	Community members	F, M	Youth	12 to 19	HIV negative	CT, NG
SA	Taku 2021[666]	2018	South Africa	PHC/OPD attendees	F	Adult	30+	Non-stratified	CT, NG, TV
SA	Celum 2022[612]	2019	South Africa	FP attendees	F	Youth	16 to 25	HIV negative	CT, NG
SA	Chetty 2020[667]	2019	South Africa	ANC attendees	F	Adult	18+	Non-stratified	TV
SA	Oree 2022[668]	2019	South Africa	ANC attendees	F	Adult	20 to 40	Non-stratified	NG
SA	de Voux 2023[669]	2020	South Africa	ANC attendees	F	Adult	16+	HIV negative	CT, NG, TV
SA	Mabaso 2022[670]	2020	South Africa	ANC attendees	F	Adult	18+	Non-stratified	CT
SA	Nair 2023[620]	2020	South Africa	Prevention trial participants	F	Youth	16 to 21	HIV negative	CT, NG, TV

Continued...

Region	Reference	Year	Country	Population	Sex	Age	Age range	HIV status	Infections
SA	Jarolimova 2023[671]	2021	South Africa	Population-representative survey participants	F, M	Youth	16 to 29	Non-stratified	CT, NG, TV
SA	Mullick 2023[672]	2021	South Africa	PHC/OPD attendees	F, M	Youth	15 to 24	HIV negative	CT, NG, TV
SA	Mussa 2023[673]	2022	Botswana	ANC attendees	F	Adult	15+	Non-stratified	CT, NG
SA	de Voux 2024[674]	2022	South Africa	ANC attendees	F	Adult	18+	Non-stratified	CT, NG, TV
Multiple	Delany-Moretlwe 2022[286]	2019	Botswana, Eswatini, Kenya, Malawi, South Africa, Uganda, Zimbabwe	Prevention trial participants	F	Adult	18 to 45	HIV negative	CT, NG, TV

Year is midpoint between start and end of data collection.*For studies without data collection dates reported, year was estimated by subtracting the median publication lag from the publication year (three years, based on difference among studies with dates reported).

CA: Central Africa, EA: Eastern Africa, SA: Southern Africa, WA: Western Africa.

ANC: antenatal care, FP: family planning clinic, GYN: gynaecology clinic, PHC/OPD: primary healthcare or outpatient department.

F: Female, M: Male.

Youth: 12–25 years, Adult: 12+ years.

CT: *Chlamydia trachomatis*, NG: *Neisseria gonorrhoeae*, TV: *Trichomonas vaginalis*.

Table A8: Sexually transmitted infection prevalence in 2010 and 2020

Region	Female, % (95% CI)		Male, % (95% CI)	
	2010	2020	2010	2020
Chlamydia				
Western and Central Africa	4.4 (3.1-6.2)	3.9 (2.5-6.2)	2.8 (1.7-4.6)	2.5 (1.4-4.5)
Eastern Africa	4.6 (3.7-5.8)	9.2 (7.0-12.1)	2.8 (2.1-3.7)	5.6 (4.1-7.6)
Southern Africa	13.5 (10.6-17.1)	16.1 (12.2-21.2)	8.2 (6.3-10.5)	9.7 (7.3-13.0)
Sub-Saharan Africa	**5.1 (3.9-6.3)**	**6.9 (5.2-8.7)**	**3.2 (2.2-4.1)**	**4.2 (3.0-5.5)**
Gonorrhoea				
Western and Central Africa	1.3 (0.8-2.0)	1.1 (0.6-2.2)	1.3 (0.6-2.9)	1.2 (0.5-2.9)
Eastern Africa	2.1 (1.5-3.0)	2.4 (1.6-3.6)	1.5 (1.0-2.2)	1.7 (1.1-2.7)
Southern Africa	3.3 (2.3-4.7)	3.9 (2.6-5.9)	2.5 (1.7-3.7)	3.0 (1.9-4.6)
Sub-Saharan Africa	**1.8 (1.2-2.3)**	**1.8 (1.1-2.5)**	**1.5 (0.8-2.2)**	**1.5 (0.8-2.2)**
Trichomoniasis				
Western and Central Africa	8.6 (5.8-12.6)	7.9 (5.1-12.4)	1.2 (0.7-2.0)	1.1 (0.6-1.9)
Eastern Africa	9.7 (7.0-13.4)	7.4 (5.0-11.0)	3.5 (2.4-5.1)	2.7 (1.7-4.2)
Southern Africa	9.9 (7.2-13.6)	6.3 (4.2-9.4)	1.5 (1.0-2.3)	0.9 (0.6-1.6)
Sub-Saharan Africa	**9.1 (6.2-12.0)**	**7.6 (5.1-10.2)**	**2.1 (1.4-2.9)**	**1.7 (1.1-2.4)**

Estimates of chlamydia, gonorrhoea, and trichomoniasis prevalence by sex and sub-region in 2010 and 2020. Sub-regional estimates generated using log-binomial generalised linear mixed-effects models for each infection. Sub-Saharan African estimates represent sex-matched population-weighted means.

Table A9: Adjusted prevalence ratios for chlamydia, gonorrhoea, and trichomoniasis in SSA, estimated via log-binomial generalised linear mixed-effects models: within-study analysis using observations adjusted for diagnostic test performance

Variable	Chlamydia aPR (95% CI)	Gonorrhoea aPR (95% CI)	Trichomoniasis aPR (95% CI)
Intercept	0.13 (0.08-0.21)	0.04 (0.02-0.07)	0.21 (0.17-0.26)
Sub-region			
Western and Central	0.28 (0.13-0.61)	0.54 (0.17-1.66)	0.46 (0.28-0.74)
Eastern	0.43 (0.27-0.68)	0.52 (0.26-1.04)	1.20 (0.86-1.67)
Southern	Ref	Ref	Ref
Sub-region:year*			
Western and Central:Year	1.04 (0.96-1.12)	1.18 (1.04-1.33)	1.02 (1.00-1.04)
Eastern:Year	1.07 (1.03-1.10)	1.00 (0.95-1.05)	1.06 (0.82-1.39)
Southern:Year	1.05 (1.00-1.11)	1.05 (0.97-1.14)	0.89 (0.84-0.95)
Sex			
Female	Ref	Ref	Ref
Male	0.61 (0.56-0.65)	0.73 (0.65-0.83)	0.23 (0.20-0.27)
Age group			
Adult	Ref	Ref	Ref
Youth	1.17 (0.76-1.80)	1.25 (0.66-2.34)	0.36 (0.22-0.57)
HIV status			
Non-stratified	Ref	Ref	Ref
HIV negative	1.08 (0.67-1.73)	1.05 (0.52-2.14)	0.73 (0.19-2.70)
Diagnostic test			
NAAT	Ref	Ref	Ref
Culture	-	7.87 (1.92-32.19)	2.48 (1.50-4.10)
DFA	1.76 (0.52-5.94)	-	-
Rapid antigen test	-	-	0.40 (0.05-3.40)
Wet mount	-	-	0.88 (0.13-6.02)
Model variance			
τ^2 fixed	0.68	0.59	1.16
τ^2 random	0.17	0.37	<0.01
Number groups			
Number studies	26	26	12
Number observations	52	52	24

*Midpoint year between start and end of data collection period; centred at 2012. aPR: adjusted prevalence ratio, 95% CI: 95% confidence interval, DFA: direct fluorescent antibody, NAAT: nucleic acid amplification test, τ^2: variance.

Table A10: Adjusted prevalence ratios for chlamydia, gonorrhoea, and trichomoniasis in SSA, estimated via log-binomial generalised linear mixed-effects models: between-study sensitivity analysis using observations unadjusted for diagnostic test performance

Variable	Chlamydia aPR (95% CI)	Gonorrhoea aPR (95% CI)	Trichomoniasis aPR (95% CI)
Intercept	0.13 (0.10-0.16)	0.03 (0.02-0.05)	0.10 (0.07-0.13)
Sub-region			
Western and Central	0.29 (0.21-0.40)	0.32 (0.20-0.52)	0.89 (0.64-1.23)
Eastern	0.36 (0.34-0.40)	0.63 (0.56-0.72)	0.99 (0.85-1.17)
Southern	Ref	Ref	Ref
Sub-region:year*			
Western and Central:Year	0.99 (0.95-1.02)	0.99 (0.93-1.04)	0.99 (0.96-1.02)
Eastern:Year	1.07 (1.05-1.10)	1.01 (0.99-1.04)	0.97 (0.94-1.01)
Southern:Year	1.01 (0.99-1.03)	1.01 (0.98-1.04)	0.96 (0.92-0.99)
Sub-region:sex†			
Western and Central:Male	0.79 (0.57-1.08)	0.86 (0.45-1.66)	0.15 (0.10-0.23)
Eastern:Male	0.73 (0.65-0.82)	0.58 (0.48-0.70)	0.53 (0.46-0.62)
Southern:Male	0.64 (0.59-0.69)	0.60 (0.51-0.69)	0.26 (0.22-0.30)
Population			
ANC attendees	Ref	Ref	Ref
FP attendees	0.97 (0.64-1.48)	0.87 (0.46-1.66)	0.50 (0.29-0.85)
GYN attendees	0.97 (0.53-1.76)	0.69 (0.31-1.56)	1.47 (0.89-2.43)
PHC/OPD attendees	0.77 (0.55-1.09)	1.43 (0.86-2.35)	0.85 (0.56-1.29)
Students	1.05 (0.69-1.62)	1.21 (0.58-2.56)	1.12 (0.70-1.79)
Community members	0.99 (0.67-1.46)	1.14 (0.65-1.99)	0.88 (0.46-1.68)
Prevention trial participants	0.87 (0.60-1.26)	1.10 (0.64-1.89)	0.97 (0.60-1.55)
Population-representative survey participants	0.92 (0.59-1.44)	1.25 (0.65-2.40)	1.29 (0.71-2.35)
Age group			
Adult	Ref	Ref	Ref
Youth	1.12 (0.84-1.49)	1.01 (0.66-1.54)	0.63 (0.40-0.99)
HIV status			
Non-stratified	Ref	Ref	Ref
HIV negative	1.45 (1.09-1.94)	1.21 (0.79-1.85)	0.82 (0.57-1.17)
Diagnostic test			
NAAT	Ref	Ref	Ref
Culture	-	1.04 (0.63-1.71)	0.84 (0.55-1.28)
DFA	0.78 (0.37-1.64)	-	-
ELISA	1.55 (0.58-4.12)	-	-
Rapid antigen test	1.92 (1.16-3.18)	1.95 (0.35-10.89)	0.87 (0.48-1.58)
Wet mount	-	-	0.74 (0.53-1.04)
Model variance			
τ^2 fixed	0.47	0.22	0.22
τ^2 random	0.34	0.69	0.59
Number groups			
Number studies	139	140	162
Number observations	202	202	207

*Midpoint year between start and end of data collection period; centred at 2012. †Reference sex is female. aPR: adjusted prevalence ratio, 95% CI: 95% confidence interval, DFA: direct fluorescent antibody, ELISA: enzyme-linked immunosorbent assay, NAAT: nucleic acid amplification test, τ^2: variance.

Table A11: Adjusted prevalence ratios for chlamydia, gonorrhoea, and trichomoniasis in SSA, estimated via log-binomial generalised linear mixed-effects models: between-study sensitivity analysis using NAAT-diagnosed observations adjusted for test performance

Variable	Chlamydia aPR (95% CI)	Gonorrhoea aPR (95% CI)	Trichomoniasis aPR (95% CI)
Intercept	0.15 (0.12-0.18)	0.04 (0.03-0.06)	0.14 (0.09-0.23)
Sub-region			
Western and Central	0.29 (0.21-0.40)	0.28 (0.17-0.47)	0.99 (0.40-2.46)
Eastern	0.38 (0.35-0.41)	0.64 (0.56-0.73)	0.58 (0.36-0.93)
Southern	Ref	Ref	Ref
Sub-region:year*			
Western and Central:Year	1.01 (0.97-1.05)	1.06 (0.99-1.13)	0.88 (0.75-1.03)
Eastern:Year	1.07 (1.05-1.09)	1.02 (0.99-1.04)	0.95 (0.88-1.02)
Southern:Year	1.02 (1.00-1.04)	1.02 (0.99-1.05)	0.96 (0.90-1.02)
Sub-region:sex†			
Western and Central:Male	0.55 (0.33-0.91)	1.13 (0.57-2.21)	0.04 (0.01-0.33)
Eastern:Male	0.61 (0.53-0.70)	0.70 (0.56-0.86)	0.39 (0.31-0.47)
Southern:Male	0.61 (0.56-0.67)	0.80 (0.69-0.94)	0.15 (0.11-0.20)
Population			
ANC attendees	Ref	Ref	Ref
FP attendees	0.94 (0.61-1.45)	0.93 (0.50-1.73)	0.06 (0.00-0.82)
GYN attendees	0.98 (0.56-1.71)	0.65 (0.27-1.56)	0.69 (0.28-1.70)
PHC/OPD attendees	0.79 (0.57-1.11)	1.25 (0.77-2.03)	0.97 (0.48-1.93)
Students	0.84 (0.54-1.30)	0.92 (0.48-1.76)	2.24 (1.05-4.77)
Community members	0.93 (0.64-1.35)	0.82 (0.48-1.39)	0.68 (0.23-2.02)
Prevention trial participants	0.84 (0.59-1.19)	1.07 (0.65-1.73)	0.95 (0.39-2.31)
Population-representative survey participants	0.91 (0.60-1.40)	1.13 (0.63-2.02)	0.79 (0.31-1.99)
Age group			
Adult	Ref	Ref	Ref
Youth	1.22 (0.93-1.62)	1.24 (0.85-1.79)	0.33 (0.16-0.69)
HIV status			
Non-stratified	Ref	Ref	Ref
HIV negative	1.42 (1.07-1.88)	0.96 (0.65-1.42)	0.62 (0.32-1.22)
Model variance			
τ^2 fixed	0.53	0.24	1.07
τ^2 random	0.29	0.48	0.65
Number groups			
Number studies	127	115	56
Number observations	188	176	68

*Midpoint year between start and end of data collection period; centred at 2012. †Reference sex is female. aPR: adjusted prevalence ratio, 95% CI: 95% confidence interval, NAAT: nucleic acid amplification test, τ^2: variance.

Table A12: Adjusted prevalence ratios for chlamydia, gonorrhoea, and trichomoniasis in SSA, estimated via log-binomial generalised linear mixed-effects models: between-study sensitivity analysis using NAAT-diagnosed observations unadjusted for test performance

Variable	Chlamydia aPR (95% CI)	Gonorrhoea aPR (95% CI)	Trichomoniasis aPR (95% CI)
Intercept	0.13 (0.11-0.16)	0.04 (0.03-0.06)	0.16 (0.11-0.24)
Sub-region			
Western and Central	0.27 (0.20-0.38)	0.28 (0.17-0.47)	0.98 (0.45-2.14)
Eastern	0.37 (0.34-0.40)	0.63 (0.56-0.71)	0.64 (0.44-0.94)
Southern	Ref	Ref	Ref
Sub-region:year*			
Western and Central:Year	1.01 (0.97-1.05)	1.05 (0.99-1.12)	0.89 (0.78-1.02)
Eastern:Year	1.07 (1.05-1.10)	1.02 (0.99-1.05)	0.94 (0.89-1.00)
Southern:Year	1.01 (0.99-1.03)	1.02 (0.99-1.05)	0.96 (0.92-1.01)
Sub-region:sex†			
Western and Central:Male	0.74 (0.50-1.10)	0.90 (0.46-1.74)	0.00 (0.00-Inf)
Eastern:Male	0.73 (0.65-0.82)	0.57 (0.47-0.69)	0.58 (0.49-0.67)
Southern:Male	0.64 (0.59-0.69)	0.62 (0.54-0.72)	0.26 (0.22-0.30)
Population			
ANC attendees	Ref	Ref	Ref
FP attendees	0.98 (0.64-1.51)	0.97 (0.51-1.85)	0.19 (0.03-1.08)
GYN attendees	0.92 (0.52-1.61)	0.50 (0.20-1.27)	0.64 (0.29-1.40)
PHC/OPD attendees	0.76 (0.54-1.06)	1.15 (0.70-1.90)	0.83 (0.46-1.50)
Students	0.84 (0.55-1.31)	0.95 (0.48-1.88)	1.71 (0.90-3.25)
Community members	0.95 (0.66-1.37)	0.83 (0.48-1.43)	0.49 (0.19-1.23)
Prevention trial participants	0.83 (0.58-1.17)	0.99 (0.60-1.65)	0.82 (0.39-1.74)
Population-representative survey participants	0.93 (0.61-1.42)	1.14 (0.62-2.08)	0.71 (0.32-1.56)
Age group			
Adult	Ref	Ref	Ref
Youth	1.21 (0.92-1.59)	1.14 (0.78-1.68)	0.44 (0.24-0.82)
HIV status			
Non-stratified	Ref	Ref	Ref
HIV negative	1.45 (1.10-1.92)	1.07 (0.72-1.60)	0.64 (0.36-1.14)
Model variance			
τ^2 fixed	0.51	0.26	6.56
τ^2 random	0.29	0.54	0.48
Number groups			
Number studies	127	115	56
Number observations	188	176	68

*Midpoint year between start and end of data collection period; centred at 2012. †Reference sex is female. aPR: adjusted prevalence ratio, 95% CI: 95% confidence interval, NAAT: nucleic acid amplification test, τ^2: variance.

Table A13: Adjusted prevalence ratios for chlamydia, gonorrhoea, and trichomoniasis in SSA, estimated via log-binomial generalised linear mixed-effects models: within-study sensitivity analysis using observations unadjusted for diagnostic test performance

Variable	Chlamydia aPR (95% CI)	Gonorrhoea aPR (95% CI)	Trichomoniasis aPR (95% CI)
Intercept	0.12 (0.08-0.19)	0.04 (0.02-0.08)	0.22 (0.18-0.25)
Sub-region			
Western and Central	0.28 (0.13-0.57)	0.45 (0.15-1.35)	0.35 (0.23-0.52)
Eastern	0.44 (0.28-0.67)	0.48 (0.24-0.95)	0.80 (0.63-1.02)
Southern	Ref	Ref	Ref
Sub-region:year*			
Western and Central:Year	1.03 (0.95-1.11)	1.19 (1.05-1.35)	1.02 (1.00-1.04)
Eastern:Year	1.07 (1.03-1.10)	1.00 (0.95-1.05)	0.95 (0.80-1.12)
Southern:Year	1.05 (1.00-1.10)	1.04 (0.96-1.13)	0.90 (0.86-0.94)
Sex			
Female	Ref	Ref	Ref
Male	0.67 (0.63-0.71)	0.59 (0.52-0.66)	0.34 (0.31-0.38)
Age group			
Adult	Ref	Ref	Ref
Youth	1.08 (0.72-1.62)	1.04 (0.56-1.93)	0.38 (0.27-0.54)
HIV status			
Non-stratified	Ref	Ref	Ref
HIV negative	1.14 (0.73-1.77)	1.17 (0.59-2.32)	0.60 (0.26-1.40)
Diagnostic test			
NAAT	Ref	Ref	Ref
Culture	-	6.18 (1.55-24.62)	2.07 (1.31-3.25)
DFA	1.71 (0.55-5.31)	-	-
Rapid antigen test	-	-	1.13 (0.28-4.62)
Wet mount	-	-	0.00 (0.00-Inf)
Model variance			
τ^2 fixed	0.61	0.66	33.86
τ^2 random	0.15	0.35	<0.01
Number groups			
Number studies	26	26	12
Number observations	52	52	24

*Midpoint year between start and end of data collection period; centred at 2012. aPR: adjusted prevalence ratio, 95% CI: 95% confidence interval, DFA: direct fluorescent antibody, NAAT: nucleic acid amplification test, τ^2: variance.

Table A14: Adjusted prevalence ratios for chlamydia, gonorrhoea, and trichomoniasis in SSA, estimated via log-binomial generalised linear mixed-effects models: within-study sensitivity analysis using NAAT-diagnosed observations adjusted for test performance

Variable	Chlamydia aPR (95% CI)	Gonorrhoea aPR (95% CI)	Trichomoniasis aPR (95% CI)
Intercept	0.13 (0.08-0.21)	0.04 (0.02-0.08)	0.21 (0.17-0.26)
Sub-region			
Western and Central	0.28 (0.12-0.62)	0.54 (0.17-1.70)	0.48 (0.30-0.77)
Eastern	0.43 (0.27-0.69)	0.52 (0.25-1.05)	1.18 (0.85-1.65)
Southern	Ref	Ref	Ref
Sub-region:year*			
Western and Central:Year	1.04 (0.95-1.12)	1.18 (1.04-1.33)	-
Eastern:Year	1.07 (1.03-1.10)	1.00 (0.95-1.05)	1.07 (0.82-1.40)
Southern:Year	1.05 (1.00-1.11)	1.05 (0.97-1.14)	0.89 (0.84-0.95)
Sex			
Female	Ref	Ref	Ref
Male	0.60 (0.56-0.65)	0.74 (0.65-0.84)	0.25 (0.22-0.30)
Age group			
Adult	Ref	Ref	Ref
Youth	1.17 (0.75-1.81)	1.24 (0.65-2.36)	0.35 (0.22-0.56)
HIV status			
Non-stratified	Ref	Ref	Ref
HIV negative	1.08 (0.66-1.75)	1.05 (0.51-2.18)	0.74 (0.20-2.76)
Model variance			
τ^2 fixed	0.70	0.49	1.09
τ^2 random	0.17	0.39	<0.01
Number groups			
Number studies	25	25	8
Number observations	50	50	16

*Midpoint year between start and end of data collection period; centred at 2012. †Reference sex is female. aPR: adjusted prevalence ratio, 95% CI: 95% confidence interval, NAAT: nucleic acid amplification test, τ^2: variance.

Table A15: Adjusted prevalence ratios for chlamydia, gonorrhoea, and trichomoniasis in SSA, estimated via log-binomial generalised linear mixed-effects models: within-study sensitivity analysis using NAAT-diagnosed observations unadjusted for test performance

Variable	Chlamydia aPR (95% CI)	Gonorrhoea aPR (95% CI)	Trichomoniasis aPR (95% CI)
Intercept	0.12 (0.08-0.19)	0.04 (0.02-0.08)	0.21 (0.18-0.25)
Sub-region			
Western and Central	0.28 (0.13-0.58)	0.45 (0.15-1.37)	0.37 (0.25-0.55)
Eastern	0.43 (0.28-0.68)	0.48 (0.24-0.96)	0.80 (0.63-1.01)
Southern	Ref	Ref	Ref
Sub-region:year*			
Western and Central:Year	1.03 (0.95-1.11)	1.19 (1.05-1.35)	-
Eastern:Year	1.07 (1.03-1.10)	1.00 (0.95-1.05)	0.95 (0.80-1.13)
Southern:Year	1.05 (0.99-1.10)	1.04 (0.96-1.13)	0.90 (0.86-0.94)
Sex			
Female	Ref	Ref	Ref
Male	0.66 (0.62-0.71)	0.59 (0.52-0.66)	0.37 (0.33-0.41)
Age group			
Adult	Ref	Ref	Ref
Youth	1.08 (0.72-1.63)	1.04 (0.56-1.95)	0.38 (0.26-0.54)
HIV status			
Non-stratified	Ref	Ref	Ref
HIV negative	1.14 (0.72-1.79)	1.17 (0.58-2.35)	0.61 (0.26-1.43)
Model variance			
τ^2 fixed	0.63	0.58	0.79
τ^2 random	0.15	0.36	<0.01
Number groups			
Number studies	25	25	8
Number observations	50	50	16

*Midpoint year between start and end of data collection period; centred at 2012. †Reference sex is female. aPR: adjusted prevalence ratio, 95% CI: 95% confidence interval, NAAT: nucleic acid amplification test, τ^2: variance.

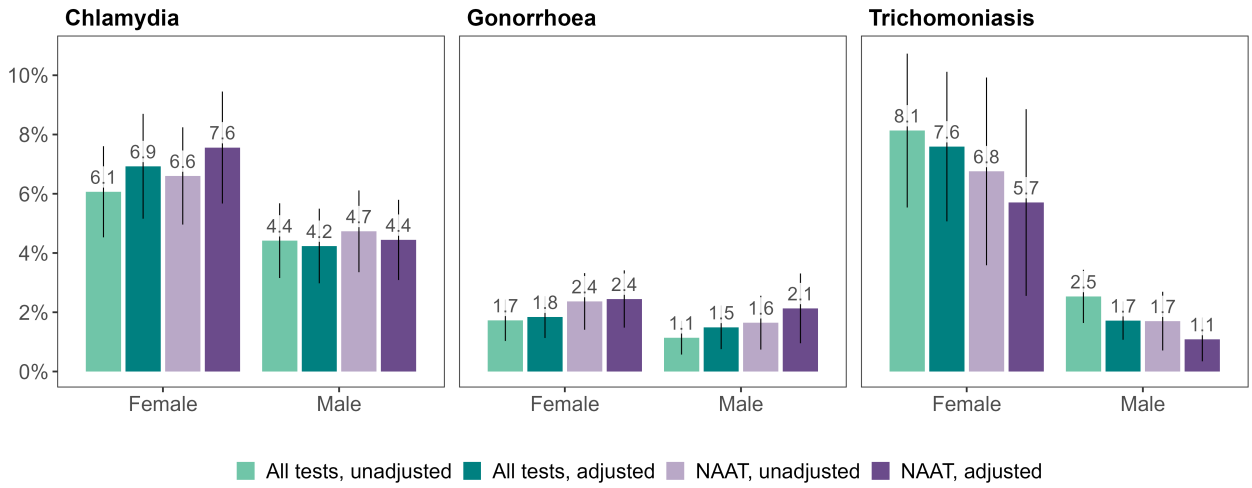


Figure A2: Estimated sexually transmitted infection prevalence in SSA, with and without accounting for diagnostic test performance, 2020

Estimates of chlamydia, gonorrhoea, and trichomoniasis prevalence by sex for SSA in 2020. Sub-regional estimates generated using log-binomial generalised linear mixed-effects models for each infection, using either observations as reported or adjusted for diagnostic test performance, with all diagnostic tests or NAAT only. SSA estimates represent sex-matched population-weighted means. Bars and error lines depict mean prevalence estimates with 95% confidence intervals. NAAT: Nucleic acid amplification test.

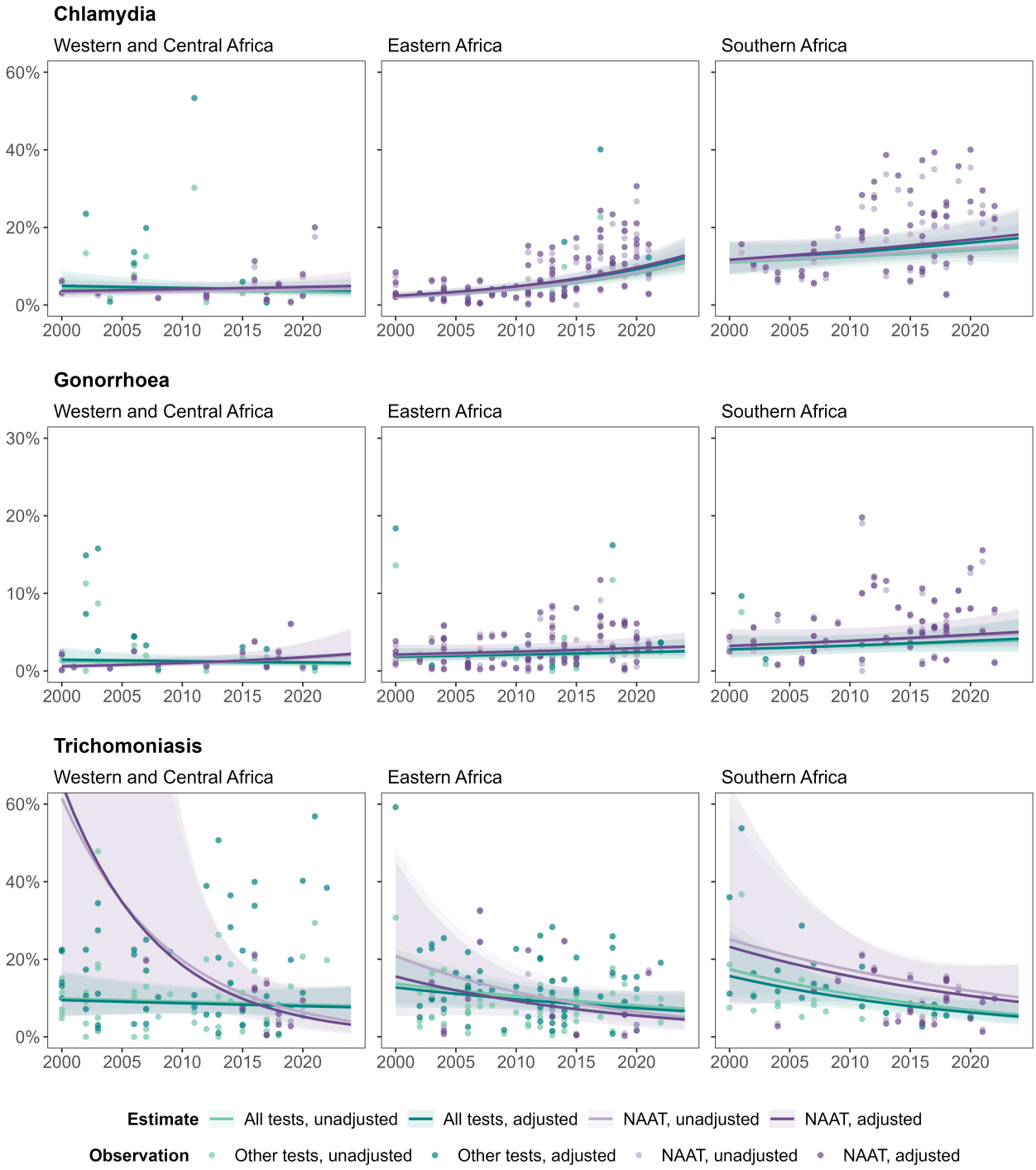


Figure A3: Estimated sexually transmitted infection prevalence among females in SSA, with and without accounting for diagnostic test performance, 2000-2024

Estimates of chlamydia, gonorrhoea, and trichomoniasis prevalence among females between 2000 and 2024. Sub-regional estimates generated using log-binomial generalised linear mixed-effects models for each infection, using either observations as reported or adjusted for diagnostic test performance, with all diagnostic tests or NAAT only. Lines and shading depict mean prevalence estimates with 95% confidence intervals. Points represent study observations. NAAT: Nucleic acid amplification test.

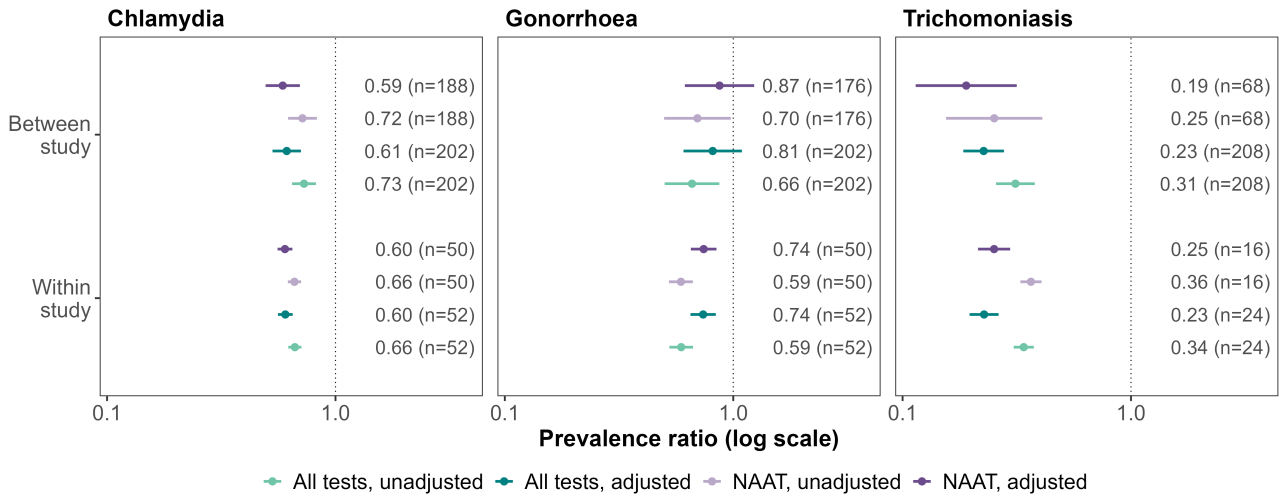


Figure A4: Male-to-female prevalence ratio estimates in SSA, with and without accounting for diagnostic test performance, 2020

Male-to-female prevalence ratios for chlamydia, gonorrhoea, and trichomoniasis. Ratios estimated using log-binomial generalized linear mixed-effects models per infection, using either observations as reported or adjusted for diagnostic test performance, with all diagnostic tests or NAAT only. Models for within-study ratios used study observations among both sexes and between-study ratios used all study observations. Points and error lines depict population-weighted mean ratios and 95% confidence intervals for SSA in 2020.

Table A16: Summary of risk of bias for included studies

Criteria	Chlamydia (N = 139)	Gonorrhoea (N = 140)	Trichomoniasis (N = 162)	Total (N = 211)
Selection bias: Participant sampling				
Lower risk	19 (13.7%)	21 (15.0%)	29 (17.9%)	37 (17.5%)
Higher risk	120 (86.3%)	119 (85.0%)	133 (82.1%)	174 (82.5%)
Selection bias: Participant characterisation				
Lower risk	64 (46.0%)	64 (45.7%)	58 (35.8%)	78 (37.0%)
Higher risk	75 (54.0%)	76 (54.3%)	104 (64.2%)	133 (63.0%)
Measurement bias: Diagnostic method consistency				
Lower risk	135 (97.1%)	136 (97.1%)	151 (93.2%)	198 (93.8%)
Higher risk	4 (2.9%)	4 (2.9%)	11 (6.8%)	13 (6.2%)
Precision: Sample size adequacy				
Lower risk	135 (97.1%)	135 (96.4%)	153 (94.4%)	201 (95.3%)
Higher risk	4 (2.9%)	5 (3.5%)	9 (5.6%)	9 (4.7%)

Table A17: Adjusted prevalence ratios for chlamydia, gonorrhoea, and trichomoniasis in SSA, estimated via log-binomial generalised linear mixed-effects models: assessment of risk of bias criteria using observations adjusted for diagnostic test performance

Variable	Chlamydia aPR (95% CI)	Gonorrhoea aPR (95% CI)	Trichomoniasis aPR (95% CI)
Intercept	0.17 (0.10-0.26)	0.03 (0.01-0.05)	0.09 (0.05-0.14)
Sub-region			
Western and Central	0.31 (0.22-0.43)	0.37 (0.24-0.58)	0.78 (0.55-1.11)
Eastern	0.38 (0.34-0.41)	0.63 (0.54-0.72)	0.98 (0.82-1.17)
Southern	Ref	Ref	Ref
Sub-region:year*			
Western and Central:Year	0.99 (0.95-1.03)	0.98 (0.93-1.04)	1.00 (0.96-1.03)
Eastern:Year	1.07 (1.05-1.09)	1.01 (0.98-1.04)	0.97 (0.94-1.00)
Southern:Year	1.02 (1.00-1.04)	1.02 (0.99-1.05)	0.96 (0.92-1.00)
Sub-region:sex†			
Western and Central:Male	0.66 (0.44-0.98)	1.15 (0.56-2.35)	0.14 (0.09-0.20)
Eastern:Male	0.61 (0.53-0.70)	0.69 (0.57-0.85)	0.36 (0.30-0.44)
Southern:Male	0.60 (0.55-0.66)	0.74 (0.63-0.87)	0.15 (0.11-0.20)
Population			
ANC attendees	Ref	Ref	Ref
FP attendees	1.00 (0.66-1.52)	0.96 (0.54-1.71)	0.68 (0.40-1.17)
GYN attendees	1.07 (0.59-1.93)	0.84 (0.41-1.73)	1.32 (0.79-2.20)
PHC/OPD attendees	0.77 (0.54-1.09)	1.40 (0.87-2.23)	0.93 (0.60-1.46)
Students	0.97 (0.62-1.51)	1.07 (0.53-2.13)	1.23 (0.75-2.03)
Community members	0.91 (0.61-1.36)	1.07 (0.63-1.82)	0.94 (0.47-1.91)
HIV/STI prevention trial participants	0.88 (0.61-1.28)	1.21 (0.74-1.99)	1.04 (0.63-1.72)
Population-representative survey participants	0.80 (0.47-1.36)	1.34 (0.67-2.71)	1.14 (0.57-2.26)
Age group			
Adult	Ref	Ref	Ref
Youth	1.25 (0.93-1.67)	1.32 (0.90-1.94)	0.55 (0.34-0.89)
HIV status			
Non-stratified	Ref	Ref	Ref
HIV negative	1.34 (0.99-1.80)	0.96 (0.64-1.45)	0.94 (0.64-1.37)
Diagnostic test			
NAAT	Ref	Ref	Ref
Culture	-	1.62 (1.01-2.59)	1.27 (0.81-1.99)
DFA	0.61 (0.28-1.36)	-	-
ELISA	2.35 (0.89-6.16)	-	-
Rapid antigen test	3.00 (1.81-5.00)	0.53 (0.06-4.60)	0.85 (0.45-1.62)
Wet mount	-	-	1.55 (1.08-2.24)
Risk of selection bias: participant sampling			
Lower risk	Ref	Ref	Ref
Higher risk	0.88 (0.60-1.29)	1.29 (0.79-2.11)	0.81 (0.55-1.21)

Continued...

Variable	Chlamydia aPR (95% CI)	Gonorrhoea aPR (95% CI)	Trichomoniasis aPR (95% CI)
Risk of selection bias: participant characterisation			
Lower risk	Ref	Ref	Ref
Higher risk	0.91 (0.72-1.14)	1.00 (0.73-1.36)	1.52 (1.22-1.89)
Risk of measurement bias: diagnostic method consistency			
Lower risk	Ref	Ref	Ref
Higher risk	1.64 (0.91-2.94)	1.24 (0.59-2.62)	1.10 (0.66-1.83)
Risk of poor precision: sample size adequacy			
Lower risk	Ref	Ref	Ref
Higher risk	1.37 (1.06-1.76)	1.93 (1.32-2.82)	1.52 (0.95-2.43)
Model variance			
τ^2 fixed	0.56	0.22	0.45
τ^2 random	0.34	0.57	0.65
Number groups			
Number studies	139	140	162
Number observations	202	202	207

*Midpoint year between start and end of data collection period; centred at 2012. †Reference sex is female. aPR: adjusted prevalence ratio, 95% CI: 95% confidence interval, DFA: direct fluorescent antibody, ELISA: enzyme-linked immunosorbent assay, NAAT: nucleic acid amplification test, τ^2: variance.

Appendix B

Supplement to Chapter 3

B.1 Supplementary methods

B.1.1 Search strategy and data extraction

Table B1: Search strategy for systematic review

MEDLINE: Search conducted 20 December 2023 with 1881 articles retrieved.
Symptom domain: exp ulcer/ or exp vaginal discharge/ or exp urethritis/ or exp uterine cervicitis/ OR (GUD or ((genital* or penile or penis or vagin*) adj2 (ulcer* or vesicle* or papule* or chancr* or granulom* or sore* or lesion*)) or ((genital* or penile or penis or urethra* or vagin* or cervic* or cervix) adj2 discharge) or urethritis or cervicitis or vaginitis).ab,kw,ti
AND RTI pathogen domain: exp Sexually Transmitted Diseases/ or exp Chancroid/ or exp Haemophilus ducreyi/ or exp Chlamydia infections/ or exp Chlamydia trachomatis/ or exp Lymphogranuloma Venereum/ or exp Gonorrhea/ or exp Neisseria gonorrhoeae/ or exp Syphilis/ or exp Treponema pallidum/ or exp Herpes Simplex/ or exp Herpesvirus 1, Human/ or exp Herpesvirus 2, Human/ or exp Trichomonas Infections/ or exp Trichomonas vaginalis/ or exp Mycoplasma genitalium/ or exp Vaginosis, Bacterial/ or exp Candidiasis, Vulvovaginal/ or exp Candida albicans/ OR (sexually transmitted disease* or sexually transmitted infection* or STI* or STD* or chancroid* or ducreyi or chlamydia* or Lymphogranuloma Venereum or LGV or gonorrhoea* or gonorrhoeae* or gonorrhea* or syphili* or treponema palli* or herpe* or HSV* or trichomonas* or trichomoniasis* or vaginalis* or mycoplasma or bacterial vaginosis* or candida* or candidiasis* or albicans).ab,kw,ti
AND sub-Saharan Africa domain: exp Africa south of the Sahara/ OR (Africa* or Afriq* or Angola*or Benin* or Botswana* or Motswana* or Batswana* or Burkina* or Burundi* or Cabo Verde* or Cap-Vert* or Cape verde* or Camero* or Central African Republic* or Republique centrafricaine or Chad* or Comor* or Congo* or Democratic Republic of Congo or Republique democratique du Congo DRC or RDC or Cote d'Ivoire or Ivory Coast or Ivorian* or Guinea* or Eritrea* or Erythree*or Ethiop* or Gabon* or Gambi* or Ghana* or Guine* or Equatorial Guinea* or Guinee Equatoriale or Equatoguinean* or Guinea-Bissau* or Kenya* or Lesotho* or Basotho* or Liberia* or Madagas* or Malawi* or Mali* or Mauritani* or Mauri* or Mozambi* or Namibi* or Niger* or Nigeria* or Rwanda* or Rouanda* or Ruanda* or Sao Tome* or Senegal* or Seychel* or Sierra Leone* or Somali* or South Africa* or Afrique du Sud or Sudan* or Soudan* or Swazi* or eSwatini* or Tanzani* or Togo* or Uganda* or Ouganda* or Zambi* or Zimbabwe*).mp
EMBASE: Search conducted 20 December 2023 with 2835 articles retrieved.
Symptom domain: exp genital ulcer/ or exp vagina discharge/ or exp urethritis/ or exp urethral discharge/ or exp uterine cervicitis/ OR (GUD or ((genital* or penile or penis or vagin*) adj2 (ulcer* or vesicle* or papule* or chancr* or granulom* or sore* or lesion*)) or ((genital* or penile or penis or urethra* or vagin* or cervic* or cervix) adj2 discharge) or urethritis or cervicitis or vaginitis).ab,kw,ti
AND RTI pathogen domain: exp sexually transmitted disease/ or exp ulcus molle/ or exp Haemophilus ducreyi/ or exp Chlamydia/ or exp Chlamydia trachomatis/ or exp lymphogranuloma venereum/ or exp gonorrhea/ or exp Neisseria gonorrhoeae/ or exp syphilis/ or exp Treponema pallidum/ or exp Herpes simplex virus/ or exp Herpes simplex virus 1/ or exp Herpes simplex virus 2/ or exp Human immunodeficiency virus/ or exp Trichomonas/ or Trichomonas vaginalis/ or exp trichomoniasis/ or exp Mycoplasma genitalium/ or exp vaginitis/ or exp vagina candidiasis/ or Candida albicans/ OR (sexually transmitted disease* or sexually transmitted infection* or STI* or STD* or chancroid* or ducreyi or chlamydia* or Lymphogranuloma Venereum or LGV or gonorrhoea* or gonorrhoeae* or gonorrhea* or chancre* or syphili* or treponema palli* or herpe* or HSV* or trichomonas* or trichomoniasis* or vaginalis* or mycoplasma or genitalium* or vaginosis* or bacterial vaginosis* or candida* or candidiasis* or albicans).ab,kw,ti

AND sub-Saharan Africa domain: exp Africa south of the Sahara/ or exp africa, eastern/ or exp africa, western/ or exp africa, central/ or exp africa, southern/ OR (Africa* or Afriq* or Angola*or Benin* or Botswana* or Motswana* or Batswana* or Burkina* or Burundi* or Cabo Verde* or Cap-Vert* or Cape verde* or Camero* or Central African Republic* or Republique centrafricaine or Chad* or Comor* or Congo* or Democratic Republic of Congo or Republique democratique du Congo DRC or RDC or Cote d'Ivoire or Ivory Coast or Ivorian* or Guinea* or Eritrea* or Erythree*or Ethiop* or Gabon* or Gambi* or Ghana* or Guine* or Equatorial Guinea* or Guinee Equatoriale or Equatoguinean* or Guinea-Bissau* or Kenya* or Lesotho* or Basotho* or Liberia* or Madagas* or Malawi* or Mali* or Mauritani* or Mauri* or Mozambi* or Namibi* or Niger* or Nigeria* or Rwanda* or Rouanda* or Ruanda* or Sao Tome* or Senegal* or Seychel* or Sierra Leone* or Somali* or South Africa* or Afrique du Sud or Sudan* or Soudan* or Swazi* or eSwatini* or Tanzani* or Togo* or Uganda* or Ouganda* or Zambi* or Zimbabwe*).mp

Global Health: Search conducted 20 December 2023 with 1566 articles retrieved.

Symptom domain: exp genital ulcers/ or urethritis/ or exp cervicitis/ OR (GUD or ((genital* or penile or penis or vagin*) adj2 (ulcer* or vesicle* or papule* or chancr* or granulom* or sore* or lesion*)) or ((genital* or penile or penis or urethra* or vagin* or cervic* or cervix) adj2 discharge) or urethritis or cervicitis or vaginitis).ab,ti,mp.

AND RTI pathogen domain: exp sexually transmitted disease/ or exp chancroid/ or exp Haemophilus ducreyi/ or exp Chlamydia/ or exp Chlamydia trachomatis/ or exp gonorrhoea/ or exp Neisseria gonorrhoeae/ or exp syphilis/ or exp Treponema pallidum/ or exp herpes simplex virus/ or exp Human herpesvirus 1/ or exp Human herpesvirus 2/ or exp human immunodeficiency virus/ or exp Trichomonas/ or Trichomonas vaginalis/ or exp trichomoniasis/ or exp Mycoplasma genitalium/ or exp bacterial vaginitis/ or exp Candida albicans/ OR (sexually transmitted disease* or sexually transmitted infection* or STI* or STD* or chancroid* or ducreyi or chlamydia* or Lymphogranuloma Venereum or LGV or gonorrhoea* or gonorrhoeae* or gonorrhea* or chancre* or syphili* or treponema palli* or herpe* or herpes simplex* or HSV* or trichomonas* or trichomoniasis* or vaginalis* or mycoplasma or genitalium* or vaginosis* or bacterial vaginosis* or candida* or candidiasis* or albicans).ab,ti,mp.

AND sub-Saharan Africa domain: exp Africa south of the Sahara/ or exp Central Africa/ or exp East Africa/ or exp Southern Africa/ or exp West Africa/ OR (Africa* or Afriq* or Angola*or Benin* or Botswana* or Motswana* or Batswana* or Burkina* or Burundi* or Cabo Verde* or Cap-Vert* or Cape verde* or Camero* or Central African Republic* or Republique centrafricaine or Chad* or Comor* or Congo* or Democratic Republic of Congo or Republique democratique du Congo DRC or RDC or Cote d'Ivoire or Ivory Coast or Ivorian* or Guinea* or Eritrea* or Erythree*or Ethiop* or Gabon* or Gambi* or Ghana* or Guine* or Equatorial Guinea* or Guinee Equatoriale or Equatoguinean* or Guinea-Bissau* or Kenya* or Lesotho* or Basotho* or Liberia* or Madagas* or Malawi* or Mali* or Mauritani* or Mauri* or Mozambi* or Namibi* or Niger* or Nigeria* or Rwanda* or Rouanda* or Ruanda* or Sao Tome* or Senegal* or Seychel* or Sierra Leone* or Somali* or South Africa* or Afrique du Sud or Sudan* or Soudan* or Swazi* or eSwatini* or Tanzani* or Togo* or Uganda* or Ouganda* or Zambi* or Zimbabwe*).mp

Web of Science: Search conducted 20 December 2023 with 1498 articles retrieved.

Symptom domain: TS=(((genital* or penile or penis or vagin*) near/2 (ulcer* or vesicle* or papule* or chancr* or granulom* or sore* or lesion*)) or ((genital* or penile or penis or urethra* or vagin* or cervic* or cervix) near/2 discharge) or GUD or urethritis or cervicitis or vaginitis)

AND RTI pathogen domain: TS=("sexually transmitted disease*" or "sexually transmitted infection*" or STI* or STD* or chancroid* or ducreyi or chlamydia* or "Lymphogranuloma Venereum" or LGV or gonorrhoea* or gonorrhoeae* or gonorrhea* or chancre* or syphili* or "treponema palli*" or herpe* or "herpes simplex*" or HSV* or trichomonas* or trichomoniasis* or vaginalis* or mycoplasma or genitalium* or vaginosis* or bacterial vaginosis* or candida* or candidiasis* or albicans)

AND sub-Saharan Africa domain: TS=(Africa* or Afriq* or Angola*or Benin* or Botswana* or Motswana* or Batswana* or Burkina* or Burundi* or Cabo Verde* or Cap-Vert* or Cape verde* or Camero* or Central African Republic* or Republique centrafricaine or Chad* or Comor* or Congo* or Democratic Republic of Congo or Republique democratique du Congo DRC or RDC or Cote d'Ivoire or Ivory Coast or Ivorian* or Guinea* or Eritrea* or Erythree*or Ethiop* or Gabon* or Gambi* or Ghana* or Guine* or Equatorial Guinea* or Guinee Equatoriale or Equatoguinean* or Guinea-Bissau* or Kenya* or Lesotho* or Basotho* or Liberia* or Madagas* or Malawi* or Mali* or Mauritani* or Mauri* or Mozambi* or Namibi* or Niger* or Nigeria* or Rwanda* or Rouanda* or Ruanda* or Sao Tome* or Senegal* or Seychel* or Sierra Leone* or Somali* or South Africa* or Afrique du Sud or Sudan* or Soudan* or Swazi* or eSwatini* or Tanzani* or Togo* or Uganda* or Ouganda* or Zambi* or Zimbabwe*)

Table B2: Classification of included countries by sub-region in SSA

Sub-region	Country
Central Africa	Angola, Cameroon, Central African Republic, Chad, Congo, Democratic Republic of Congo, Equatorial Guinea, Gabon, Sao Tome and Principe
Western Africa	Benin, Burkina Faso, Cabo Verde, Cote d'Ivoire, Gambia, Ghana, Guinea-Bissau, Guinea, Liberia, Mali, Mauritania, Niger, Nigeria, Senegal, Sierra Leone, Togo
Eastern Africa	Burundi, Comoros, Djibouti, Eritrea, Ethiopia, Kenya, Madagascar, Malawi, Mauritius, Mozambique, Rwanda, Seychelles, Somalia, South Sudan, Uganda, United Republic of Tanzania, Zambia, Zimbabwe
Southern Africa	Botswana, Eswatini, Lesotho, Namibia, South Africa

Countries classified according to UN M49 Standard.[282]

Table B3: Variables extracted from included studies

Category	Variable
Study characteristics	• Authors • Publication title • Publication year • Dates of data collection • Country of study • Sub-national region or city of study • Study name (as applicable) • Study design • Study sampling method
Participant characteristics	• Study population category • Study population age (mean, median, range) • Study population HIV status • Study population HIV prevalence
Diagnostic methodology	• Diagnostic test per infection • Diagnostic specimen per infection • Number tested per infection • Number positive per infection • Proportion diagnosed with each infection

B.1.2 Preparation of unpublished data

The literature search identified two databases. The first, from the South Africa National Institute for Communicable Diseases (NICD), covered surveillance among primary healthcare clinic attendees in South Africa from 2006 to 2022. These data were reported in several publications,[306–308,675–684] yet the database included unpublished surveillance rounds and enabled additional outcome stratification. The second was the Centre for Sexual Health and HIV/AIDS Research (CeSHHAR) female sex worker population size estimation study in Zimbabwe in 2017. Conference abstracts reporting study results prompted request for special tabulation. Data extracted from each database, following approaches outlined below, were combined with published study data prior to analysis.

South Africa NICD data extraction

Data were available from 13 routine aetiologic surveillance rounds, which were cross-sectional aetiologic prevalence surveys among clinic attendees examined and diagnosed with vaginal discharge, urethral discharge, and genital ulcer syndromes (Table B4). Variables were standardised and combined into a single database. Simple (unweighted) diagnosed proportions were extracted, with stratification by year, sex, HIV status, and age group. HIV-prevalence and mean age were calculated for each stratum. Prior full-text articles of these data were excluded from the review.

CeSHHAR female sex worker survey data extraction

The female sex worker population size estimation study in Zimbabwe included 2507 women (1497 Harare, 808 Bulawayo, 202 Shamva) recruited using respondent-driven sampling (RDS).[309] A random sample of one third of participants were offered genital examination and STI testing, of whom 80 were identified as having abnormal vaginal discharge. Data were survey-weighted using RDS weights to determine diagnosed proportions and 95% CIs, with stratification by HIV status and age group. Weighted HIV-prevalence and mean age were calculated for each stratum. Numerators and denominators for each proportion accounted for the sampling design by using the method described by Stannah et al. (2023).[685] For each observation, a design effect was estimated using the ratio of variances of the RDS-adjusted proportion and simple random sample proportion, which was used to derive the effective sample size and number of individuals who tested positive. Ethical approval for the study was obtained from the Medical Research Council of Zimbabwe (MRCZ/A/1942) and London School of Hygiene & Tropical Medicine (#9686).

Table B4: Surveys by National Institute for Communicable Diseases in South Africa

Year(s)	Province	Facilities	Participants
2006-2007	Gauteng	Alexandra Health Centre	529 (4.0%)
2006-2007	Northern Cape	Betty Gaetsewe Clinic, City Clinic, Masakhane Clinic	416 (3.2%)
2006-2007	Western Cape	Spencer Road Clinic	243 (1.9%)
2007-2008	Free State	Central Park Clinic, Pelonomi Poly Clinic	360 (2.7%)
2007-2009	Gauteng	Alexandra Health Centre	992 (7.5%)
2010	Gauteng	Alexandra Health Centre	1 057 (8.0%)
2010-2012	Gauteng	Alexandra Health Centre	720 (5.5%)
2010-2012	Limpopo	Rethabile Heath Centre	402 (3.0%)
2011-2012	Gauteng	Alexandra Health Centre	722 (5.5%)
2013-2014	Gauteng	Alexandra Health Centre	546 (4.0%)
2013-2014	KwaZulu-Natal	Central Durban Clinic	406 (3.1%)
2013-2014	Mpumalanga	Nelspruit Clinic	243 (1.9%)
2014-2016	Eastern Cape	Gqebera Clinic	92 (0.7%)
2014-2016	Gauteng	Alexandra Health Clinic	735 (5.6%)
2014-2016	KwaZulu-Natal	East Boom Clinic, Phoenix Clinic	438 (3.3%)
2014-2016	Mpumalanga	Hluvukani Clinic, Kabokweni Clinic	436 (3.3%)
2014-2016	North West	Jouberton Clinic	227 (1.7%)
2017	Eastern Cape	Zwide Clinic	259 (1.9%)
2017	Free State	Heidedal Clinic	129 (1.0%)
2017	Gauteng	Alexandra Clinic	373 (2.8%)
2017	Western Cape	Khayelitsha Site B Clinic, Khayelitsha Site C Clinic	223 (1.7%)
2018-2019	Gauteng	Alexandra Clinic	356 (2.7%)
2018-2019	Limpopo	Rethabile Health Centre	139 (1.1%)
2018-2019	Northern Cape	Kimberly Central Clinic	209 (1.6%)
2020-2022	Gauteng	Alexandra Health Centre	855 (6.5%)
2020-2022	KwaZulu-Natal	Prince Cyril Zulu Communicable Disease Centre	1 032 (7.8%)
2020-2022	Western Cape	Spencer Road Clinic	399 (3.8%)
Total			13 159 (100%)

B.1.3 Adjustment for diagnostic test performance

As described in Chapter 2 (Appendix A, Section A.1.2), diagnostic tests from included studies were classified into broad categories. Antibody tests included enzyme immunoassays or enzyme-linked immunosorbent assays. Culture included any culture test with a valid medium. NAAT included polymerase chain reaction, strand displacement assay, transcription mediated assay, ligase chain reaction, and GeneXpert. Treponemal tests for syphilis included *Treponema pallidum* Particle Agglutination Assay, *Treponema pallidum* Hemagglutination Assay, Fluorescent Treponemal Antibody Absorption, Microhemagglutination Assay for *Treponema pallidum*, and point of care rapid tests. Non-treponemal tests for syphilis included the Venereal Disease Research Laboratory test and Rapid Plasma Reagin test. Syphilis tests were categorised regardless of titre due to variations in reporting. In studies investigating candidiasis, observations labelled as *Candida albicans* according to study descriptions were reclassified more generally as *Candida* species if the diagnostic methods used (culture, wet mount, and gram stain) were not suitable to identify *Candida albicans*. Studies that reported detecting *Gardnerella vaginalis* using diagnostic criteria appropriate for bacterial vaginosis (Amsel Criteria or Nugent Score) were reclassified as such.

Point value sensitivity and specificity estimates for each diagnostic test category were compiled from literature, with priority given to characteristics published by WHO (Tables B5, B6, and B7).[145,230] In cases where multiple sources were available with wide variation, a weighted mean was calculated for the sensitivity and specificity. The following assumptions were made when data were lacking on performance characteristics. For *Candida* species, the accuracy of gram stain was assumed equivalent to wet mount, although the sensitivity of the former test is likely higher. For *T. vaginalis*, the accuracy of culture and wet mount using urine samples was assumed to be equivalent to that of genital fluid samples.[142,230,230,461] In studies using two diagnostic tests to identify a pathogen, combined sensitivity and specificity was determined: (i) if either test could be positive, the lower sensitivity and lower specificity of the two tests was used, and (ii) if both tests had to be positive, the lower sensitivity and higher specificity of the two tests was used.

To correct for imperfect diagnostic test performance, the same Bayesian approach using the Rogan-Gladen estimator was applied as described in Chapter 2 (Appendix A, Section A.1.2).

Table B5: Performance of diagnostic tests among women with vaginal discharge

RTI	Test	Specimen	Number studies	Sensitivity (%)	Specificity (%)	Source
BV	Amsel criteria	Genital fluid	9	51.0	98.0	Dadhwal 2010[686]
	Nugent score	Genital fluid	16	87.0	95.0	Thomason 1992[687] Schwebke 1996[688]
	Amsel criteria and Nugent score	Genital fluid	2	51.0	98.0	Combined test estimate*
	Amsel criteria or Nugent score	Genital fluid	1	51.0	95.0	Combined test estimate*
CA	Germ tube	Genital fluid	7	98.9	98.5	Hoppe 1999[689]
	NAAT	Genital fluid	2	90.9	94.1	Gaydos 2017[690]
CS	Culture	Genital fluid	8	67.0	66.0	WHO 1999[145]
	Gram stain	Genital fluid	11	54.6	89.6	Assumed equivalent to wet mount, but sensitivity likely higher.
	Wet mount	Genital fluid	6	54.6	89.6	Chatwani 2007[691] Schwebke 2018[692]
	Gram stain and culture	Genital fluid	6	54.6	89.6	Combined test estimate*
	Wet mount and culture	Genital fluid	3	54.6	89.6	Combined test estimate*
CT	Culture	Genital fluid	1	63.4	100.0	WHO 2011[230]
	DFA	Genital fluid	5	82.0	98.5	WHO 1999[145]
	ELISA	Genital fluid	6	65.0	100.0	WHO 2011[230]
	NAAT	Genital fluid	18	88.6	99.6	WHO 2011[230]
	NAAT	Urine	2	87.0	99.8	WHO 2011[230]
	Rapid antigen test	Genital fluid	1	48.0	98.0	Grillo-Ardila 2020[463]
	ELISA and NAAT	Genital fluid	1	65.0	99.6	Combined test estimate*
	Culture and DFA	Genital fluid	1	97.0	99.8	Combined test estimate*
MG	NAAT	Genital fluid	7	93.3	97.6	Gaydos 2019[693]
NG	Culture	Genital fluid	26	75.7	100.0	WHO 2011[230]
	NAAT	Genital fluid	15	93.3	99.2	WHO 2011[230]
	NAAT	Urine	1	91.6	100.0	WHO 2011[230]
TV	Culture	Genital fluid	6	68.8	100.0	WHO 2011[230]
	NAAT	Genital fluid	17	95.0	98.0	WHO 2011[230]
	Wet mount	Genital fluid	33	52.0	100.0	WHO 2011[230]
	Culture and NAAT	Genital fluid	1	68.8	100.0	Combined test estimate*
	Culture or NAAT	Genital fluid	1	68.8	98.0	Combined test estimate*
	Culture and wet mount	Genital fluid	2	52.0	100.0	Combined test estimate*

Number of studies indicates the total count of studies included in the review that used each diagnostic test and specimen combination for the detection of a specific pathogen. *Combined sensitivity and specificity estimate determined for studies reporting two diagnostic tests to identify a pathogen. If either test could be positive, the lower sensitivity and lower specificity values were used from the two tests. If both tests had to be positive, the lower sensitivity and higher specificity values were used from the two tests. BV: Bacterial vaginosis, CA: *Candida albicans*, CS: *Candida* species (any), CT: *Chlamydia trachomatis*, MG: *Mycoplasma genitalium*, NG: *Neisseria gonorrhoeae*, TV: *Trichomonas vaginalis*.

Table B6: Performance of diagnostic tests among men with urethral discharge

RTI	Test	Specimen	Number studies	Sensitivity (%)	Specificity (%)	Source
CT	Culture	Genital fluid	1	63.4	100.0	WHO 2011[230]
	DFA	Genital fluid	1	82.0	98.5	WHO 2011[230]
	ELISA	Genital fluid	10	84.0	98.0	WHO 1999[145]
	ELISA	Urine	1	84.0	98.0	WHO 1999[145]
	NAAT	Genital fluid	9	87.5	99.2	WHO 2011[230]
	NAAT	Urine	7	87.8	99.3	WHO 2011[230]
	Culture and DFA	Genital fluid	2	63.4	100.0	Combined test estimate*
MG	NAAT	Genital fluid	4	98.1	99.9	Gaydos 2019[693]
	NAAT	Urine	4	89.4	99.1	Gaydos 2019[693]
NG	Culture	Genital fluid	30	87.6	100.0	WHO 2011[230]
	Gram stain	Genital fluid	3	87.6	100.0	WHO 2011[230]
	NAAT	Genital fluid	7	96.1	99.0	WHO 2011[230]
	NAAT	Urine	6	80.9	99.9	WHO 2011[230]
	Gram stain and culture	Genital fluid	2	87.6	100.0	Combined test estimate*
	Gram stain or NAAT	Genital fluid and urine	1	87.6	99.9	Combined test estimate*
	Culture or NAAT	Genital fluid	1	87.6	99.0	Combined test estimate*
TV	Culture	Genital fluid	4	74.5	100.0	WHO 2011[230]
	Culture	Urine	1	74.5	100.0	Performance of urine culture and urethral swab culture assumed equivalent based on WHO 2011[230] and Kaydos-Daniels 2004[142]
	NAAT	Genital fluid	7	81.6	97.7	WHO 2011[230]
	NAAT	Urine	4	96.0	97.7	WHO 2011[230]
	Rapid antigen	Genital fluid	1	92.5	97.5	Gaydos 2017[148]
	Wet mount	Genital fluid	5	44.0	100.0	WHO 2011[230]
	Wet mount	Urine	1	44.0	100.0	Performance of urine wet mount and urethral swab wet mount assumed equivalent, although the former is likely less sensitive based on WHO 2011[230] and Hobbs 2006[461]
	Culture or NAAT	Genital fluid and urine	1	74.5	97.7	Combined test estimate*
	Culture and wet mount	Genital fluid	2	44.0	100.0	Combined test estimate*
	Culture or wet mount	Urine	1	44.0	100.0	Combined test estimate*

Number of studies indicates the total count of studies included in the review that used each diagnostic test and specimen combination for the detection of a specific pathogen. *Combined sensitivity and specificity estimate determined for studies reporting two diagnostic tests to identify a pathogen. If either test could be positive, the lower sensitivity and lower specificity values were used from the two tests. If both tests had to be positive, the lower sensitivity and higher specificity values were used from the two tests. CT: *Chlamydia trachomatis*, MG: *Mycoplasma genitalium*, NG: *Neisseria gonorrhoeae*, TV: *Trichomonas vaginalis*.

Table B7: Performance of diagnostic tests among adults with genital ulcer

RTI	Test	Specimen	Number studies	Sensitivity (%)	Specificity (%)	Source
HD	Culture	Genital ulcer	30	73.0	100.0	WHO 1999[145]
	NAAT	Genital ulcer	28	95.1	99.9	Paz-Bailey 2005[630]
	Culture or NAAT	Genital ulcer	1	73.0	99.9	Combined test estimate*
	Culture and NAAT	Genital ulcer	1	73.0	100.0	Combined test estimate*
HSV	Culture	Genital ulcer	16	75.0	100.0	Legoff 2014[694]
	DFA	Genital ulcer	1	80.0	95.0	Legoff 2014[694]
	NAAT	Genital ulcer	14	98.0	100.0	Legoff 2014[694]
HSV-1	DFA	Genital ulcer	1	80.0	95.0	Legoff 2014[694]
	NAAT	Genital ulcer	14	99.0	99.0	Arshad 2019[153]
HSV-2	DFA	Genital ulcer	2	80.0	95.0	Legoff 2014[694]
	NAAT	Genital ulcer	21	100.0	98.5	Arshad 2019[153]
LGV	DFA	Genital ulcer	2	95.5	99.4	Diagnostic Hybrids 2008[695]
	NAAT	Genital ulcer	8	95.5	98.5	Touati 2021[696]
TP	Darkfield	Genital ulcer	10	80.0	98.5	WHO 1999[145]
	DFA	Genital ulcer	1	86.5	94.5	Peeling 2004[152]
	NAAT	Genital ulcer	30	91.0	99.0	WHO 1999[145]
	Non-treponemal	Blood	6	96.6	97.5	Mean sensitivity and specificity, from Hambie 1983,[697] Perryman 1982,[698] and Parham 1984,[699] regardless of titre due to varied reporting
	Treponemal	Blood	3	93.1	99.4	Mean sensitivity and specificity from Cantor 2016,[700] Sena 2010,[701] and Park 2019[702]
	Non-treponemal and treponemal	Blood	20	100.0	100.0	Estimated performance; non-treponemal test performance regardless of titre due to varied reporting
	Darkfield or treponemal	Genital ulcer and blood	1	80.0	95.7	Combined test estimate*
	Darkfield, or non-treponemal and treponemal	Genital ulcer and blood	1	80.0	98.5	Combined test estimate*
	NAAT, or non-treponemal and treponemal	Genital ulcer and blood	1	91.0	99.0	Combined test estimate*

Number of studies indicates the total count of studies included in the review that used each diagnostic test and specimen combination for the detection of a specific pathogen. *Combined sensitivity and specificity estimate determined for studies reporting two diagnostic tests to identify a pathogen. If either test could be positive, the lower sensitivity and lower specificity values were used from the two tests. If both tests had to be positive, the lower sensitivity and higher specificity values were used from the two tests. HD: *Haemophilus ducreyi*, HSV: Herpes simplex virus, HSV-1: Herpes simplex virus type 1, HSV-2: Herpes simplex virus type 2, LGV: Lymphogranuloma venereum, TP: *Treponema pallidum*.

B.1.4 Risk of bias

Table B8: Risk of bias criteria

Criteria	Category	Definition of yes
1. Was the study specifically designed to estimate the outcome/s of interest?	Study design	The primary objective of the study was to determine sexually transmitted / reproductive tract infection aetiology among individuals symptomatic with genital ulcer, vaginal discharge and/or urethral discharge.
2. Were the criteria for inclusion in the sample clearly defined?	Selection bias	The inclusion and exclusion criteria clearly define the population being recruited into the study. This includes their sex, age group (age range, or classified as youth or adult), and population type (e.g. general population, key population, symptomatic clinic attendee).
3. Were study participants and their setting described in sufficient detail to determine representativeness of the target population?	Selection bias	There is a clear description of study participant characteristics, including their sex, age group and HIV status. The study site locations are described. The name of the health facility is reported, or details of the facility are described in terms of urban/rural and sector (private, government, non-profit) classification.
4. Were study participants recruited in an appropriate way?	Selection bias	The study used either consecutive or random sampling methods for participant recruitment. If it is claimed that a random sample was chosen, the method of randomization should be stated. It is acceptable if studies do not say 'consecutive' but instead describe consecutive enrolment, i.e., 'all patients from .... till .... were included'.
5. Was the participation rate adequate?	Selection bias	The participation rate in the study was at least 80%. Participation is defined as providing specimens for STI testing, among those identified as having symptoms.
6. Were objective, standard criteria used to define participant symptoms?	Measurement bias	There is a clear, consistently applied definition to identify participants with the symptom(s) of focus in the study. Specifically, all participants were examined by a healthcare provider to identify their symptoms. The classification of symptoms is not based solely on participant self-reporting.
7. Were the outcomes measured in a reliable and consistent way?	Measurement bias	All data were collected directly from the participants. Proxy data (i.e. medical records) were not used in any instances. The same mode of sample collection and diagnosis was used for all participants.
8. Were multiple pathogens assessed to prevent potential misdiagnoses?	Measurement bias	Symptomatic participants were tested for at least 3 different sexually transmitted / reproductive tract infection pathogens.
9. Was the sample size sufficient to measure the outcome?	Study precision	The sample size is justified through a calculation, or the sample includes at least 100 participants.
10. Was the reporting of the outcome(s) unambiguous?	Reporting quality	The numerators, denominator(s), and proportion are clearly reported for each infection. The results are internally consistent. The reported proportion was not different when calculated from reported numerators and denominators.

Criteria adapted from the Joanna Briggs Institute critical appraisal tool for prevalence studies.[289]

Table B9: Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) guideline checklist

Topic	Item	Checklist item	Location
Title			
Title	1	Identify the report as a systematic review.	Title
Abstract			
Abstract	2	See the PRISMA 2020 for Abstracts checklist.	Abstract
Introduction			
Rationale	3	Describe the rationale for the review in the context of existing knowledge.	Introduction
Objectives	4	Provide an explicit statement of the objective(s) or question(s) the review addresses.	Introduction
Methods			
Eligibility criteria	5	Specify the inclusion and exclusion criteria for the review and how studies were grouped for the syntheses.	Methods
Information sources	6	Specify all databases, registers, websites, organisations, reference lists and other sources searched or consulted to identify studies. Specify the date when each source was last searched or consulted.	Methods
Search strategy	7	Present the full search strategies for all databases, registers and websites, including any filters and limits used.	Methods; Tables B1, B2
Selection process	8	Specify the methods used to decide whether a study met the inclusion criteria of the review, including how many reviewers screened each record and each report retrieved, whether they worked independently, and if applicable, details of automation tools used in the process.	Methods
Data collection process	9	Specify the methods used to collect data from reports, including how many reviewers collected data from each report, whether they worked independently, any processes for obtaining or confirming data from study investigators, and if applicable, details of automation tools used in the process.	Methods; Section B.1.2; Table B4
Data items	10a	List and define all outcomes for which data were sought. Specify whether all results that were compatible with each outcome domain in each study were sought (e.g. for all measures, time points, analyses), and if not, the methods used to decide which results to collect.	Methods
	10b	List and define all other variables for which data were sought (e.g. participant and intervention characteristics, funding sources). Describe any assumptions made about any missing or unclear information.	Methods; Table B3
Study risk of bias assessment	11	Specify the methods used to assess risk of bias in the included studies, including details of the tool(s) used, how many reviewers assessed each study and whether they worked independently, and if applicable, details of automation tools used in the process.	Methods; Table B8
Effect measures	12	Specify for each outcome the effect measure(s) (e.g. risk ratio, mean difference) used in the synthesis or presentation of results.	Methods

Continued...

Topic	Item	Checklist item	Location
Synthesis methods	13a	Describe the processes used to decide which studies were eligible for each synthesis (e.g. tabulating the study intervention characteristics and comparing against the planned groups for each synthesis (item #5)).	Methods
	13b	Describe any methods required to prepare the data for presentation or synthesis, such as handling of missing summary statistics, or data conversions.	Methods; Section B.1.2 and B.1.3; Tables B5, B6, B7
	13c	Describe any methods used to tabulate or visually display results of individual studies and syntheses.	Methods
	13d	Describe any methods used to synthesize results and provide a rationale for the choice(s). If meta-analysis was performed, describe the model(s), method(s) to identify the presence and extent of statistical heterogeneity, and software package(s) used.	Methods
	13e	Describe any methods used to explore possible causes of heterogeneity among study results (e.g. subgroup analysis, meta-regression).	Methods
	13f	Describe any sensitivity analyses conducted to assess robustness of the synthesized results.	Methods
Reporting bias assessment	14	Describe any methods used to assess risk of bias due to missing results in a synthesis (arising from reporting biases).	Not applicable
Certainty assessment	15	Describe any methods used to assess certainty (or confidence) in the body of evidence for an outcome.	Not applicable
Results			
Study selection	16a	Describe the results of the search and selection process, from the number of records identified in the search to the number of studies included in the review, ideally using a flow diagram.	Results, Figure 3.1
	16b	Cite studies that might appear to meet the inclusion criteria, but which were excluded, and explain why they were excluded.	Figure 3.1, Section B.1.2
Study characteristics	17	Cite each included study and present its characteristics.	Table B10; Online supplementary data
Risk of bias in studies	18	Present assessments of risk of bias for each included study.	Online supplementary data
Results of individual studies	19	For all outcomes, present, for each study: (a) summary statistics for each group (where appropriate) and (b) an effect estimate and its precision (e.g. confidence/credible interval), ideally using structured tables or plots.	Online supplementary data
Results of syntheses	20a	For each synthesis, briefly summarise the characteristics and risk of bias among contributing studies.	Results; Tables 3.1, B11, B12, B13, B18; Figure B1
	20b	Present results of all statistical syntheses conducted. If meta-analysis was done, present for each the summary estimate and its precision (e.g. confidence/credible interval) and measures of statistical heterogeneity. If comparing groups, describe the direction of the effect.	Results; Figures 3.2, 3.3, 3.4; Tables 3.2, B14, B15, B16

Continued...

Topic	Item	Checklist item	Location
	20c	Present results of all investigations of possible causes of heterogeneity among study results.	Figures B2, B5; Tables B14, B15, B16
	20d	Present results of all sensitivity analyses conducted to assess the robustness of the synthesized results.	Results; Figures B5, B3, B4; Tables B18, B17
Reporting biases	21	Present assessments of risk of bias due to missing results (arising from reporting biases) for each synthesis assessed.	Not applicable
Certainty of evidence	22	Present assessments of certainty (or confidence) in the body of evidence for each outcome assessed.	Not applicable
Discussion			
Discussion	23a	Provide a general interpretation of the results in the context of other evidence.	Discussion
	23b	Discuss any limitations of the evidence included in the review.	Discussion
	23c	Discuss any limitations of the review processes used.	Discussion
	23d	Discuss implications of the results for practice, policy, and future research.	Discussion
Other information			
Registration and protocol	24a	Provide registration information for the review, including register name and registration number, or state that the review was not registered.	Methods
	24b	Indicate where the review protocol can be accessed, or state that a protocol was not prepared.	Methods
	24c	Describe and explain any amendments to information provided at registration or in the protocol.	Not applicable
Support	25	Describe sources of financial or non-financial support for the review, and the role of the funders or sponsors in the review.	Online publication
Competing interests	26	Declare any competing interests of review authors.	Online publication
Availability of data, code, other materials	27	Report which of the following are publicly available and where they can be found: template data collection forms; data extracted from included studies; data used for all analyses; analytic code; any other materials used in the review.	Online publication

B.2 Supplementary results

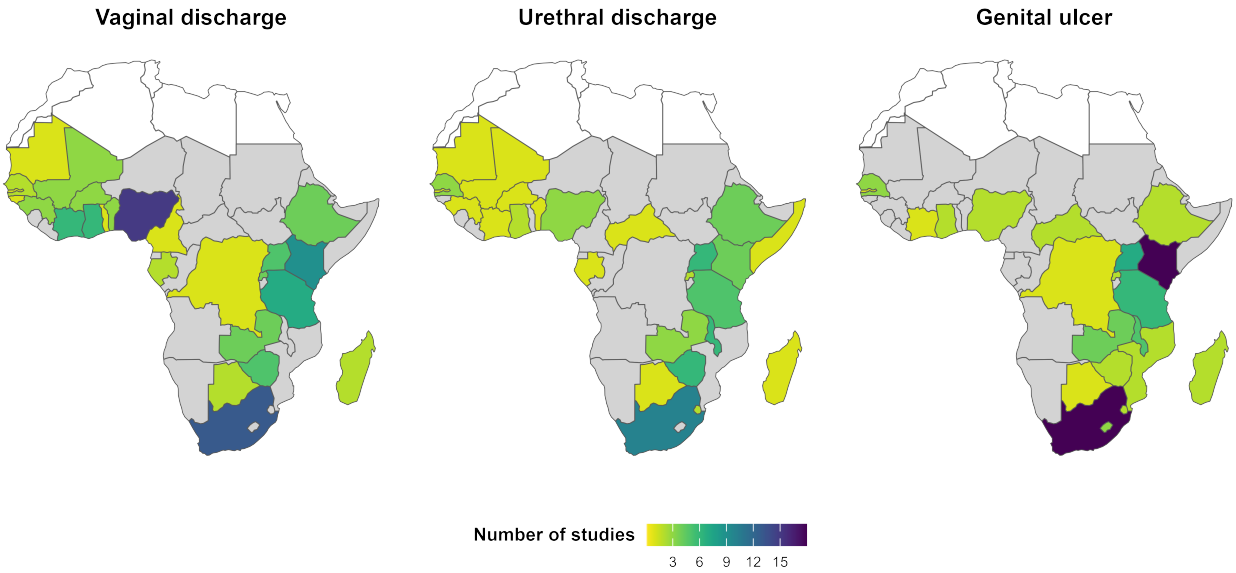


Figure B1: Number of studies included per country in SSA

Number of studies among individuals with vaginal discharge, urethral discharge, and genital ulcer that were included in the systematic review. Total included studies were 87 for vaginal discharge, 55 for urethral discharge, and 80 for genital ulcer. Base map source is Natural Earth.[465]

Table B10: Overview of studies included in the systematic review

Reference	Year	Country	Population	Sex	Age	HIV status	Infections extracted
Vaginal discharge							
Rubin 1980[703]	1978	South Africa	ANC attendees	F	NR	NR	CA, NG, TV, None
Hoosen 1981[704]	1981*	South Africa	ANC attendees	F	NR	NR	CS, NG, TV
Mirza 1983[705]	1982	Kenya	Symptomatic clinic attendees	F	NR	NR	CA, CT, NG, TV
Mabey 1984[706]	1982	Gambia	ANC attendees	F	16 to 40	NR	TV
Leclerc 1988[707]	1988*	Gabon	Symptomatic clinic attendees	F	NR	NR	CT
Faye-Kette 1993[708]	1989	Côte d'Ivoire	Symptomatic clinic attendees	F	17 to 35	NR	CS, NG, TV
Alary 1998[709]	1994	Benin	Symptomatic clinic attendees	F	NR	Non-stratified	CS, CT, NG, TV
Thomas 1996[710]	1994	Kenya	ANC attendees	F	NR	Non-stratified	BV, CS, TV
Schneider 1998[711]	1994	South Africa	FP attendees	F	NR	NR	TV
Mayaud 1997[712]	1994	Tanzania	ANC attendees	F	NR	NR	NG, TV
Gateau 1996[713]	1995	Madagascar	FSW	F	14 to 49	NR	TV
Latif 1999[714]	1995	Zimbabwe	Symptomatic clinic attendees, non-pregnant	F	NR	Non-stratified	CS, CT, NG, TV
Farina 2000[715]	1996	Comoros Islands	Symptomatic clinic attendees	F	NR	NR	CA
Hoosen 1997[716]	1997*	South Africa	Symptomatic clinic attendees	F	NR	Non-stratified	BV, CS, CT, NG, TV
Gomes 2001[717]	1997	Guinea-Bissau	Symptomatic clinic attendees	F	15 to 58	NR	CT, NG
Fonck 2001[191]	1997	Kenya	Symptomatic clinic attendees	F	15 to 52	Non-stratified	BV, CS, CT, NG, TV, None
Anorlu 2001[718]	1998	Nigeria	Symptomatic clinic attendees, non-pregnant	F	16 to 45	NR	TV
Msuya 2002[719]	1999	Tanzania	ANC/FP/PED attendees	F	16 to 46	NR	CT
Mbizvo 2004[720]	1999	Zimbabwe	ANC/FP/PED attendees	F	15 to 49	Non-stratified	BV
Vuylsteke 2003[721]	2000	Côte d'Ivoire	FSW	F	18 to 53	NR	BV, CS, TV
Anorlu 2004[722]	2000	Nigeria	Symptomatic clinic attendees	F	18 to 50	Non-stratified	CS, TV
Moodley 2002[723]	2000	South Africa	Symptomatic clinic attendees	F	NR	Non-stratified	BV, CT, NG, TV
Pepin 2004[724]	2001	Benin, Burkina Faso, Ghana, Guinea, Mali	Non-pregnant, non-FSW; Symptomatic clinic attendees	F	12+	NR	CT, NG, TV
Bankole 2001[725]	2001*	Côte d'Ivoire	Symptomatic clinic attendees	F	NR	NR	CT

Continued...

Reference	Year	Country	Population	Sex	Age	HIV status	Infections extracted
Romoren 2007[726]	2001	Botswana	ANC attendees	F	15 to 43	NR	BV, TV
Otuonye 2004[727]	2002	Nigeria	Symptomatic clinic attendees	F	15 to 50	Non-stratified	CS, NG, TV, None
Namkinga 2005[728]	2002	Tanzania	Symptomatic clinic attendees	F	NR	Non-stratified	CS
Msuya 2009[729]	2003	Tanzania	ANC attendees	F	14 to 43	Non-stratified	BV, CS, TV
Abauleth 2006[730]	2004	Côte d'Ivoire	Symptomatic clinic attendees	F	19 to 49	NR	CS, NG, TV
Onyekonwu 2011[731]	2004	Nigeria	Symptomatic clinic attendees	F	NR	NR	BV, CS, NG, TV
Pepin 2011[732]	2005	Ghana, Guinea, Mali, Togo	Symptomatic clinic attendees	F	NR	Non-stratified	BV, CS, CT, MG, NG, TV
Pepin 2006[733]	2005	Ghana, Guinea, Mali, Togo	Symptomatic clinic attendees	F	11+	Non-stratified	BV, CS, CT, MG, NG, TV
Ojuromi 2007[734]	2006	Nigeria	Symptomatic clinic attendees	F	16 to 57	NR	CS, TV
Nwadioha 2011[735]	2007	Nigeria	Symptomatic clinic attendees	F	0 to 60	Non-stratified	BV
Nwadioha 2011[736]	2007	Nigeria	Symptomatic clinic attendees	F	0 to 60	NR	BV, CS, NG, TV, None
Nwadioha 2012[737]	2007	Nigeria	Symptomatic clinic attendees	F	0 to 60	Non-stratified	TV
Muvunyi 2009[738]	2007	Rwanda	Symptomatic clinic attendees	F	16 to 57	NR	TV
Djohan 2012[739]	2008	Côte d'Ivoire	Symptomatic clinic attendees	F	NR	NR	CS
Nwadioha 2010[740]	2008	Nigeria	Symptomatic clinic attendees	F	0 to 60	Non-stratified	BV, CS, NG, TV, None
Nwadioha 2010[741]	2008	Nigeria	Symptomatic clinic attendees	F	0 to 60	Non-stratified	CA
Karou 2012[742]	2009	Burkina Faso	Symptomatic clinic attendees	F	19 to 45	NR	CA
Okunlola 2009[743]	2009	Nigeria	IUD users; Symptomatic clinic attendees	F	28 to 51	NR	BV, CS, NG, TV
Nwadioha 2013[744]	2009	Nigeria	Symptomatic clinic attendees	F	0 to 60	NR	CA, CS
Tine 2019[745]	2009	Senegal	Symptomatic clinic attendees	F	18+	NR	TV
Vandepitte 2012[746]	2009	Uganda	FSW or entertainment facility workers	F	NR	Non-stratified	MG
De Jongh 2010[638]	2010*	South Africa	Women receiving TOP	F	13 to 41	NR	CT, NG, TV, None
Konate 2014[747]	2011	Côte d'Ivoire	Symptomatic clinic attendees	F	NR	NR	CS
Ibrahim 2013[748]	2011	Nigeria	ANC attendees; Symptomatic clinic attendees	F	15 to 42	NR	CA
Ibrahim 2014[749]	2011	Nigeria	ANC attendees; Symptomatic clinic attendees	F	15 to 42	NR	BV

Continued...

Reference	Year	Country	Population	Sex	Age	HIV status	Infections extracted
Van der Eem 2016[750]	2012	South Africa	Sexually active PHC attendees	F	18 to 49	Non-stratified	CT, MG, NG, TV, None
Kerubo 2016[578]	2013	Kenya	School girls	F	14 to 17	NR	BV
Tine 2019[751]	2013	Senegal	Symptomatic clinic attendees	F	18+	NR	TV
Nkwabong 2015[752]	2014	Cameroon	Women with acute PID; Symptomatic clinic attendees	F	15 to 48	NR	CT, NG, TV
Asmah 2018[753]	2014	Ghana	Clients referred for STI symptoms; Symptomatic clinic attendees	F	16 to 50	NR	TV
Konadu 2019[505]	2015	Ghana	ANC attendees	F	12 to 54	NR	BV, CS
Oyeyipo 2015[754]	2015	Nigeria	Non-pregnant symptomatic clinic attendees	F	17 to 31	NR	CS
Odunvbun 2018[755]	2015	Nigeria	Infertile GYN attendees	F	20 to 43	NR	CT
Marcotte 2019[756]	2015	South Africa	Sexually active women; Symptomatic clinic attendees	F	18 to 40	HIV-negative	BV
Nabweyambo 2017[757]	2015	Uganda	Symptomatic clinic attendees	F	18 to 60	NR	TV
Chirenje 2018[758]	2015	Zimbabwe	Symptomatic clinic attendees	F	18 to 53	Non-stratified	BV, CT, MG, NG, TV, None
Gadoth 2020[759]	2017	Democratic Republic of the Congo	ANC attendees	F	18+	Non-stratified	TV
Aduloju 2019[760]	2017	Nigeria	ANC attendees; Symptomatic clinic attendees	F	NR	NR	BV
Kashibu 2018[513]	2017	Nigeria	ANC attendees	F	15 to 39	NR	TV
Chabata 2023[761]	2017	Zimbabwe	FSW	F	18 to 50	Non-stratified	CT, NG, TV, None
Happel 2020[762]	2018	South Africa	Non-pregnant, non-breastfeeding symptomatic clinic attendees	F	18 to 45	HIV-negative	BV, CT, MG, NG, TV, None
Connolly 2020[763]	2018	Zambia	FSW or single mothers	F	18 to 45	Non-stratified	CT, NG
Omosa-Manyonyi 2023[764]	2019	Kenya	Symptomatic clinic attendees	F	18 to 50	HIV-negative	BV, CS, CT, MG, NG, TV, None
Pulei 2023[765]	2019	Kenya	ANC/GYN/FP attendees	F	18 to 45	HIV-negative	CT
Mabaso 2020[766]	2019	South Africa	ANC attendees	F	18 to 39	NR	TV
Zenebe 2021[617]	2020	Ethiopia	ANC attendees	F	NR	NR	CT, NG, TV
Mabaso 2022[670]	2020	South Africa	ANC attendees	F	18+	NR	CT

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Reference	Year	Country	Population	Sex	Age	HIV status	Infections extracted
Ahmed 2022[767]	2021	Ethiopia	ANC attendees	F	17 to 43	NR	BV
Urethral discharge							
Arya 1972[768]	1969	Uganda	University students	M	NR	NR	NG, TV
Fakunle 1976[769]	1976*	Nigeria	Soldiers	M	NR	NR	NG
Ballard 1977[770]	1977*	South Africa	Symptomatic clinic attendees	M	NR	NR	NG
Meheus 1980[771]	1979	Eswatini	Symptomatic clinic attendees	M	NR	NR	NG
Mabey 1982[772]	1982*	Gambia	Symptomatic clinic attendees	M	NR	NR	CT, NG
Ismail 1990[773]	1986	Somalia	Symptomatic clinic attendees	M	15 to 42	HIV-negative	CT, NG
Meiring 1989[774]	1986	South Africa	Symptomatic clinic attendees	M	NR	NR	NG
Grosskurth 1996[775]	1992	Tanzania	Men in community	M	NR	NR	CT, NG
Tswana 1995[776]	1992	Zimbabwe	Symptomatic clinic attendees	M	15 to 55	Non-stratified	NG
Dallabetta 1998[777]	1993	Malawi	Symptomatic clinic attendees	M	13 to 70	NR	CT, NG
Pillay 1994[778]	1994*	South Africa	Symptomatic clinic attendees	M	NR	NR	NG, TV
Ramuthaga 1995[779]	1995*	South Africa	Symptomatic clinic attendees	M	17 to 58	NR	CT
Jackson 1997[780]	1995	Kenya	Truck drivers	M	18 to 57	Non-stratified	CT, NG, TV, None
Bakare 2002[781]	1995	Nigeria	Symptomatic clinic attendees	M	NR	NR	CT, NG, TV, None
Cohen 1997[782]	1996	Malawi	Symptomatic clinic attendees	M	NR	Non-stratified	CT, NG, TV
Dieye 2003[783]	1996	Senegal	Symptomatic clinic attendees	M	NR	NR	CT, NG
Pepin 2001[784]	1997	Benin, Burkina Faso, Cote d'Ivoire, Ghana, Guinea, Mali, Senegal	Symptomatic clinic attendees	M	NR	NR	CT, MG, NG, TV
Morency 2001[785]	1997	Central African Republic	Symptomatic clinic attendees	M	NR	NR	CT, MG, NG, TV, None
Price 2004[786]	2000	Malawi	Symptomatic clinic attendees	M	18+	Non-stratified	NG, TV
Bakare 2002[787]	2000	Nigeria	Symptomatic clinic attendees	M	NR	NR	NG, TV
Watson-Jones 2000[44]	2000*	Tanzania	Men in community	M	NR	NR	TV
Mason 2000[788]	2000*	Zimbabwe	Symptomatic clinic attendees	M	NR	NR	CT, NG, TV
Zachariah 2002[789]	2001	Malawi	Symptomatic clinic attendees	M	16 to 47	NR	CT, NG
Tadesse 2001[790]	2001*	Ethiopia	Symptomatic clinic attendees	M	¡64	NR	NG
De Jongh 2009[791]	2009*	South Africa	Symptomatic clinic attendees	M	15 to 73	NR	CT, NG, TV, None

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Reference	Year	Country	Population	Sex	Age	HIV status	Infections extracted
Brown 2010[792]	2007	Malawi	Symptomatic clinic attendees	M	NR	NR	NG
Moodley 2013[793]	2007	South Africa	Symptomatic clinic attendees	M	15 to 55	Non-stratified	CT, NG
Le Roux 2010[794]	2008	South Africa	Symptomatic clinic attendees	M	19 to 64	NR	CT, MG, NG, TV, None
Takuva 2014[795]	2011	Zimbabwe	Symptomatic clinic attendees	M	18+	NR	CT, MG, NG, TV
Mulu 2017[796]	2013	Ethiopia	Symptomatic clinic attendees	M	NR	NR	NG
Fentaw 2020[797]	2014	Ethiopia	Symptomatic clinic attendees	M	18+	NR	NG
Rietmeijer 2018[798]	2015	Zimbabwe	Symptomatic clinic attendees	M	18+	Non-stratified	CT, MG, NG, TV, None
Van Liere 2019[799]	2016	South Africa	Men who have sex with men	M	NR	Non-stratified	CT, MG, NG, TV, None
Latif 2018[800]	2016	Zimbabwe	Symptomatic clinic attendees	M	18 to 63	NR	NG
Nacht 2020[801]	2018	Kenya	Symptomatic clinic attendees	M	NR	Non-stratified	NG
Matoga 2023[802]	2018	Malawi	Symptomatic clinic attendees	M	NR	HIV-positive	CT, NG, TV, None
Melendez 2022[127]	2020	Uganda	Symptomatic clinic attendees	M	NR	Non-stratified	CT, MG, NG, TV, None
Beck 2023[803]	2020	Uganda	Symptomatic clinic attendees	M	NR	HIV-positive	CT, NG, TV, None
Genital ulcer							
Ongom 1971[804]	1970	Uganda	Symptomatic clinic attendees	M, F	NR	NR	TP
Meheus 1983[805]	1979	Eswatini	Symptomatic clinic attendees	M, F	NR	NR	HSV, TP
Ursi 1981[806]	1979	Eswatini	Symptomatic clinic attendees	M, F	15 to 50	NR	TP
Nsanze 1981[807]	1980	Kenya	Symptomatic clinic attendees	M, F	NR	NR	HD, HSV, TP, None
Fast 1983[808]	1981	Kenya	Symptomatic clinic attendees	M, F	15 to 40	NR	HD, TP
Crewe-Brown 1982[809]	1981	South Africa	Symptomatic clinic attendees	M	16 to 59	NR	HD, HSV, TP
D'Costa 1985[810]	1982	Kenya	FSW	F	NR	NR	HD
Plummer 1983[811]	1983*	Kenya	Symptomatic clinic attendees	M	18 to 60	NR	HD, HSV
Mauff 1983[812]	1983*	South Africa	Symptomatic clinic attendees	M	NR	NR	HD, TP
Coovadia 1985[813]	1984	South Africa	Symptomatic clinic attendees	M	17 to 51	NR	HD, HSV, TP, None
Plummer 1985[814]	1985*	Kenya	Symptomatic clinic attendees	F	NR	NR	HD, HSV, TP
Greenblatt 1988[815]	1985	Kenya	Symptomatic clinic attendees	M	18 to 70	Non-stratified	HD, HSV, TP, None
Kreiss 1986[816]	1985	Kenya	FSW	F	NR	NR	HD
Dangor 1989[817]	1986	South Africa	Mineworkers	M	19 to 59	NR	HD, HSV, TP, None
Mabey 1987[818]	1987*	Gambia	Symptomatic clinic attendees	M, F	NR	NR	HD, HSV, TP, None

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Reference	Year	Country	Population	Sex	Age	HIV status	Infections extracted
Bogaerts 1998[819]	1989	Rwanda	Symptomatic clinic attendees	M, F	NR	Non-stratified	HD, HSV, TP, None
O'Farrell 1994[820]	1989	South Africa	Symptomatic clinic attendees	M, F	14 to 58	NR	HD, HSV, LGV, TP, None
Daly 1994[821]	1990	Kenya	FP Symptomatic clinic attendees	F	15 to 49	Non-stratified	TP
Thior 1997[822]	1990	Senegal	Symptomatic clinic attendees	M	18+	Non-stratified	HD, TP
Ndinya-Achola 1996[823]	1991	Kenya	Symptomatic clinic attendees	M, F	NR	NR	HD, TP
Kamya 1995[824]	1991	Uganda	Symptomatic clinic attendees	M, F	NR	Non-stratified	HSV-1, HSV-2, TP, None
Kimani 1995[825]	1992	Kenya	Symptomatic clinic attendees	M	NR	Non-stratified	HD, TP
Oni 1997[826]	1992	Nigeria	Symptomatic clinic attendees	M	15+	NR	HSV
Totten 2000[827]	1992	Senegal	Symptomatic clinic attendees	M, F	21 to 53	NR	HD, HSV, TP, None
Htun 2007[828]	1992	South Africa	Mineworkers	M	NR	Non-stratified	HD, HSV, TP, None
Ghys 1995[829]	1993	Côte d'Ivoire	FSW	F	NR	HIV-positive	HD, HSV
Behets 1995[830]	1993	Malawi	Symptomatic clinic attendees	M	NR	Non-stratified	HD, TP
Cossa 1994[831]	1993	Mozambique	Displaced pregnant women	F	14 to 45	Non-stratified	TP
King 1998[832]	1994	Kenya	Symptomatic clinic attendees	M	NR	Non-stratified	HD
Morse 1997[833]	1994	Lesotho	Symptomatic clinic attendees	M, F	NR	Non-stratified	HD, HSV, TP, None
Htun 1998[834]	1994	Lesotho	Symptomatic clinic attendees	M, F	NR	NR	HD, HSV, LGV, TP, None
Chen 2000[835]	1994	South Africa	Symptomatic clinic attendees	M	15 to 65	Non-stratified	HD, HSV-2, TP, None
Aseffa 1998[836]	1995	Ethiopia	ANC/GYN/FP attendees	F	NR	Non-stratified	TP
MacDonald 2001[837]	1995	Kenya	Symptomatic clinic attendees	M	NR	Non-stratified	HD, TP
Ahmed 1995[838]	1995*	Zambia	Symptomatic clinic attendees	M, F	16 to 46	NR	HD
Malonza 1999[839]	1996	Kenya	Symptomatic clinic attendees	M, F	18 to 75	Non-stratified	HD, HSV, TP, None
Ballard 1996[840]	1996*	South Africa	Migrant mineworkers	M	NR	Non-stratified	HD
Lai 2003[841]	1996	South Africa	Symptomatic clinic attendees	M	20 to 58	Non-stratified	HD, HSV, TP, None
Ballard 2007[842]	1997	Lesotho, South Africa	Symptomatic clinic attendees	M, F	NR	Non-stratified	HD, HSV, TP, None
Behets 1999[843]	1997	Madagascar	Symptomatic clinic attendees	M, F	18+	NR	HD, HSV, TP, None
Rajagopal 1999[844]	1997	South Africa	Symptomatic clinic attendees	F	NR	Non-stratified	TP
Ramjee 1998[845]	1997	South Africa	FSW	F	17 to 41	Non-stratified	HD
Paz-Bailey 2005[630]	1998	Botswana	Symptomatic clinic attendees	M, F	NR	Non-stratified	HD, HSV, HSV-1, HSV-2, TP, None

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Reference	Year	Country	Population	Sex	Age	HIV status	Infections extracted
Nzila 1991[846]	1998	Democratic Republic of the Congo	FSW	F	NR	Non-stratified	HD, HSV
Ballard 2002[847]	1998	South Africa	Migrant mineworkers	M	NR	Non-stratified	HD, HSV-2, TP, None
Hoyo 2005[848]	1999	Malawi	Symptomatic clinic attendees	M, F	NR	NR	HD, HSV, TP, None
Ahmed 2003[849]	1999	Tanzania	Symptomatic clinic attendees	M, F	14 to 52	Non-stratified	HD, HSV-2, TP, None
Nilsen 2007[850]	2000	Tanzania	Symptomatic clinic attendees	M, F	17 to 60	Non-stratified	HD, HSV-1, HSV-2, TP, None
Riedner 2007[851]	2000	Tanzania	Female bar workers	F	NR	Non-stratified	HD, HSV, TP, None
Moodley 2003[852]	2001	South Africa	Symptomatic clinic attendees	M, F	NR	Non-stratified	HD, HSV-2, LGV, TP, None
Mwansasu 2002[853]	2002*	Tanzania	Symptomatic clinic attendees	M, F	NR	Non-stratified	HSV-1, HSV-2
Fayemiwo 2011[854]	2003	Nigeria	FSW	F	15 to 55	Non-stratified	HD, TP
Weiss 2011[855]	2004	Central African Republic, Ghana	Symptomatic clinic attendees	F	18+	Non-stratified	HSV-1, HSV-2
Legoff 2007[856]	2004	Central African Republic, Ghana	Symptomatic clinic attendees	F	15+	Non-stratified	HD, HSV-2, TP, None
Mehta 2012[857]	2004	Kenya	Uncircumcised, sexually active men	M	18 to 24	HIV-negative	HD, TP, None
O'Farrell 2008[858]	2004	South Africa	Symptomatic clinic attendees	M	16 to 75	Non-stratified	HD, HSV-2, LGV, TP, None
Suntoke 2009[859]	2004	Uganda	Rakai Community Cohort; Symptomatic clinic attendees	M, F	19+	Non-stratified	HD, HSV-1, HSV-2, TP, None
Phiri 2013[860]	2005	Malawi	Symptomatic clinic attendees	M, F	18+	Non-stratified	HD, HSV-2, LGV, TP, None
Zimba 2011[861]	2005	Mozambique	Symptomatic clinic attendees	M, F	NR	Non-stratified	HD, HSV-2, LGV, TP, None
Brankin 2009[862]	2005	Uganda	Wives or long-term partners of men in circumcision trial	F	15 to 49	Non-stratified	HD, HSV-1, HSV-2, TP, None
Paz-Bailey 2009[863]	2006	South Africa	Symptomatic clinic attendees	M	18 to 60	Non-stratified	HD, HSV-1, HSV-2, LGV, TP, None
Paz-Bailey 2010[864]	2006	South Africa	Symptomatic clinic attendees	M	NR	HIV-positive	HSV, TP, None
Vandenhoudt 2013[383]	2008	Kenya	FSW	F	NR	Non-stratified	HD, HSV-1, HSV-2, TP, None
Vandepitte 2011[865]	2009	Uganda	FSW or entertainment facility workers	F	NR	Non-stratified	HD, HSV-1, HSV-2, LGV, TP, None
Singa 2013[866]	2010	Kenya	HIV clinic attendees; Symptomatic clinic attendees	M, F	18 to 70	HIV-positive	HSV-1, HSV-2, TP, None
Makasa 2012[361]	2010	Zambia	Symptomatic clinic attendees	M, F	NR	Non-stratified	HD, HSV-1, HSV-2, TP, None

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Reference	Year	Country	Population	Sex	Age	HIV status	Infections extracted
Sakala 2016[867]	2015	Zambia	ANC attendees	F	NR	Non-stratified	TP
Rietmeijer 2019[868]	2015	Zimbabwe	Symptomatic clinic attendees	M, F	18 to 55	Non-stratified	TP
Kilmarx 2018[869]	2015	Zimbabwe	Symptomatic clinic attendees	M, F	NR	Non-stratified	HSV-2
Mungati 2018[870]	2015	Zimbabwe	Symptomatic clinic attendees	M, F	18+	Non-stratified	HD, HSV, HSV-1, LGV, TP, None
Chen 2020[871]	2017	Malawi	Symptomatic clinic attendees	M, F	NR	Non-stratified	TP
Mehta 2017[872]	2017*	Kenya	Symptomatic clinic attendees	F	18 to 59	Non-stratified	HSV-1, HSV-2, TP, None
Tshaka 2022[873]	2019	South Africa	Symptomatic clinic attendees	M, F	16 to 57	Non-stratified	HD, HSV-1, HSV-2, LGV, TP, None
Chen 2023[367]	2021	Malawi	Symptomatic clinic attendees	M, F	18+	Non-stratified	HD, HSV, TP, None
Multiple symptoms							
Van Binsbergen 1980[874]	1976	Kenya	Symptomatic clinic attendees	M, F	NR	NR	**VD**: CA, NG, TV; **UD**: NG, TV
Meheus 1982[875]	1978	Eswatini	Symptomatic clinic attendees	M, F	NR	NR	**UD**: NG; **GU**: TP
Yvert 1984[876]	1981	Gabon	ANC attendees	M, F	15+	NR	**VD:** CS, NG, TV; **UD:** NG, TV
Hanson 1996[877]	1991	Zambia	Symptomatic clinic attendees	M, F	NR	Non-stratified	**VD:** CS, CT, NG, TV **UD:** NG; **GU:** TP
LeBacq 1993[878]	1991	Zimbabwe	Men and non-pregnant women	M, F	10+	Non-stratified	**VD:** CS, CT, NG, TV **UD:** CT, NG, TV **GU:** HD, HSV, TP
Meless 1995[879]	1993	Ethiopia	Symptomatic clinic attendees	M, F	15+	NR	**UD:** NG; **VD:** NG
Harms 1994[880]	1993	Madagascar	Symptomatic clinic attendees	M, F	14 to 52	NR	**VD:** BV, CS, CT, NG, TV, None **UD:** CT, NG, TV, None **UD:** HD, HSV-2, LGV, TP, None
Lo 1997[881]	1994	Mauritania	Symptomatic clinic attendees	M, F	NR	NR	**VD:** CS, CT, NG, TV, None **UD:** CT, NG, TV, None
Obasi 2001[882]	1998	Tanzania	Youth	M, F	15 to 19	Non-stratified	**VD:** CT; **UD:** CT
Chalamilla 2006[883]	1999	Tanzania	GYN/FP attendees	M, F	15 to 24	Non-stratified	**VD:** CS, CT, NG, TV **UD:** CT, NG, TV; **GU:** TP
Msuya 2006[884]	1999	Tanzania	ANC/FP/PED attendees	F	16 to 46	NR	**VD:** TV; **GU:** TP
Pickering 2005[885]	2000	Uganda	Symptomatic clinic attendees	M, F	15+	Non-stratified	**VD:** BV, CS, CT, NG, TV, None **UD:** CT, NG, TV **GU:** HD, HSV-2, TP
Wolday 2004[886]	2001	Ethiopia	Symptomatic clinic attendees	F	17 to 51	Non-stratified	**VD:** BV, CT, TV; **GU:** TP

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Reference	Year	Country	Population	Sex	Age	HIV status	Infections extracted
Ly 2006[887]	2001	Senegal	Symptomatic clinic attendees	M	NR	NR	**UD:** CT, NG, **GU:** TP
Rassjo 2006[888]	2002	Uganda	Sexually experienced youth; Symptomatic clinic attendees	M, F	14 to 19	Non-stratified	**VD:** TV; **UD:** TV; **GU:** TP
Bochner 2017[889]	2007	Botswana, South Africa, Zambia, Kenya, Rwanda, Tanzania, Uganda	HIV-1 serodiscordant cohort study participants	M, F	18+	Non-stratified	**VD:** TV; **UD:** TV
Djomand 2016[890]	2010	Kenya	HIV clinic attendees	F	18 to 70	HIV-positive	**VD:** BV, CS, NG, TV; **GU:** HSV
Dela 2019[891]	2014	Ghana	Symptomatic clinic attendees	M, F	18+	NR	**VD:** CT, NG, **UD:** CT, NG
NICD 2023[892]	2014	South Africa	Symptomatic clinic attendees	M, F	15+	Non-stratified	**VD:** BV, CS, CT, MG, NG, TV, None **UD:** CT, MG, NG, TV, None **GU:** HD, HSV, TP, None
Verwijs 2019[893]	2017	Rwanda	Women at risk of STIs	F	18+	Non-stratified	**VD:** CA, TV, **GU:** TP
Wall 2021[894]	2018	Rwanda	Symptomatic clinic attendees	M, F	NR	Non-stratified	**VD:** CT, NG; **UD:** CT, NG
Sarenje 2022[895]	2021	Zambia	Symptomatic clinic attendees	M, F	15 to 54	Non-stratified	**VD:** NG; **UD:** NG

Year is midpoint between start and end of data collection. *For studies without data collection dates reported, study year was assumed equivalent to the year of publication.
ANC: antenatal care, FP: family planning clinic, GYN: gynaecology clinic, PED: paediatric clinic, FSW: Female sex worker.
F: Female, M: Male.
VD: Vaginal discharge, UD: Urethral discharge, GU: genital ulcer.
BV: Bacterial vaginosis, CA: *Candida albicans*, CS: *Candida* species (any), CT: *Chlamydia trachomatis*, HD: *Haemophilus ducreyi*, HSV: Herpes simplex virus (unspecified), HSV-1: Herpes simplex virus type 1, HSV-2: Herpes simplex virus type 2, LGV: lymphogranuloma venereum, MG: *Mycoplasma genitalium*, NG: *Neisseria gonorrhoeae*, TP: *Treponema pallidum*, TV: *Trichomonas vaginalis*, None: unknown aetiology.
NR: Not reported.

Table B11: Summary of characteristics for studies included in the analysis of regional trends in aetiology, among adults of non-stratified or unmeasured HIV status

	Vaginal discharge	Urethral discharge	Genital ulcer	All symptoms
	$N_{VD} = 78$	$N_{UD} = 50$	$N_{GU} = 74$	N = 176
Population group[1]				
Symptomatic clinic attendee	54 (69.2%)	45 (90.0%)	59 (79.7%)	136 (77.3%)
General	24 (30.8%)	2 (4.0%)	5 (6.8%)	30 (17.0%)
Higher-risk general	2 (2.6%)	3 (6.0%)	7 (9.5%)	10 (5.7%)
Key populations	6 (7.7%)	1 (2.0%)	7 (9.5%)	13 (7.4%)
Sex				
Male	-	50 (100.0%)	19 (25.7%)	53 (30.1%)
Female	78 (100.0%)	-	19 (25.7%)	79 (44.9%)
Non-stratified	-	-	36 (48.6%)	44 (25.0%)
Mean or median age				
$<$ 25 years	7 (9.0%)	2 (4.0%)	1 (1.4%)	7 (4.0%)
$\geq$ 25 years	17 (21.8%)	12 (24.0%)	21 (28.4%)	45 (25.6%)
NR	54 (69.2%)	36 (72.0%)	52 (70.3%)	124 (70.5%)
HIV prevalence				
$<$ 25%	6 (7.7%)	1 (2.0%)	4 (5.4%)	11 (6.3%)
$\geq$ 25%	8 (10.3%)	6 (12.0%)	34 (45.9%)	40 (22.7%)
NR	64 (82.1%)	43 (86.0%)	36 (48.6%)	125 (71.0%)
Region[1,2]				
Central Africa	4 (5.1%)	2 (4.0%)	1 (1.4%)	6 (3.4%)
Eastern Africa	30 (38.5%)	27 (54.0%)	44 (59.5%)	82 (46.6%)
Southern Africa	10 (12.8%)	11 (22.0%)	22 (29.7%)	41 (23.3%)
Western Africa	33 (42.3%)	9 (18.0%)	6 (8.1%)	45 (25.6%)
Multiple (not stratified)	1 (1.3%)	1 (2.0%)	1 (1.4%)	2 (1.1%)
Study midpoint year[1,3]				
<1990	8 (10.3%)	9 (18.0%)	18 (24.3%)	32 (18.2%)
1990 - 1999	19 (24.4%)	16 (32.0%)	34 (45.9%)	60 (34.1%)
2000 - 2009	27 (34.6%)	14 (28.0%)	17 (23.0%)	50 (28.4%)
2010 - 2023	27 (34.6%)	12 (24.0%)	9 (12.2%)	40 (22.7%)
Study design				
Cross sectional	67 (85.9%)	42 (84.0%)	58 (78.4%)	148 (84.1%)
Cohort baseline	9 (11.5%)	7 (14.0%)	12 (16.2%)	22 (12.5%)
RCT baseline	2 (2.6%)	1 (2.0%)	4 (5.4%)	6 (3.4%)
Sampling method				
Non-probability based	67 (85.9%)	48 (96.0%)	70 (94.6%)	160 (90.9%)
Probability based	5 (6.4%)	1 (2.0%)	3 (4.1%)	8 (4.5%)
NR	6 (7.7%)	1 (2.0%)	1 (1.4%)	8 (4.5%)
Sample size[4]				
$<$ 100	22 (28.2%)	16 (32.0%)	43 (58.1%)	68 (38.6%)
$\geq$ 100	56 (71.8%)	34 (68.0%)	31 (41.9%)	108 (61.4%)

[1]The same study is included in more than one subcategory when it reports across different variable levels or multiple variable levels are relevant. [2]Regions classified according to UN M49 Standard. [3]Mid-point year between start and end dates of data collection. If dates were not reported, year of publication was used as proxy. [4]Sample size defined as the number of symptomatic individuals tested in the study. If a study used different samples across tests, the largest sample size was reported. N: number of studies.

Table B12: Summary of characteristics for studies included in the analysis to assess the effect of HIV status, among adults of known HIV status

	Vaginal discharge	Urethral discharge	Genital ulcer	All symptoms
	$N_{VD} = 12$	$N_{UD} = 6$	$N_{GU} = 28$	N = 42
Population group[1]				
Symptomatic clinic attendee	10 (83.3%)	6 (100.0%)	24 (85.7%)	36 (85.7%)
General	1 (8.3%)	-	1 (3.6%)	2 (4.8%)
Higher-risk general	-	-	2 (7.1%)	2 (4.8%)
Key populations	1 (8.3%)	-	3 (10.7%)	4 (9.5%)
Youth	-	-	-	-
Sex[1]				
Male	-	6 (100.0%)	11 (39.3%)	18 (42.9%)
Female	12 (100.0%)	-	9 (32.1%)	16 (38.1%)
Non-stratified	-	-	12 (42.9%)	12 (28.6%)
HIV status[1]				
Positive	8 (66.7%)	5 (83.3%)	27 (96.4%)	38 (90.5%)
Negative	11 (91.7%)	5 (83.3%)	25 (89.3%)	36 (85.7%)
Region[1,2]				
Central Africa	-	-	-	-
Eastern Africa	5 (41.7%)	4 (66.7%)	16 (57.1%)	22 (52.4%)
Southern Africa	4 (33.3%)	2 (33.3%)	11 (39.3%)	16 (38.1%)
Western Africa	3 (25.0%)	-	2 (7.1%)	5 (11.9%)
Study midpoint year[1,3]				
<1990	-	1 (16.7%)	2 (7.1%)	3 (7.1%)
1990 - 1999	-	1 (16.7%)	13 (46.4%)	14 (33.3%)
2000 - 2009	5 (41.7%)	2 (33.3%)	8 (28.6%)	13 (31.0%)
2010 - 2023	8 (66.7%)	3 (50.0%)	7 (25.0%)	14 (33.3%)
Study design				
Cross sectional	8 (66.7%)	4 (66.7%)	20 (71.4%)	28 (66.7%)
Cohort baseline	2 (16.7%)	2 (33.3%)	5 (17.9%)	9 (21.4%)
RCT baseline	2 (16.7%)	-	3 (10.7%)	5 (11.9%)
Sampling method				
Non-probability based	9 (75.0%)	6 (100.0%)	25 (89.3%)	37 (88.1%)
Probability based	3 (25.0%)	-	3 (10.7%)	5 (11.9%)
Sample size[4]				
< 100	3 (25.0%)	2 (33.3%)	15 (53.6%)	19 (45.2%)
≥ 100	9 (75.0%)	4 (66.7%)	13 (46.4%)	23 (54.8%)

[1]The same study is included in more than one subcategory when it reports across different variable levels or multiple variable levels are relevant. [2]Regions classified according to UN M49 Standard. [3]Mid-point year between start and end dates of data collection. If dates were not reported, year of publication was used as proxy. [4]Sample size defined as the number of symptomatic individuals tested in the study. If a study used different samples across tests, the largest sample size was reported. N: number of studies.

Table B13: Summary of characteristics for studies included in the analysis to assess the effect of age, among youth < 25 years and adults ≥ 25 years

	Vaginal discharge	Urethral discharge	Genital ulcer	All symptoms
	$N_{VD} = 17$	$N_{UD} = 7$	$N_{GU} = 7$	N = 24
Population group[1]				
Symptomatic clinic attendee	14 (82.4%)	6 (85.7%)	6 (85.7%)	20 (83.3%)
General	1 (5.9%)	-	1 (14.3%)	2 (8.3%)
Higher-risk general	-	-	-	-
Key populations	1 (5.9%)	-	-	1 (4.2%)
Youth	15 (88.2%)	7 (100.0%)	6 (85.7%)	20 (83.3%)
Sex				
Male	-	7 (100.0%)	1 (14.3%)	4 (16.7%)
Female	17 (100.0%)	-	1 (14.3%)	14 (58.3%)
Non-stratified	-	-	5 (71.4%)	6 (25.0%)
Age group[1]				
< 25 years	15 (88.2%)	7 (100.0%)	6 (85.7%)	21 (87.5%)
≥ 25 years	13 (76.5%)	3 (42.9%)	4 (57.1%)	18 (75.0%)
Region[1,2]				
Central Africa	-	-	-	-
Eastern Africa	6 (35.3%)	5 (71.4%)	5 (71.4%)	11 (45.8%)
Southern Africa	1 (5.9%)	1 (14.3%)	1 (14.3%)	1 (4.2%)
Western Africa	10 (58.8%)	1 (14.3%)	-	11 (45.8%)
Multiple (not stratified)	-	-	1 (14.3%)	1 (4.2%)
Study midpoint year[1,3]				
<1990	1 (5.9%)	1 (14.3%)	-	2 (8.3%)
1990 - 1999	2 (11.8%)	3 (42.9%)	1 (14.3%)	3 (12.5%)
2000 - 2009	10 (58.8%)	3 (42.9%)	5 (71.4%)	14 (58.3%)
2010 - 2023	4 (23.5%)	1 (14.3%)	2 (28.6%)	6 (25.0%)
Study design				
Cross sectional	14 (82.4%)	7 (100.0%)	4 (57.1%)	18 (75.0%)
Cohort baseline	2 (11.8%)	-	1 (14.3%)	3 (12.5%)
RCT baseline	1 (5.9%)	-	2 (28.6%)	3 (12.5%)
Sampling method				
Non-probability based	13 (76.5%)	5 (71.4%)	6 (85.7%)	20 (83.3%)
Probability based	2 (11.8%)	2 (28.6%)	1 (14.3%)	2 (8.3%)
NR	2 (11.8%)	-	-	2 (8.3%)
Sample size[4]				
< 100	4 (23.5%)	5 (71.4%)	3 (42.9%)	7 (29.2%)
≥ 100	13 (76.5%)	2 (28.6%)	4 (57.1%)	17 (70.8%)

[1]The same study is included in more than one subcategory when it reports across different variable levels or multiple variable levels are relevant. [2]Regions classified according to UN M49 Standard. [3]Mid-point year between start and end dates of data collection. If dates were not reported, year of publication was used as proxy. [4]Sample size defined as the number of symptomatic individuals tested in the study. If a study used different samples across tests, the largest sample size was reported. N: number of studies.

Table B14: Adjusted odds of vaginal discharge, urethral discharge, and genital ulcer among adults of non-stratified or unmeasured HIV status, estimated via generalised linear mixed-effects models

Vaginal discharge		**Urethral discharge**		**Genital ulcer**	
Variable	**aOR (95% CI)**	**Variable**	**aOR (95% CI)**	**Variable**	**aOR (95% CI)**
BV	1.08 (0.51-2.29)	CT	0.28 (0.19-0.42)	HD	0.01 (0.01-0.02)
CA	0.45 (0.13-1.54)	MG	0.06 (0.04-0.09)	HSV	1.73 (1.04-2.90)
CS	1.57 (0.66-3.74)	NG	3.48 (2.46-4.93)	HSV-1	0.01 (0.01-0.03)
CT	0.20 (0.10-0.41)	TV	0.03 (0.02-0.04)	HSV-2	1.26 (0.83-1.91)
MG	0.06 (0.02-0.15)	None	0.11 (0.07-0.17)	LGV	0.02 (0.01-0.04)
NG	0.09 (0.04-0.18)	CT:year	1.02 (0.99-1.06)	TP	0.10 (0.07-0.14)
TV	0.17 (0.08-0.32)	MG:year	0.95 (0.91-1.00)	None	0.60 (0.42-0.85)
None	0.34 (0.14-0.81)	NG:year	1.01 (0.99-1.03)	HD:year	0.85 (0.83-0.86)
BV:year	1.01 (0.96-1.06)	TV:year	0.93 (0.90-0.96)	HSV:year	1.08 (1.06-1.11)
CA:year	1.01 (0.95-1.06)	None:year	0.95 (0.91-0.99)	HSV-1:year	1.00 (0.95-1.06)
CS:year	1.10 (1.05-1.15)			HSV-2:year	1.01 (0.98-1.04)
CT:year	1.03 (0.99-1.08)			LGV:year	0.93 (0.89-0.96)
MG:year	0.99 (0.90-1.10)			TP:year	0.98 (0.96-0.99)
NG:year	0.99 (0.96-1.03)			None:year	1.02 (1.00-1.04)
TV:year	0.94 (0.91-0.97)			HD:male	1.98 (1.27-3.08)
None:year	1.00 (0.94-1.05)			HSV:male	0.68 (0.39-1.17)
				HSV-1:male	1.37 (0.51-3.73)
				HSV-2:male	0.65 (0.38-1.13)
				LGV:male	0.92 (0.44-1.91)
				TP:male	0.79 (0.51-1.24)
				None:male	1.21 (0.82-1.79)
				HD:male and female	2.49 (1.39-4.45)
				HSV:male and female	0.72 (0.34-1.52)
				HSV-1:male and female	1.92 (0.59-6.30)
				HSV-2:male and female	0.68 (0.28-1.65)
				LGV:male and female	1.45 (0.42-5.06)
				TP:male and female	0.92 (0.56-1.53)
				None:male and female	0.88 (0.50-1.53)
Random effects		**Random effects**		**Random effects**	
Standard deviation		*Standard deviation*		*Standard deviation*	
σ_{00} region:rti	0.51	σ_{00} region:rti	0.06	σ_{00} region:rti	<0.001
σ_{00} observation	0.96	σ_{00} observation	0.88	σ_{00} observation	0.86
σ_{11} region:rti:year	0.02	σ_{11} region:rti:year	0.01	σ_{11} region:rti:year	<0.001
ρ_{01} region:rti	0.53	ρ_{01} region:rti	-0.94	ρ_{01} region:rti	0.98
Variance		*Variance*		*Variance*	
τ^2 fixed&random	1.20 (56.4%)	τ^2 fixed&random	2.70 (77.2%)	τ^2 fixed&random	2.17 (74.0%)
τ^2 dispersion	0.91 (43.0%)	τ^2 dispersion	0.78 (22.3%)	τ^2 dispersion	0.73 (25.0%)
τ^2 distribution	0.01 (0.6%)	τ^2 distribution	0.02 (0.5%)	τ^2 distribution	0.03 (1.0%)
τ^2 total	2.12 (100.0%)	τ^2 total	3.49 (100.0%)	τ^2 total	2.93 (100.0%)
Number groups		*Number groups*		*Number groups*	
N region	3	N region	3	N region	3
N observation	339	N observation	217	N observation	483

Midpoint year of study is centred at 2015. aOR: adjusted odds ratio; CI: confidence interval; σ_{00}: random intercept standard deviation; σ_{11}: random slope standard deviation; ρ_{01}: correlation between intercept and slope; τ^2: variance attributed to fixed and random effects; dispersion (observation-level) effects; and the binomial distribution; N: number.

Table B15: Adjusted odds of vaginal discharge, urethral discharge, and genital ulcer among adults of known HIV status, estimated via generalised linear mixed-effects models

Vaginal discharge		**Urethral discharge**		**Genital ulcer**	
Variable	**aOR (95% CI)**	**Variable**	**aOR (95% CI)**	**Variable**	**aOR (95% CI)**
BV	0.55 (0.33-0.92)	CT	0.40 (0.30-0.53)	HD	0.03 (0.02-0.05)
CS	0.81 (0.46-1.42)	MG	0.06 (0.05-0.08)	HSV	0.94 (0.57-1.56)
CT	0.14 (0.09-0.25)	NG	4.85 (3.66-6.43)	HSV-1	0.03 (0.01-0.06)
MG	0.04 (0.02-0.07)	TV	0.02 (0.01-0.03)	HSV-2	1.20 (0.79-1.83)
NG	0.11 (0.06-0.18)	None	0.09 (0.07-0.12)	LGV	0.04 (0.02-0.07)
TV	0.11 (0.07-0.18)	CT:year	1.03 (1.00-1.06)	TP	0.12 (0.08-0.17)
None	0.25 (0.14-0.45)	MG:year	0.95 (0.91-1.00)	None	0.72 (0.52-1.01)
BV:year	1.01 (0.99-1.04)	NG:year	1.05 (1.02-1.08)	HD:year	0.88 (0.86-0.89)
CS:year	0.99 (0.95-1.03)	TV:year	0.89 (0.86-0.93)	HSV:year	1.05 (1.03-1.07)
CT:year	1.04 (1.01-1.07)	None:year	0.92 (0.88-0.96)	HSV-1:year	1.04 (0.96-1.13)
MG:year	1.00 (0.96-1.04)	CT:HIV-positive	0.54 (0.37-0.81)	HSV-2:year	0.99 (0.96-1.02)
NG:year	1.02 (0.99-1.05)	MG:HIV-positive	1.04 (0.66-1.64)	LGV:year	0.99 (0.94-1.05)
TV:year	0.94 (0.92-0.97)	NG:HIV-positive	1.06 (0.71-1.56)	TP:year	1.00 (0.99-1.02)
None:year	1.01 (0.97-1.04)	TV:HIV-positive	1.87 (1.07-3.25)	None:year	1.01 (0.99-1.03)
BV:HIV-positive	1.85 (1.38-2.46)	None:HIV-positive	1.40 (0.92-2.14)	HD:male	1.71 (1.11-2.65)
CS:HIV-positive	0.50 (0.34-0.73)			HSV:male	1.01 (0.62-1.63)
CT:HIV-positive	0.95 (0.70-1.29)			HSV-1:male	0.74 (0.28-1.96)
MG:HIV-positive	2.32 (1.59-3.39)			HSV-2:male	0.44 (0.27-0.73)
NG:HIV-positive	1.61 (1.18-2.18)			LGV:male	0.71 (0.37-1.34)
TV:HIV-positive	2.15 (1.61-2.87)			TP:male	1.42 (0.96-2.09)
None:HIV-positive	0.72 (0.53-0.99)			None:male	0.86 (0.59-1.24)
				HD:male and female	1.61 (0.97-2.68)
				HSV:male and female	0.61 (0.31-1.18)
				HSV-1:male and female	2.04 (0.63-6.63)
				HSV-2:male and female	0.47 (0.26-0.86)
				LGV:male and female	2.50 (0.95-6.59)
				TP:male and female	1.55 (0.96-2.49)
				None:male and female	0.79 (0.50-1.26)
				HD:HIV-positive	0.83 (0.58-1.19)
				HSV:HIV-positive	1.78 (1.15-2.75)
				HSV-1:HIV-positive	1.17 (0.52-2.63)
				HSV-2:HIV-positive	1.63 (1.07-2.46)
				LGV:HIV-positive	0.95 (0.53-1.72)
				TP:HIV-positive	0.89 (0.64-1.24)
				None:HIV-positive	0.66 (0.48-0.92)
Random effects		**Random effects**		**Random effects**	
Standard deviation		*Standard deviation*		*Standard deviation*	
σ_{00} region:rti	0.39	σ_{00} region:rti	<0.001	σ_{00} region:rti	<0.001
σ_{00} observation	0.42	σ_{00} observation	0.54	σ_{00} observation	0.72
Variance		*Variance*		*Variance*	
τ^2 fixed&random	0.90 (81.9%)	τ^2 fixed&random	3.24 (91.1%)	τ^2 fixed&random	1.78 (75.6%)
τ^2 dispersion	0.18 (16.0%)	τ^2 dispersion	0.29 (8.2%)	τ^2 dispersion	0.52 (22.1%)
τ^2 distribution	0.02 (2.1%)	τ^2 distribution	0.02 (0.7%)	τ^2 distribution	0.05 (2.2%)
τ^2 total	1.10 (100.0%)	τ^2 total	3.56 (100.0%)	τ^2 total	2.36 (100.0%)
Number groups		*Number groups*		*Number groups*	
N region	3	N region	3	N region	3
N observation	275	N observation	177	N observation	512

Midpoint year of study is centred at 2015. aOR: adjusted odds ratio; CI: confidence interval; σ_{00}: random intercept standard deviation; τ^2: variance attributed to fixed and random effects, dispersion (observation-level) effects, and the binomial distribution; N: number.

Table B16: Adjusted odds of vaginal discharge, urethral discharge, and genital ulcer among youth (<25 years) and adults (≥ 25 years), estimated via generalised linear mixed-effects models

Vaginal discharge		Urethral discharge		Genital ulcer	
Variable	**aOR (95% CI)**	**Variable**	**aOR (95% CI)**	**Variable**	**aOR (95% CI)**
BV	0.69 (0.29-1.60)	CT	0.28 (0.22-0.37)	HD	0.02 (0.02-0.04)
CA	0.49 (0.07-3.53)	MG	0.05 (0.04-0.07)	HSV	1.25 (0.78-1.99)
CS	0.49 (0.19-1.25)	NG	3.75 (2.92-4.82)	HSV-1	1.05 (0.00-0.05)
CT	0.08 (0.03-0.19)	TV	0.03 (0.02-0.04)	HSV-2	1.50 (1.11-2.02)
MG	0.04 (0.01-0.13)	None	0.10 (0.08-0.13)	LGV	0.02 (0.01-0.04)
NG	0.05 (0.02-0.13)	CT:year	1.04 (1.01-1.08)	TP	0.08 (0.06-0.11)
TV	0.11 (0.04-0.25)	MG:year	0.92 (0.88-0.96)	None	0.57 (0.46-0.71)
None	0.36 (0.13-0.98)	NG:year	1.05 (1.03-1.07)	HD:year	1.01 (0.95-1.08)
BV:year	1.05 (1.02-1.09)	TV:year	0.94 (0.92-0.96)	HSV:year	0.99 (0.91-1.07)
CA:year	1.05 (0.96-1.14)	None:year	0.90 (0.86-0.94)	HSV-1:year	1.09 (0.88-1.36)
CS:year	1.01 (0.96-1.05)	CT:<25 years	1.14 (0.92-2.30)	HSV-2:year	0.97 (0.93-1.02)
CT:year	1.04 (1.01-1.08)	MG:<25 years	1.01 (0.65-1.56)	LGV:year	1.02 (0.95-1.10)
MG:year	0.99 (0.95-1.03)	NG:<25 years	1.39 (0.92-2.01)	TP:year	1.10 (1.07-1.14)
NG:year	1.00 (0.96-1.03)	TV:<25 years	1.35 (0.79-2.31)	None:year	0.97 (0.95-1.00)
TV:year	0.92 (0.89-0.95)	None:<25 years	0.73 (0.48-1.11)	HD:Male	0.65 (0.36-1.16)
None:year	0.98 (0.95-1.02)			HSV:Male	0.89 (0.61-1.30)
BV:<25 years	1.12 (0.84-1.51)			HSV-1:Male	0.81 (0.30-2.18)
CA:<25 years	2.15 (0.74-6.26)			HSV-2:Male	0.64 (0.43-0.96)
CS:<25 years	1.82 (1.25-2.66)			LGV:Male	0.76 (0.41-1.41)
CT:<25 years	2.39 (1.72-3.32)			TP:Male	2.01 (1.37-2.94)
MG:<25 years	1.99 (1.33-2.98)			None:Male	0.93 (0.71-1.22)
NG:<25 years	1.65 (1.20-2.27)			HD:Male and female	0.40 (0.11-1.48)
TV:<25 years	0.85 (0.63-1.13)			HSV-1:Male and female	13.76 (0.35-540)
None:<25 years	0.67 (0.49-0.92)			HSV-2:Male and female	0.31 (0.16-0.59)
				TP:Male and female	2.30 (1.12-4.72)
				None:Male and female	1.46 (0.84-2.53)
				HD:<25 years	2.30 (1.29-4.07)
				HSV:<25 years	1.01 (0.68-1.51)
				HSV-1:<25 years	2.65 (0.92-7.63)
				HSV-2:<25 years	1.02 (0.69-1.51)
				LGV:<25 years	2.40 (1.27-4.54)
				TP:<25 years	1.39 (0.96-2.04)
				None:<25 years	1.12 (0.85-1.48)
Random effects		**Random effects**		**Random effects**	
Standard deviation		*Standard deviation*		*Standard deviation*	
σ_{00} region:rfl	0.68	σ_{00} region:rfl	<0.001	σ_{00} region:rfl	<0.001
σ_{00} observation	0.42	σ_{00} observation	0.47	σ_{00} observation	0.36
Variance		*Variance*		*Variance*	
τ^2 fixed&random	1.47 (87.9%)	τ^2 fixed&random	3.08 (92.6%)	τ^2 fixed&random	2.58 (92.7%)
τ^2 dispersion	0.18 (10.7%)	τ^2 dispersion	0.23 (6.8%)	τ^2 dispersion	0.13 (4.6%)
τ^2 distribution	0.02 (1.4%)	τ^2 distribution	0.02 (0.7%)	τ^2 distribution	0.07 (2.6%)
τ^2 total	1.67 (100.0%)	τ^2 total	3.33 (100.0%)	τ^2 total	2.78 (100.0%)
Number groups		*Number groups*		*Number groups*	
N region	3	N region	3	N region	3
N observation	281	N observation	163	N observation	287

Midpoint year of study is centred at 2015. aOR: adjusted odds ratio; CI: confidence interval; σ_{00}: random intercept standard deviation; τ^2: variance attributed to fixed and random effects, dispersion (observation-level) effects, and the binomial distribution; N: number.

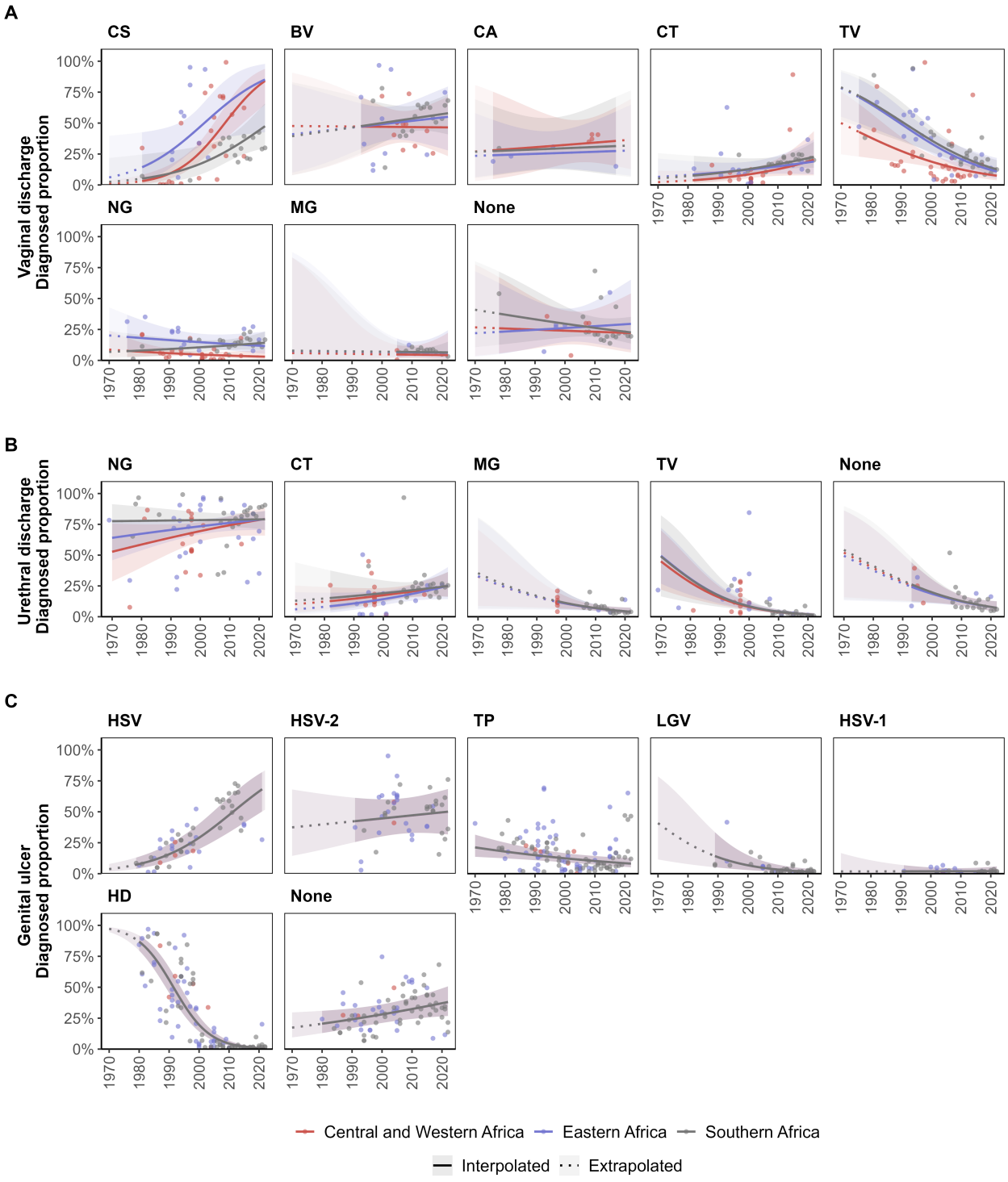


Figure B2: Estimated diagnosed proportion per pathogen among symptomatic adults by sub-region in SSA, 1970-2023

Diagnosed proportion per pathogen among adults of non-stratified or unmeasured HIV status symptomatic with (A) vaginal discharge, (B) urethral discharge, and (C) genital ulcer. Lines and shaded areas represent sex-matched population-weighted mean proportions and 95% CIs. Solid lines and darker shading denote estimates and CIs within the observed data range (interpolated), while dotted lines and lighter shading indicate estimates and 95% CIs beyond that time frame (extrapolated). Points represent study observations adjusted for diagnostic test performance.

Table B17: Estimated diagnosed proportion per pathogen in SSA, with and without diagnostic test performance adjustment

RTI	Adjusted proportion		Unadjusted proportion	
	1980	2015	1980	2015
Vaginal discharge				
CS	5.6 (1.5-18.2)	69.4 (44.3-86.6)	24.7 (14.5-38.9)	36.2 (23.9-50.6)
BV	45.5 (14.7-80.1)	50.0 (32.3-67.8)	24.7 (10.4-48.1)	37.6 (25.6-51.3)
CA	26.8 (7.6-62.1)	31.5 (12.4-59.9)	27.1 (12.8-48.6)	33.0 (16.4-55.3)
CT	5.3 (1.7-15.0)	16.2 (8.6-28.5)	4.0 (1.9-8.6)	13.1 (8.0-20.7)
TV	51.6 (32.3-70.5)	12.9 (7.8-20.7)	24.1 (15.2-36.1)	9.7 (6.5-14.2)
NG	10.6 (4.9-21.7)	6.6 (3.3-12.7)	7.0 (3.6-13.2)	5.9 (3.3-10.3)
MG	6.4 (0.2-65.2)	5.4 (1.8-14.8)	9.8 (1.0-53.4)	7.0 (3.1-15.4)
None	25.6 (6.5-63.1)	25.1 (10.7-48.4)	29.8 (12.9-54.8)	22.9 (11.9-39.5)
Urethral discharge				
NG	63.9 (47.1-77.8)	77.1 (68.1-84.1)	54.5 (40.9-67.5)	70.3 (61.6-77.7)
CT	10.6 (4.4-23.2)	21.9 (15.4-30.3)	8.3 (4.0-16.3)	19.1 (13.9-25.7)
MG	23.5 (7.0-55.4)	5.5 (3.7-8.3)	22.7 (8.3-49.1)	5.0 (3.4-7.4)
TV	29.3 (15.6-48.0)	2.8 (1.8-4.3)	12.5 (6.9-21.5)	3.9 (2.6-5.6)
None	38.9 (13.6-72.0)	10.0 (6.8-14.5)	39.0 (16.3-67.7)	9.5 (6.6-13.5)
Genital ulcer				
HSV	7.8 (4.3-13.7)	57.1 (40.9-71.9)	5.0 (2.6-9.5)	58.7 (41.1-74.4)
HSV-2	39.8 (19.4-64.4)	48.3 (32.9-64.1)	36.6 (16.1-63.4)	49.5 (32.6-66.5)
TP	17.9 (12.4-25.1)	9.3 (6.4-13.4)	16.2 (10.8-23.6)	7.8 (5.1-11.7)
LGV	24.6 (7.7-55.9)	2.3 (1.0-5.3)	47.4 (16.2-80.7)	1.2 (0.5-3.2)
HSV-1	1.8 (0.3-10.8)	2.0 (0.8-4.7)	2.6 (0.3-21.5)	0.6 (0.2-2.2)
HD	86.9 (79.7-91.8)	2.0 (1.2-3.3)	81.8 (71.8-88.9)	0.9 (0.5-1.7)
None	20.5 (12.9-30.9)	34.7 (25.5-45.1)	19.9 (12.0-31.2)	33.9 (24.2-45.3)

Estimated proportions are with or without diagnostic test performance adjustment and presented as percentages with 95% confidence intervals.

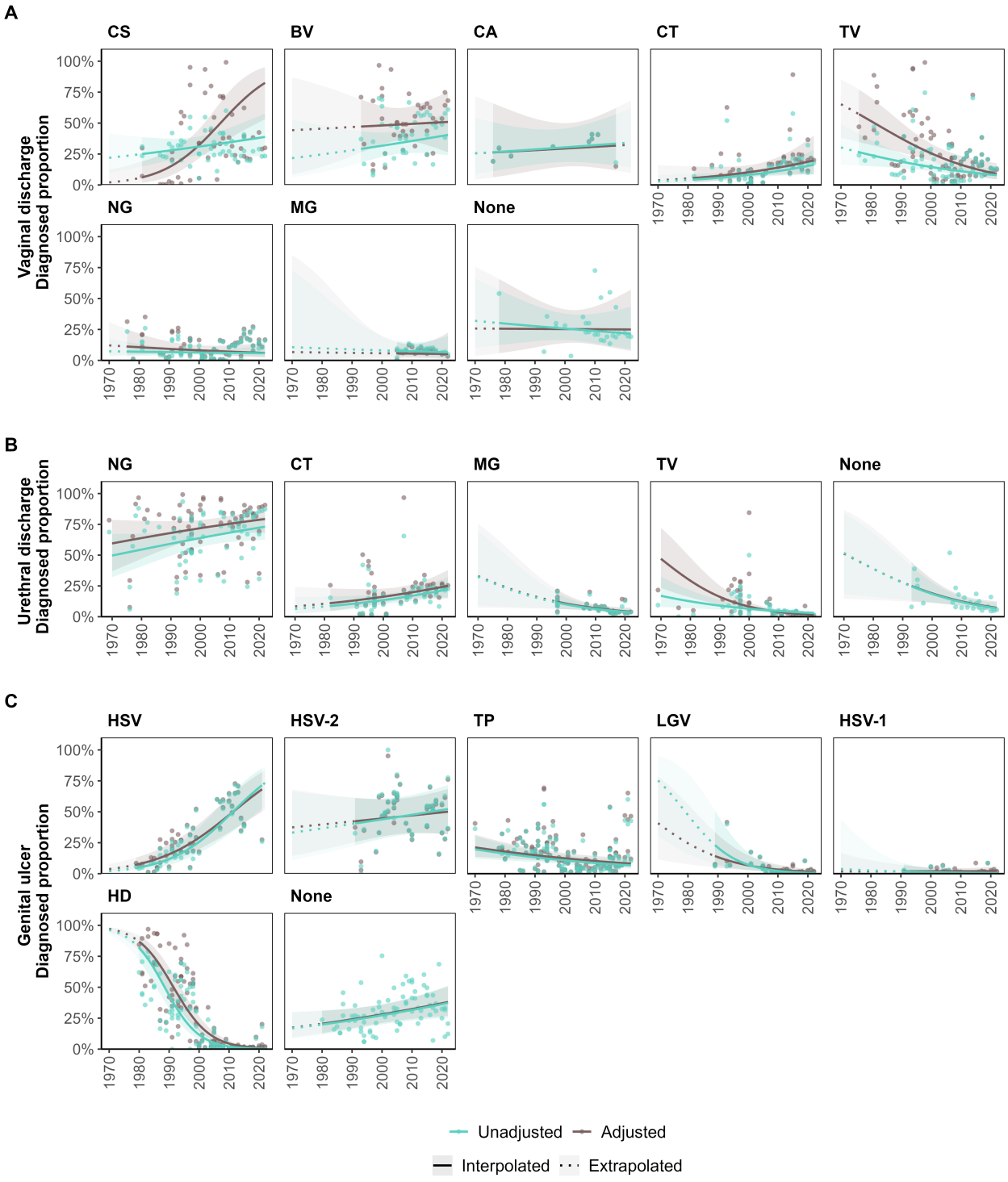


Figure B3: Estimated diagnosed proportion per pathogen among symptomatic adults in SSA, with and without diagnostic test performance adjustment, 1970-2023

Diagnosed proportion per pathogen among symptomatic adults of non-stratified or unmeasured HIV status with (A) vaginal discharge, (B) urethral discharge, and (C) genital ulcer. Proportions estimated using either observations as reported or adjusted for diagnostic test performance. Lines and shaded areas represent sex-matched population-weighted mean proportions and 95% CIs. Solid lines and darker shading denote estimates and 95% CIs within the observed data range (interpolated), while dotted lines and lighter shading indicate estimates and 95% CIs beyond that time frame (extrapolated). Points represent study observations that are unadjusted or adjusted for diagnostic test performance.

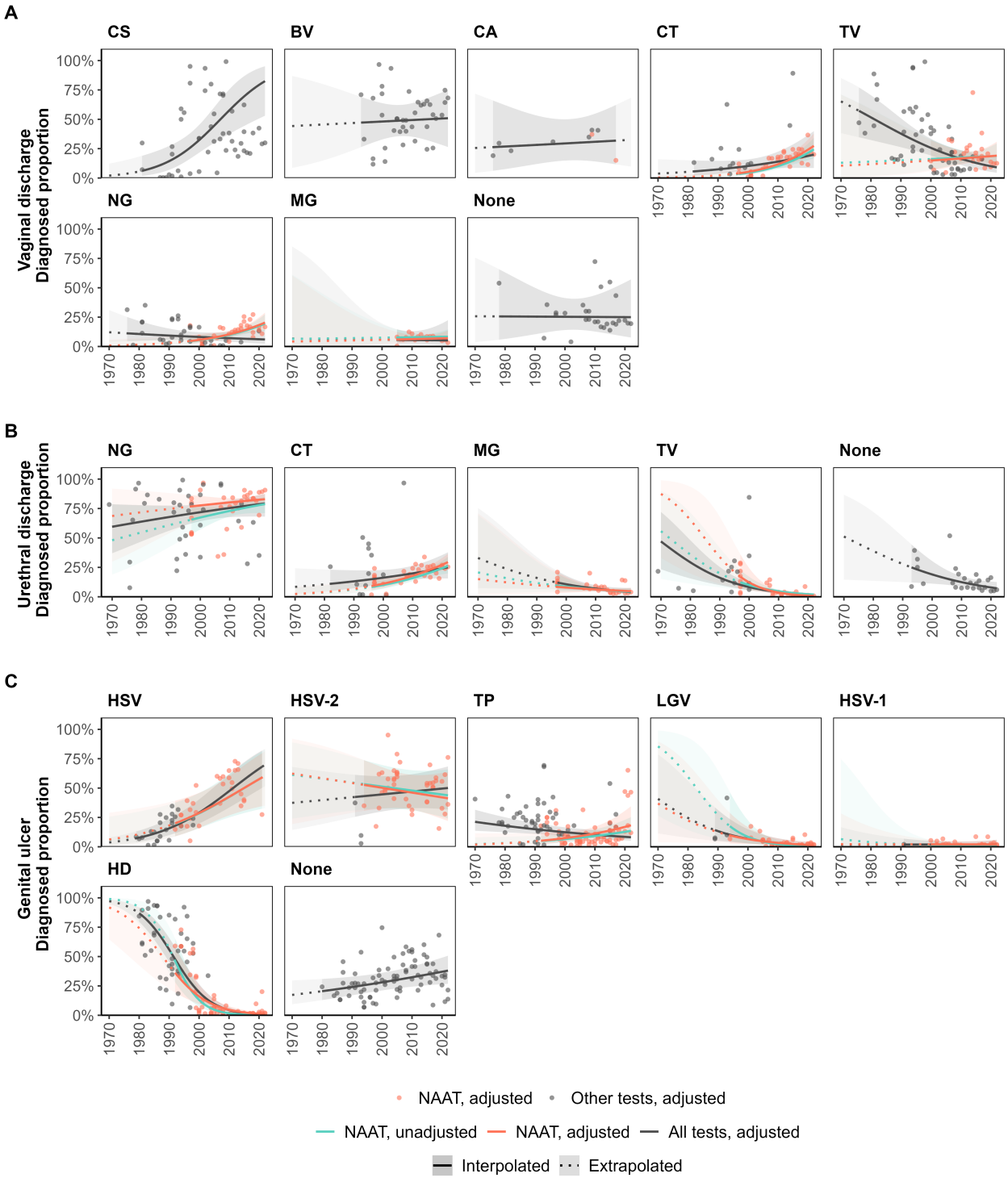


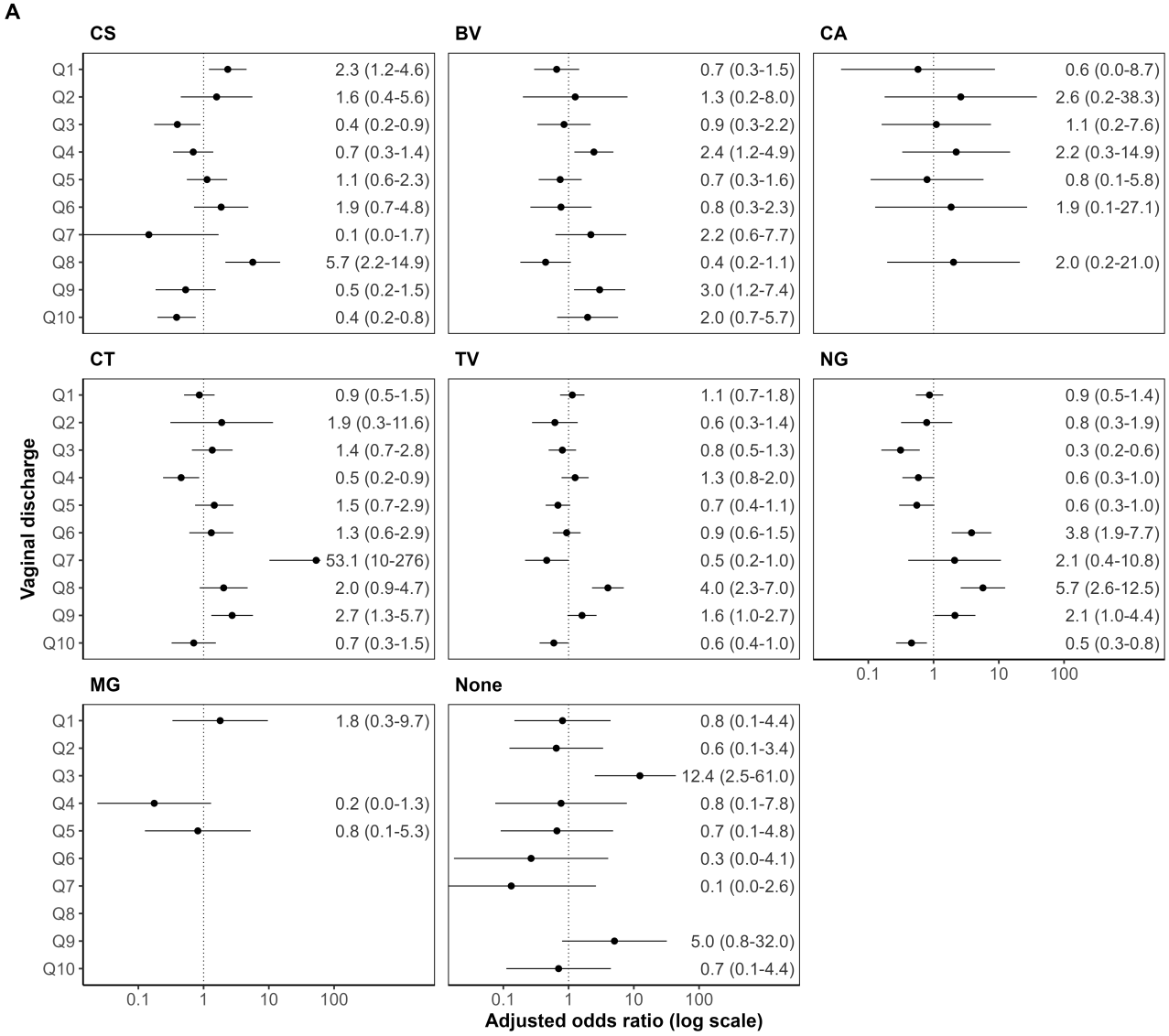
Figure B4: Estimated diagnosed proportion per pathogen among symptomatic adults in SSA, using NAAT with and without diagnostic test performance adjustment, 1970-2023

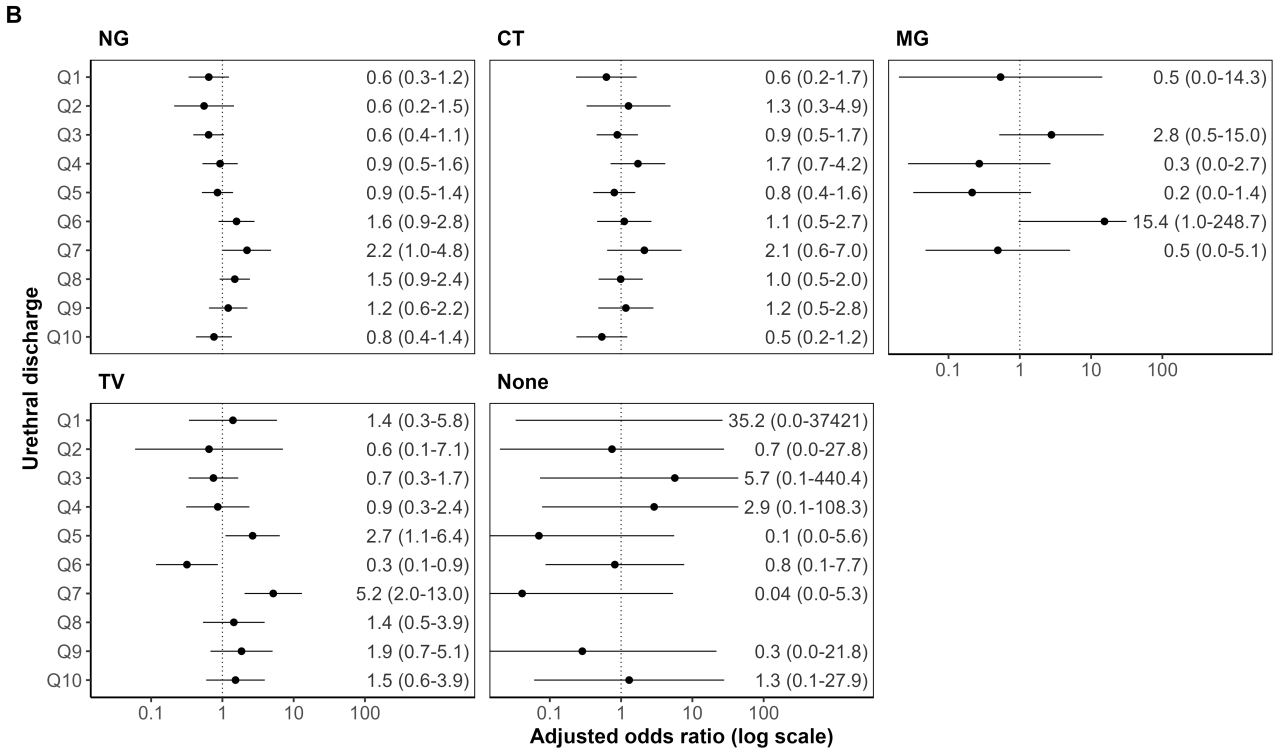
Diagnosed proportion per pathogen among adults of non-stratified or unmeasured HIV status symptomatic with (A) vaginal discharge, (B) urethral discharge, and (C) genital ulcer. Proportions estimated using NAAT-based observations as reported or adjusted for diagnostic performance, compared to all observations adjusted for diagnostic performance. Lines and shaded areas represent sex-matched population-weighted mean proportions and 95% CIs. Solid lines and darker shading denote estimates and 95% CIs within the observed data range (interpolated), while dotted lines and lighter shading indicate estimates and 95% CIs beyond that time frame (extrapolated). Points represent study observations adjusted for diagnostic performance.

Table B18: Summary of risk of bias for included studies

Criteria	Vaginal discharge $N_{VD} = 87$		Urethral discharge $N_{UD} = 55$		Genital ulcer $N_{GU} = 80$	
	Yes	**No/unclear**	**Yes**	**No/unclear**	**Yes**	**No/unclear**
Study design						
Q1	37 (42.5%)	50 (57.5%)	32 (58.2%)	23 (41.8%)	40 (50.0%)	40 (50.0%)
Selection bias						
Q2	82 (94.3%)	5 (5.7%)	50 (90.9%)	5 (9.1%)	73 (91.2%)	7 (8.8%)
Q3	36 (41.4%)	51 (58.6%)	21 (38.2%)	34 (61.8%)	56 (70.0%)	24 (30.0%)
Q4	32 (36.8%)	55 (63.2%)	27 (49.1%)	28 (50.9%)	46 (57.5%)	34 (42.5%)
Q5	34 (39.1%)	53 (60.9%)	21 (38.2%)	34 (61.8%)	40 (50.0%)	40 (50.0%)
Measurement bias						
Q6	48 (55.2%)	39 (44.8%)	36 (65.5%)	19 (34.5%)	75 (93.8%)	5 (6.2%)
Q7	81 (93.1%)	6 (6.9%)	47 (85.5%)	8 (14.5%)	75 (93.8%)	5 (6.2%)
Q8	43 (49.4%)	44 (50.6%)	22 (40.0%)	33 (60.0%)	48 (60.0%)	32 (40.0%)
Precision						
Q9	61 (70.1%)	26 (29.9%)	39 (70.9%)	16 (29.1%)	46 (57.5%)	34 (42.5%)
Reporting quality						
Q10	51 (58.6%)	36 (41.4%)	37 (67.3%)	18 (32.7%)	52 (65.0%)	28 (35.0%)

Q1: Was the study specifically designed to estimate the outcome/s of interest? **Q2:** Were criteria for inclusion in the sample clearly defined? **Q3:** Were study participants and their setting described in sufficient detail to determine representativeness of the target population? **Q4:** Were participants recruited in an appropriate way? **Q5:** Was the participation rate adequate? **Q6:** Were objective, standard criteria used to define participant symptoms? **Q7:** Were the outcomes measured in a reliable and consistent way? **Q8:** Were participants tested for multiple pathogens to prevent misclassification of infection outcomes? **Q9:** Was the sample size sufficient to measure the outcome? **Q10:** Was the reporting of the outcome(s) unambiguous?


A
CS
Q1 2.3 (1.2-4.6)
Q2 1.6 (0.4-5.6)
Q3 0.4 (0.2-0.9)
Q4 0.7 (0.3-1.4)
Q5 1.1 (0.6-2.3)
Q6 1.9 (0.7-4.8)
Q7 0.1 (0.0-1.7)
Q8 5.7 (2.2-14.9)
Q9 0.5 (0.2-1.5)
Q10 0.4 (0.2-0.8)
BV
0.7 (0.3-1.5)
1.3 (0.2-8.0)
0.9 (0.3-2.2)
2.4 (1.2-4.9)
0.7 (0.3-1.6)
0.8 (0.3-2.3)
2.2 (0.6-7.7)
0.4 (0.2-1.1)
3.0 (1.2-7.4)
2.0 (0.7-5.7)
CA
0.6 (0.0-8.7)
2.6 (0.2-38.3)
1.1 (0.2-7.6)
2.2 (0.3-14.9)
0.8 (0.1-5.8)
1.9 (0.1-27.1)
2.0 (0.2-21.0)
CT
0.9 (0.5-1.5)
1.9 (0.3-11.6)
1.4 (0.7-2.8)
0.5 (0.2-0.9)
1.5 (0.7-2.9)
1.3 (0.6-2.9)
53.1 (10-276)
2.0 (0.9-4.7)
2.7 (1.3-5.7)
0.7 (0.3-1.5)
TV
1.1 (0.7-1.8)
0.6 (0.3-1.4)
0.8 (0.5-1.3)
1.3 (0.8-2.0)
0.7 (0.4-1.1)
0.9 (0.6-1.5)
0.5 (0.2-1.0)
4.0 (2.3-7.0)
1.6 (1.0-2.7)
0.6 (0.4-1.0)
NG
0.9 (0.5-1.4)
0.8 (0.3-1.9)
0.3 (0.2-0.6)
0.6 (0.3-1.0)
0.6 (0.3-1.0)
3.8 (1.9-7.7)
2.1 (0.4-10.8)
5.7 (2.6-12.5)
2.1 (1.0-4.4)
0.5 (0.3-0.8)
Vaginal discharge
MG
Q1 1.8 (0.3-9.7)
Q4 0.2 (0.0-1.3)
Q5 0.8 (0.1-5.3)
None
0.8 (0.1-4.4)
0.6 (0.1-3.4)
12.4 (2.5-61.0)
0.8 (0.1-7.8)
0.7 (0.1-4.8)
0.3 (0.0-4.1)
0.1 (0.0-2.6)
5.0 (0.8-32.0)
0.7 (0.1-4.4)
0.1 1 10 100
Adjusted odds ratio (log scale)


B
NG
Q1 0.6 (0.3-1.2)
Q2 0.6 (0.2-1.5)
Q3 0.6 (0.4-1.1)
Q4 0.9 (0.5-1.6)
Q5 0.9 (0.5-1.4)
Q6 1.6 (0.9-2.8)
Q7 2.2 (1.0-4.8)
Q8 1.5 (0.9-2.4)
Q9 1.2 (0.6-2.2)
Q10 0.8 (0.4-1.4)
CT
0.6 (0.2-1.7)
1.3 (0.3-4.9)
0.9 (0.5-1.7)
1.7 (0.7-4.2)
0.8 (0.4-1.6)
1.1 (0.5-2.7)
2.1 (0.6-7.0)
1.0 (0.5-2.0)
1.2 (0.5-2.8)
0.5 (0.2-1.2)
MG
0.5 (0.0-14.3)
2.8 (0.5-15.0)
0.3 (0.0-2.7)
0.2 (0.0-1.4)
15.4 (1.0-248.7)
0.5 (0.0-5.1)
Urethral discharge
TV
Q1 1.4 (0.3-5.8)
Q2 0.6 (0.1-7.1)
Q3 0.7 (0.3-1.7)
Q4 0.9 (0.3-2.4)
Q5 2.7 (1.1-6.4)
Q6 0.3 (0.1-0.9)
Q7 5.2 (2.0-13.0)
Q8 1.4 (0.5-3.9)
Q9 1.9 (0.7-5.1)
Q10 1.5 (0.6-3.9)
None
35.2 (0.0-37421)
0.7 (0.0-27.8)
5.7 (0.1-440.4)
2.9 (0.1-108.3)
0.1 (0.0-5.6)
0.8 (0.1-7.7)
0.04 (0.0-5.3)
0.3 (0.0-21.8)
1.3 (0.1-27.9)
0.1 1 10 100
Adjusted odds ratio (log scale)

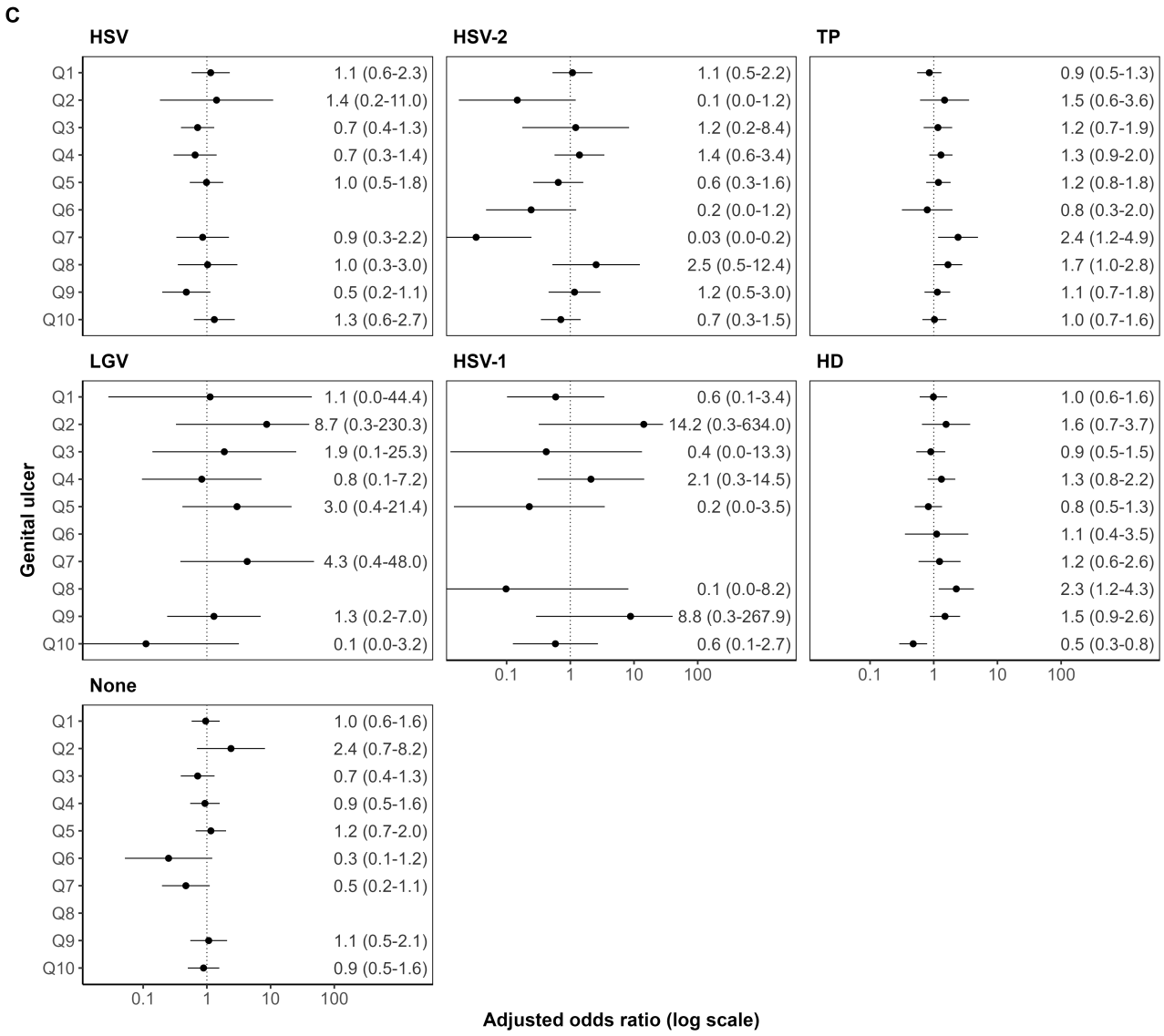


Figure B5: Effect of risk of bias criteria on the odds of diagnosis per pathogen, among symptomatic adults in SSA

Adjusted odds of diagnosis per pathogen among studies rated "no or unclear" compared to "yes" for each criterion in the critical appraisal tool, among adults of non-stratified or unmeasured HIV status symptomatic with (A) vaginal discharge, (B) urethral discharge, and (C) genital ulcer. Odds estimated via generalised linear mixed-effects model for each symptom. Points represent the adjusted odds of diagnosis among studies rated "no or unclear" compared to studies rated "yes". Solid lines represent 95% CIs. Blank criteria signify that all studies were rated "yes". X-axis truncated.

Q1: Was the study specifically designed to estimate the outcome/s of interest [study design]? **Q2:** Were the criteria for inclusion in the sample clearly defined [selection bias]? **Q3:** Were study participants and their setting described in sufficient detail to determine representativeness of the target population [selection bias]? **Q4:** Were study participants recruited in an appropriate way [selection bias]? **Q5:** Was the participation rate adequate [selection bias]? **Q6:** Were objective, standard criteria used to define participant symptoms [measurement bias]? **Q7:** Were the outcomes measured in a reliable and consistent way [measurement bias]? **Q8:** Were participants tested for multiple pathogens to prevent misclassification of infection outcomes [measurement bias]? **Q9:** Was the sample size sufficient to measure the outcome [study precision]? **Q10:** Was the reporting of the outcome(s) unambiguous [reporting quality]?

Appendix C

Supplement to Chapter 4

C.1 Supplementary methods

C.1.1 Survey variable definitions

Outcome variables were defined based on the survey questions detailed in Table C1. A subset of included surveys asked respondents who reported seeking advice or treatment for genital symptoms in the past 12 months to specify the locations where they received care. For these surveys, treatment locations were categorised into five distinct sectors: (1) public sector, covering government, non-profit and faith-based services; (2) private sector, covering private hospitals, clinics, doctors, or other commercial health services; (3) pharmacy or shop, covering pharmacies, drug stores, and informal medicine vendors; and (4) traditional or other, covering traditional healers and non-medical sources; and (5) multiple sectors. Individuals who reported seeking care from any location within a single sector were classified as utilising that sector, while those who reported accessing locations across more than one sector were categorised as "multiple sector". Table C2 presents a detailed list of the specific locations classified within each sector.

Table C1: Definition of outcome variables from survey questions

Variable	Identifier (Questionnaire)	Question
Genital ulcer in past year	v763b (IR)	Sometimes women have a genital sore or ulcer. During the past year, have you had a genital sore or ulcer?
	mv763b (MR)	Sometimes men have a sore or ulcer near their penis. During the past year, have you had a sore or ulcer on or near your penis?
Genital discharge in past year	v763c (IR)	Sometimes women experience a bad-smelling abnormal genital discharge. During the past year, have you had a bad-smelling abnormal genital discharge?
	mv763c (MR)	Sometimes men experience an abnormal discharge from their penis. During the past year, have you had an abnormal discharge from your penis?
Treatment seeking for genital symptom in past year	v770 (IR) mv770 (MR)	The last time you had (PROBLEM), did you seek any kind of advice or treatment?
Sector for treatment of genital symptom in past year	v770a-x (IR) mv770a-x (MR)	Where did you go? (See Table C2)

IR: Individual women's recode file, MR: Men's recode file.

Table C2: Definition of treatment sector variable from locations of advice or treatment

Category	Locations
Public sector	Government: • Hospitals (central, district, provincial, regional, referral) • Health centres, clinics or posts • Maternity clinics and maternal health services • Family planning clinics • Military health facilities • Laboratories and diagnostic centres • Dispensaries • Youth centres and support groups • HIV testing centres • Mobile clinics or outreach services • Community health workers, fieldworkers, door-to-door or village health services Non-profit or faith-based: • Hospitals or clinics • Family planning association clinics (PPAG, ABPF, etc.) • AIDS information centres • HIV testing centres • Community health workers or fieldworkers
Private sector	• Hospitals, clinics, or doctor practices • Doctors, nurses, or midwives • Nursing or maternity homes • Laboratories and diagnostic centres • Independent HIV or STI testing centres • Blood transfusion services • Workplace or corporate health services • University or school health services • Mobile clinics or outreach services • Other private sources (unspecified)
Pharmacy or shop	• Pharmacy, drug store, or chemist • Private dispensary • Medical cabinets • Shops, markets, kiosks, or bars • Street vendors, drug peddlers, or "pharmacy on the ground"
Traditional or other	• Traditional practitioners, healers, or herbalists • Traditional birth attendants • Spiritual medicine centres • Home • Friend, relative, or partner • Other (unspecified)

Table C3: Classification of included countries by sub-region in SSA

Sub-region	ISO3	Country
Western Africa	BEN	Benin
	BFA	Burkina Faso
	CIV	Cote d'Ivoire
	GMB	Gambia
	GHA	Ghana
	GIN	Guinea
	LBR	Liberia
	MLI	Mali
	MRT	Mauritania
	NER	Niger
	NGA	Nigeria
	SEN	Senegal
	SLE	Sierra Leone
	TGO	Togo
Central Africa	AGO	Angola
	CMR	Cameroon
	TCD	Chad
	COG	Congo
	COD	Democratic Republic of the Congo
	GAB	Gabon
	STP	Sao Tome and Principe
Eastern Africa	BDI	Burundi
	COM	Comoros
	ETH	Ethiopia
	KEN	Kenya
	MDG	Madagascar
	MWI	Malawi
	MOZ	Mozambique
	RWA	Rwanda
	TZA	Tanzania
	UGA	Uganda
	ZMB	Zambia
	ZWE	Zimbabwe
Southern Africa	SWZ	Eswatini
	LSO	Lesotho
	NAM	Namibia
	ZAF	South Africa

ISO3 refers to three-letter country codes defined by International Organization for Standardization (ISO 3166-1 alpha-3).[896] Countries classified into sub-regions according to UN M49 Standard.[282]

C.2 Supplementary results

C.2.1 Survey inclusion

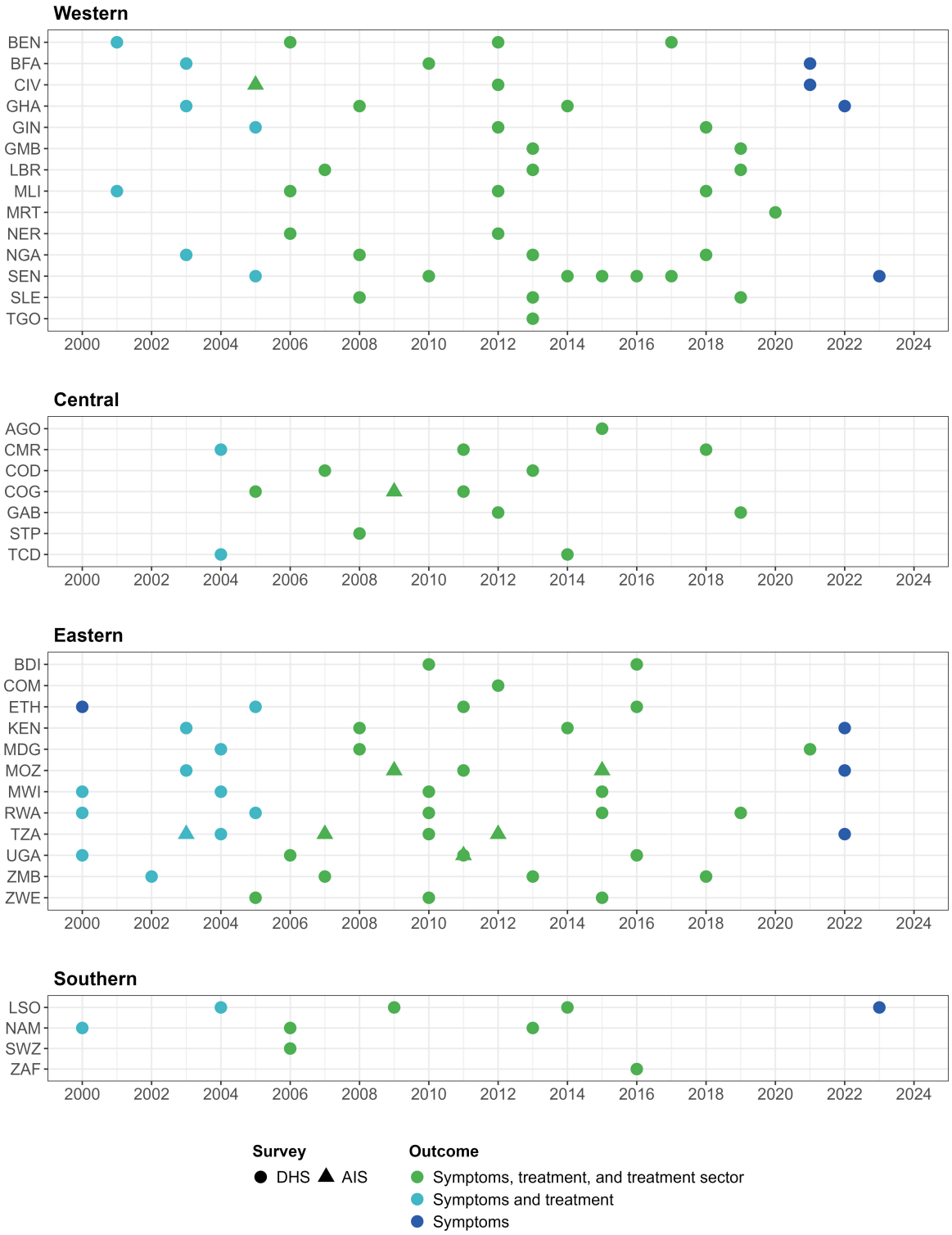


Figure C1: Surveys included in the analysis by country, year, and outcome

Surveys with questions on genital symptoms, treatment-seeking, and sector of treatment, by country and year, in SSA between 2000 and 2024. Points depict individual surveys, colours depict outcomes assessed, and shapes depict survey type. ISO3 codes defined in Table C3.

Table C4: Number of included surveys and total sample sizes by country and outcome

Country ISO3	Symptoms				Treatment				Sector			
	Female		Male		Female		Male		Female		Male	
	N	Sample	N	Sample	N	Sample	N	Sample	N	Sample	N	Sample
Western												
BEN	4	49 390	4	15 042	4	2 854	4	628	3	1 423	3	305
BFA	3	40 856	3	12 539	2	1 930	2	125	1	766	1	37
CIV	3	27 163	2	9 531	2	3 308	1	250	2	1 757	1	180
GMB	2	16 427	2	5 226	2	1 538	2	210	2	1 003	2	118
GHA	4	30 102	4	14 318	3	3 034	3	606	2	1 760	2	310
GIN	3	24 376	3	7 666	3	7 725	3	727	2	3 646	2	378
LBR	3	22 966	3	12 105	3	8 753	3	1 973	3	7 409	3	1 567
MLI	4	43 538	4	11 196	4	9 777	4	588	3	3 904	3	220
MRT	1	11 437	-	-	1	2 708	-	-	1	1 089	-	-
NER	2	17 940	2	4 514	2	808	2	101	2	372	2	44
NGA	4	103 136	4	32 778	4	8 402	4	1 168	3	4 845	3	742
SEN	7	67 609	7	18 007	6	7 667	6	261	5	3 116	5	97
SLE	3	35 908	3	13 465	3	6 890	3	1 540	3	5 022	3	1 294
TGO	1	8 379	1	3 142	1	1 552	1	93	1	861	1	58
Sub-total	44	499 227	42	159 529	40	66 946	38	8 270	33	36 973	31	5 350
Central												
AGO	1	13 072	1	4 864	1	1 174	1	268	1	672	1	165
CMR	3	34 214	3	13 914	3	3 912	3	1 005	2	1 738	2	492
TCD	2	20 476	2	4 770	2	892	2	125	1	194	1	47
COG	3	22 757	2	6 913	3	4 413	2	657	3	3 165	2	551
COD	2	25 346	2	10 311	2	3 156	2	768	2	1 904	2	512
GAB	2	16 199	2	9 517	2	2 467	2	648	2	1 609	2	461
STP	1	2 286	1	1 757	1	259	1	47	1	229	1	31
Sub-total	14	134 350	13	52 046	14	16 273	13	3 518	12	9 511	11	2 259
Eastern												
BDI	2	18 598	2	6 884	2	1 812	2	307	2	993	2	152
COM	1	3 712	1	1 543	1	379	1	73	1	214	1	51
ETH	3	35 110	4	21 853	3	946	3	389	2	291	2	156
KEN	4	67 122	4	26 571	3	1 295	3	313	2	704	2	184
MDG	3	39 110	3	15 325	3	1 593	3	761	2	787	2	482
MWI	4	63 723	4	17 149	4	6 734	4	1 167	2	2 611	2	401
MOZ	5	47 797	4	14 120	4	2 533	3	644	2	616	2	395
RWA	5	45 019	5	16 801	5	4 079	5	838	3	2 137	3	393
TZA	6	52 470	3	8 512	5	2 007	2	182	3	877	1	68
UGA	5	46 428	4	9 468	5	10 119	4	1 020	4	6 255	3	642
ZMB	4	39 103	4	27 652	4	1 679	4	1 546	3	910	3	1 120
ZWE	3	22 557	3	16 238	3	1 860	3	1 101	3	1 013	3	609
Sub-total	45	480 749	41	182 116	42	35 036	37	8 341	29	17 408	26	4 653
Southern												
SWZ	1	4 111	1	2 926	1	447	1	273	1	345	1	224
LSO	4	23 759	4	9 267	3	2 611	3	769	2	1 256	2	334
NAM	2	16 167	3	9 069	2	1 235	3	325	2	827	2	196
ZAF	1	7 476	1	2 753	1	831	1	158	1	614	1	109
Sub-total	8	51 513	9	24 015	7	5 124	8	1 525	6	3 042	6	863
Sub-Saharan Africa												
Total	111	1 165 839	105	417 706	103	123 379	96	21 654	80	66 934	74	13 125

Surveys with questions on genital symptoms, treatment-seeking, and sector of treatment in sub-Saharan Africa, 2000-2024. ISO3 codes defined in Table C3. N: number of surveys, Sample: total sexually active respondents aged 15-49 years who responded to survey questions addressing each outcome.

C.2.2 Regression results

Table C5: Adjusted odds of genital discharge, genital ulcer, and treatment for genital symptoms, estimated via generalised linear mixed-effects models

Variable	Genital discharge aOR (95% CI)	Genital ulcer aOR (95% CI)	Treatment aOR (95% CI)
Intercept	0.04 (0.03-0.04)	0.02 (0.02-0.03)	2.25 (1.93-2.61)
Sex			
Male	Ref	Ref	Ref
Female	3.57 (3.29-3.88)	3.05 (2.78-3.35)	0.78 (0.69-0.88)
Sex:sub-region			
Male:Western	Ref	Ref	Ref
Male:Central	1.00 (0.80-1.24)	1.22 (0.96-1.55)	1.94 (1.53-2.46)
Male:Eastern	0.86 (0.74-1.00)	1.37 (1.16-1.62)	1.09 (0.92-1.29)
Male:Southern	1.39 (1.05-1.82)	2.20 (1.63-2.95)	1.42 (1.05-1.93)
Female:Western	Ref	Ref	Ref
Female:Central	0.73 (0.60-0.89)	0.61 (0.49-0.76)	1.45 (1.18-1.78)
Female:Eastern	0.42 (0.36-0.48)	0.58 (0.50-0.68)	0.99 (0.86-1.14)
Female:Southern	0.71 (0.55-0.92)	0.89 (0.67-1.17)	1.84 (1.39-2.43)
Sex:age			
Male:15-19	1.10 (1.03-1.18)	0.94 (0.87-1.02)	0.35 (0.31-0.40)
Male:20-24	1.33 (1.25-1.41)	1.16 (1.09-1.24)	0.67 (0.60-0.76)
Male:25-29	1.21 (1.14-1.29)	1.18 (1.11-1.27)	0.96 (0.85-1.08)
Male:30-34	Ref	Ref	Ref
Male:35-39	0.81 (0.76-0.87)	0.90 (0.83-0.96)	0.96 (0.84-1.09)
Male:40-44	0.62 (0.58-0.67)	0.67 (0.62-0.73)	0.99 (0.85-1.14)
Male:45-49	0.54 (0.49-0.58)	0.62 (0.57-0.67)	0.75 (0.64-0.88)
Female:15-19	0.96 (0.93-0.99)	0.88 (0.85-0.91)	0.51 (0.48-0.54)
Female:20-24	1.09 (1.06-1.12)	1.03 (1.00-1.07)	0.88 (0.84-0.93)
Female:25-29	1.07 (1.04-1.10)	1.05 (1.01-1.08)	0.99 (0.94-1.04)
Female:30-34	Ref	Ref	Ref
Female:35-39	0.91 (0.88-0.93)	0.94 (0.91-0.97)	0.93 (0.88-0.98)
Female:40-44	0.80 (0.78-0.83)	0.86 (0.83-0.90)	0.85 (0.80-0.90)
Female:45-49	0.61 (0.59-0.63)	0.68 (0.65-0.71)	0.80 (0.74-0.85)
Sex:sub-region:residence[*]			
Male:Western:Urban	0.92 (0.87-0.97)	0.79 (0.73-0.85)	-
Male:Central:Urban	1.05 (0.95-1.17)	1.30 (1.16-1.46)	-
Male:Eastern:Urban	0.92 (0.86-0.98)	0.85 (0.80-0.91)	-
Male:Southern:Urban	0.80 (0.69-0.92)	0.63 (0.54-0.74)	-
Female:Western:Urban	1.20 (1.17-1.23)	1.04 (1.01-1.07)	-
Female:Central:Urban	1.41 (1.35-1.48)	1.16 (1.08-1.24)	-
Female:Eastern:Urban	1.08 (1.05-1.12)	0.94 (0.91-0.98)	-
Female:Southern:Urban	0.92 (0.86-0.99)	0.73 (0.65-0.80)	-

Continued...

Variable	Genital discharge aOR (95% CI)	Genital ulcer aOR (95% CI)	Treatment aOR (95% CI)
Sex:sub-region:year[†]			
Male:Western:Year	1.04 (1.03-1.06)	1.07 (1.05-1.09)	0.97 (0.95-0.99)
Male:Central:Year	1.01 (0.97-1.05)	1.01 (0.97-1.06)	0.93 (0.89-0.97)
Male:Eastern:Year	1.03 (1.02-1.05)	1.04 (1.02-1.06)	0.99 (0.97-1.01)
Male:Southern:Year	1.06 (1.02-1.11)	1.05 (1.01-1.10)	0.97 (0.91-1.03)
Female:Western:Year	1.05 (1.04-1.07)	1.04 (1.02-1.06)	1.01 (0.99-1.03)
Female:Central:Year	1.01 (0.97-1.05)	1.00 (0.96-1.05)	0.98 (0.94-1.02)
Female:Eastern:Year	1.04 (1.02-1.05)	1.03 (1.02-1.05)	1.00 (0.98-1.01)
Female:Southern:Year	1.05 (1.01-1.09)	1.04 (1.00-1.09)	1.00 (0.93-1.06)
Model variance			
τ^2 fixed	0.51 (34.4%)	0.30 (21.5%)	0.16 (17.6%)
τ^2 random	0.96 (64.9%)	1.07 (77.9%)	0.70 (77.3%)
country	0.68	0.80	0.55
country.year	$\leq$0.001	$\leq$0.001	$\leq$0.001
country.sex	0.21	0.20	0.10
survey	0.07	0.08	0.06
τ^2 distribution	0.01 (0.6%)	0.01 (0.6%)	0.05 (5.1%)
τ^2 total	1.47 (100%)	1.38 (100%)	0.91 (100%)
Number groups			
N country	37	37	37
N survey	113	113	104

Separate generalised linear mixed-effects models for each outcome: (1) self-reported genital discharge in the past year, among ever sexually active 15-49 year-olds; (2) self-reported genital ulcer in the past year, among ever sexually active 15-49 year-olds; and (3) self-reported care or treatment among 15-49 year-olds with genital symptoms. [*]Reference for residence is rural. Model for probability of treatment did not include residence as a fixed effect. [†]Year is centred at 2011. aOR: adjusted odds ratio, CI: confidence interval, Ref: reference category.

Table C6: Adjusted rates of treatment sector utilisation, estimated via generalized linear mixed-effects multinomial model.

Variable	Adjusted rate (95% CI)	Variable	Adjusted rate (95% CI)
Male:Western:Private	0.11 (0.10-0.13)	Male:Western:Private:Year	0.97 (0.95-0.99)
Male:Western:Public	0.50 (0.46-0.55)	Male:Western:Public:Year	1.00 (0.99-1.01)
Male:Western:Pharmacy	0.07 (0.05-0.08)	Male:Western:Pharmacy:Year	1.05 (1.03-1.07)
Male:Western:Traditional	0.13 (0.11-0.15)	Male:Western:Traditional :Year	0.97 (0.95-1.00)
Male:Western:Multiple	0.06 (0.05-0.07)	Male:Western:Multiple:Year	0.97 (0.94-1.00)
Male:Central:Private	0.11 (0.09-0.14)	Male:Central:Private:Year	1.01 (0.96-1.07)
Male:Central:Public	0.52 (0.47-0.58)	Male:Central:Public:Year	0.99 (0.97-1.02)
Male:Central:Pharmacy	0.07 (0.05-0.09)	Male:Central:Pharmacy:Year	1.03 (0.99-1.08)
Male:Central:Traditional	0.10 (0.08-0.13)	Male:Central:Traditional :Year	0.98 (0.93-1.03)
Male:Central:Multiple	0.06 (0.05-0.08)	Male:Central:Multiple:Year	0.92 (0.87-0.97)
Male:Eastern:Private	0.11 (0.09-0.12)	Male:Eastern:Private:Year	1.01 (0.99-1.03)
Male:Eastern:Public	0.61 (0.57-0.66)	Male:Eastern:Public:Year	1.01 (1.00-1.02)
Male:Eastern:Pharmacy	0.04 (0.03-0.05)	Male:Eastern:Pharmacy:Year	1.07 (1.03-1.11)
Male:Eastern:Traditional	0.08 (0.07-0.10)	Male:Eastern:Traditional :Year	0.96 (0.94-0.98)
Male:Eastern:Multiple	0.05 (0.05-0.06)	Male:Eastern:Multiple:Year	0.92 (0.88-0.95)
Male:Southern:Private	0.11 (0.08-0.14)	Male:Southern:Private:Year	0.94 (0.87-1.02)
Male:Southern:Public	0.73 (0.67-0.81)	Male:Southern:Public:Year	1.00 (0.97-1.04)
Male:Southern:Pharmacy	0.02 (0.01-0.03)	Male:Southern:Pharmacy:Year	0.95 (0.83-1.08)
Male:Southern:Traditional	0.06 (0.05-0.08)	Male:Southern:Traditional :Year	1.03 (0.96-1.11)
Male:Southern:Multiple	0.03 (0.02-0.04)	Male:Southern:Multiple:Year	0.94 (0.81-1.08)
Female:Western:Private	0.08 (0.08-0.09)	Female:Western:Private:Year	0.97 (0.95-0.98)
Female:Western:Public	0.64 (0.62-0.66)	Female:Western:Public:Year	1.00 (0.99-1.01)
Female:Western:Pharmacy	0.04 (0.03-0.04)	Female:Western:Pharmacy:Year	1.07 (1.05-1.08)
Female:Western:Traditional	0.08 (0.08-0.09)	Female:Western:Traditional :Year	0.99 (0.98-1.00)
Female:Western:Multiple	0.04 (0.03-0.04)	Female:Western:Multiple:Year	0.96 (0.95-0.98)
Female:Central:Private	0.09 (0.07-0.10)	Female:Central:Private:Year	1.07 (1.03-1.10)
Female:Central:Public	0.68 (0.65-0.72)	Female:Central:Public:Year	0.99 (0.98-1.01)
Female:Central:Pharmacy	0.03 (0.02-0.04)	Female:Central:Pharmacy:Year	1.03 (1.00-1.06)
Female:Central:Traditional	0.06 (0.05-0.07)	Female:Central:Traditional :Year	1.02 (0.98-1.06)
Female:Central:Multiple	0.04 (0.03-0.04)	Female:Central:Multiple:Year	0.93 (0.89-0.98)
Female:Eastern:Private	0.08 (0.07-0.09)	Female:Eastern:Private:Year	1.00 (0.99-1.01)
Female:Eastern:Public	0.73 (0.70-0.75)	Female:Eastern:Public:Year	1.00 (1.00-1.01)
Female:Eastern:Pharmacy	0.01 (0.01-0.02)	Female:Eastern:Pharmacy:Year	1.06 (1.03-1.10)
Female:Eastern:Traditional	0.05 (0.05-0.06)	Female:Eastern:Traditional :Year	0.96 (0.94-0.98)
Female:Eastern:Multiple	0.03 (0.02-0.03)	Female:Eastern:Multiple:Year	0.97 (0.95-1.00)
Female:Southern:Private	0.10 (0.08-0.12)	Female:Southern:Private:Year	0.97 (0.92-1.03)
Female:Southern:Public	0.82 (0.78-0.86)	Female:Southern:Public:Year	1.00 (0.98-1.03)
Female:Southern:Pharmacy	0.02 (0.02-0.03)	Female:Southern:Pharmacy:Year	1.11 (0.99-1.26)
Female:Southern:Traditional	0.02 (0.01-0.02)	Female:Southern:Traditional :Year	0.96 (0.89-1.03)
Female:Southern:Multiple	0.02 (0.01-0.02)	Female:Southern:Multiple:Year	1.07 (0.96-1.19)

Continued...

Model variance	
τ^2 fixed	1.21 (60.9%)
τ^2 random	0.57 (28.6%)
sector:country	0.46
sector:country.sex	0.12
survey	$\leq$0.001
τ^2 distribution	0.21 (10.6%)
τ^2 total	1.98 (100%)
Number groups	
N sector:country	185
N survey	81

Adjusted rates are direct parameters from multinomial-Poisson transformation model, prior to prediction and normalisation. Year is centred at 2011. CI: confidence interval.

C.2.3 Regional estimates

Table C7: Relative ratio for prevalence of genital symptoms in urban compared to rural residence in SSA

Sub-region	Female	Male	Overall
Discharge prevalence			
WA	1.19 (1.13-1.25)	1.01 (0.93-1.09)	1.10 (1.03-1.16)
CA	1.47 (1.31-1.64)	1.13 (0.97-1.31)	1.29 (1.15-1.46)
EA	1.07 (1.00-1.13)	1.00 (0.92-1.08)	1.03 (0.97-1.10)
SA	0.92 (0.84-1.00)	0.76 (0.66-0.88)	0.84 (0.76-0.92)
SSA	1.35 (1.25-1.45)	1.08 (0.99-1.17)	1.21 (1.12-1.30)
Ulcer prevalence			
WA	1.07 (1.00-1.13)	0.84 (0.76-0.92)	0.95 (0.88-1.01)
CA	1.25 (1.08-1.42)	1.39 (1.17-1.64)	1.32 (1.15-1.51)
EA	0.90 (0.84-0.96)	0.95 (0.87-1.04)	0.92 (0.86-0.99)
SA	0.73 (0.64-0.82)	0.59 (0.50-0.69)	0.66 (0.58-0.73)
SSA	1.10 (1.01-1.20)	0.96 (0.87-1.06)	1.03 (0.94-1.12)

Estimates are for 2020 and ratios derived from model simulations. WA: Western Africa, CA: Central Africa, EA: Eastern Africa, SA: Southern Africa, SSA: Sub-Saharan Africa, CI: confidence interval.

Table C8: Correlation between reported discharge prevalence and aetiologic prevalence

Group	Chlamydia		Gonorrhoea		Trichomoniasis	
	n	r (95% CI)	n	r (95% CI)	n	r (95% CI)
Vaginal discharge						
Sub-region						
Western and Central	29	0.08 (-0.29-0.44)	29	-0.23 (-0.55-0.15)	64	-0.03 (-0.27-0.22)
Eastern	86	0.09 (-0.12-0.30)	86	0.18 (-0.03-0.38)	75	-0.03 (-0.25-0.20)
Southern	46	0.47 (0.21-0.67)	47	0.40 (0.12-0.61)	42	-0.47 (-0.68–0.19)
Residence						
Urban	104	0.15 (-0.04-0.34)	103	0.14 (-0.05-0.33)	118	-0.02 (-0.20-0.16)
Rural	43	0.26 (-0.04-0.52)	43	0.27 (-0.03-0.53)	42	-0.07 (-0.37-0.24)
Non-stratified	14	0.17 (-0.40-0.64)	16	0.08 (-0.43-0.55)	21	-0.07 (-0.49-0.37)
Overall	161	0.19 (0.04-0.33)	162	0.17 (0.01-0.31)	181	-0.04 (-0.19-0.11)
Urethral discharge						
Sub-region						
Western and Central	3	-	2	-	3	-
Eastern	15	0.26 (-0.29-0.68)	15	-0.03 (-0.53-0.49)	5	0.18 (-0.83-0.92)
Southern	14	0.58 (0.08-0.85)	13	0.13 (-0.45-0.63)	10	-0.49 (-0.85-0.21)
Residence						
Urban	17	0.39 (-0.12-0.73)	15	0.03 (-0.49-0.53)	11	0.10 (-0.53-0.66)
Rural	11	0.38 (-0.29-0.80)	11	0.20 (-0.45-0.71)	5	-0.42 (-0.95-0.74)
Non-stratified	4	1.00 (0.96-1.00)	4	0.87 (-0.55-1.00)	2	-
Overall	32	0.43 (0.10-0.68)	30	0.22 (-0.15-0.54)	18	-0.25 (-0.64-0.24)

Aetiologic prevalence observations diagnosed using any test. n: number of observations, r: Pearson's correlation coefficient, calculated for values on the logit scale, CI: confidence interval.

C.2.4 National estimates

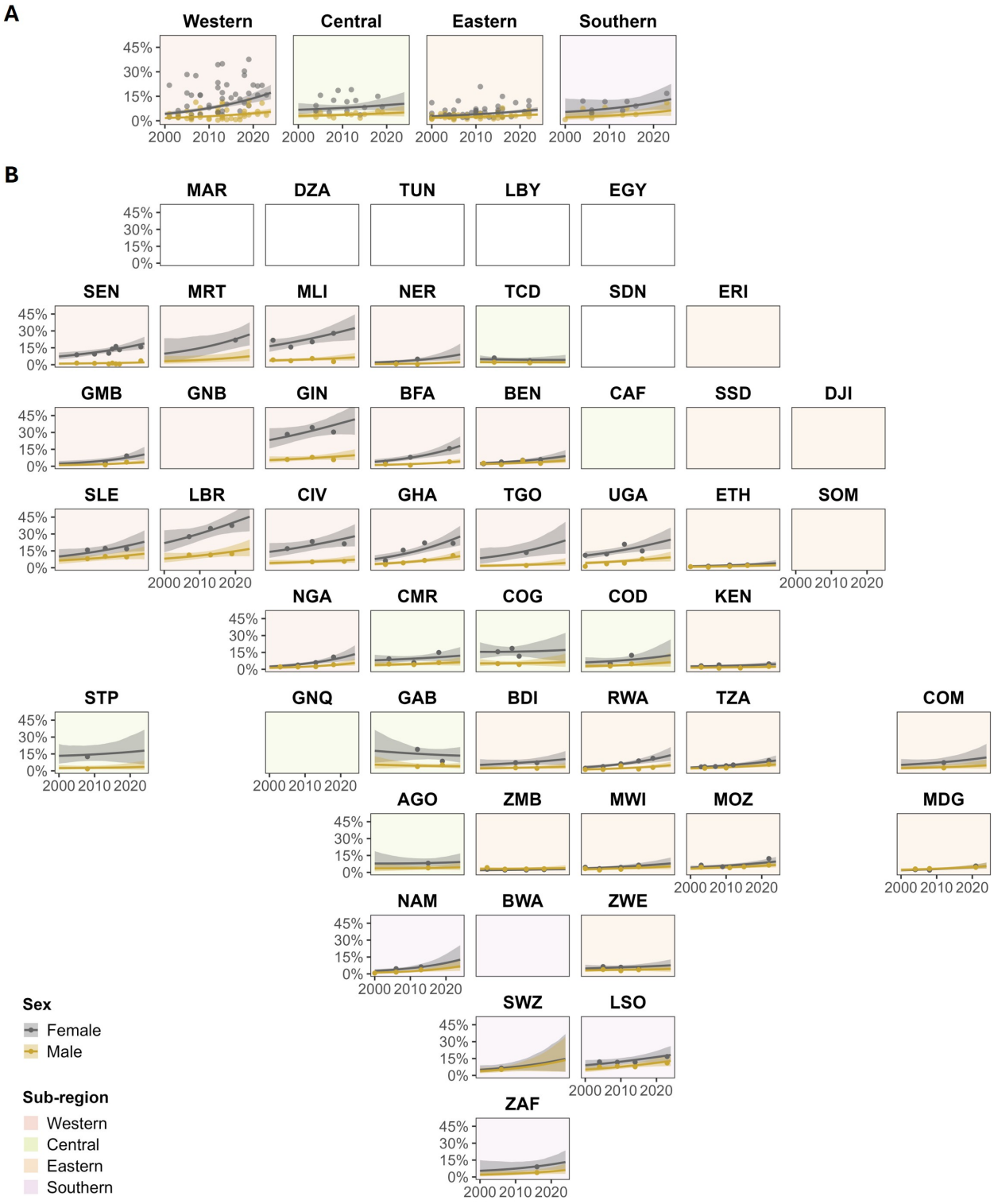


Figure C2: Prevalence of genital discharge in sub-Saharan Africa, 2000-2024.

Estimated prevalence of self-reported genital discharge at (A) sub-regional and (B) national levels, among ever sexually active 15-49 year-old males and females in sub-Saharan Africa between 2000 and 2024. National estimates generated using generalised linear mixed-effects model. Sub-regional estimates represent sex- and year-matched population-weighted means. Points depict survey observations. Lines and shaded areas depict mean estimate with 95% confidence interval. Y-axes truncated at 45%. ISO3 codes defined in Table C3.

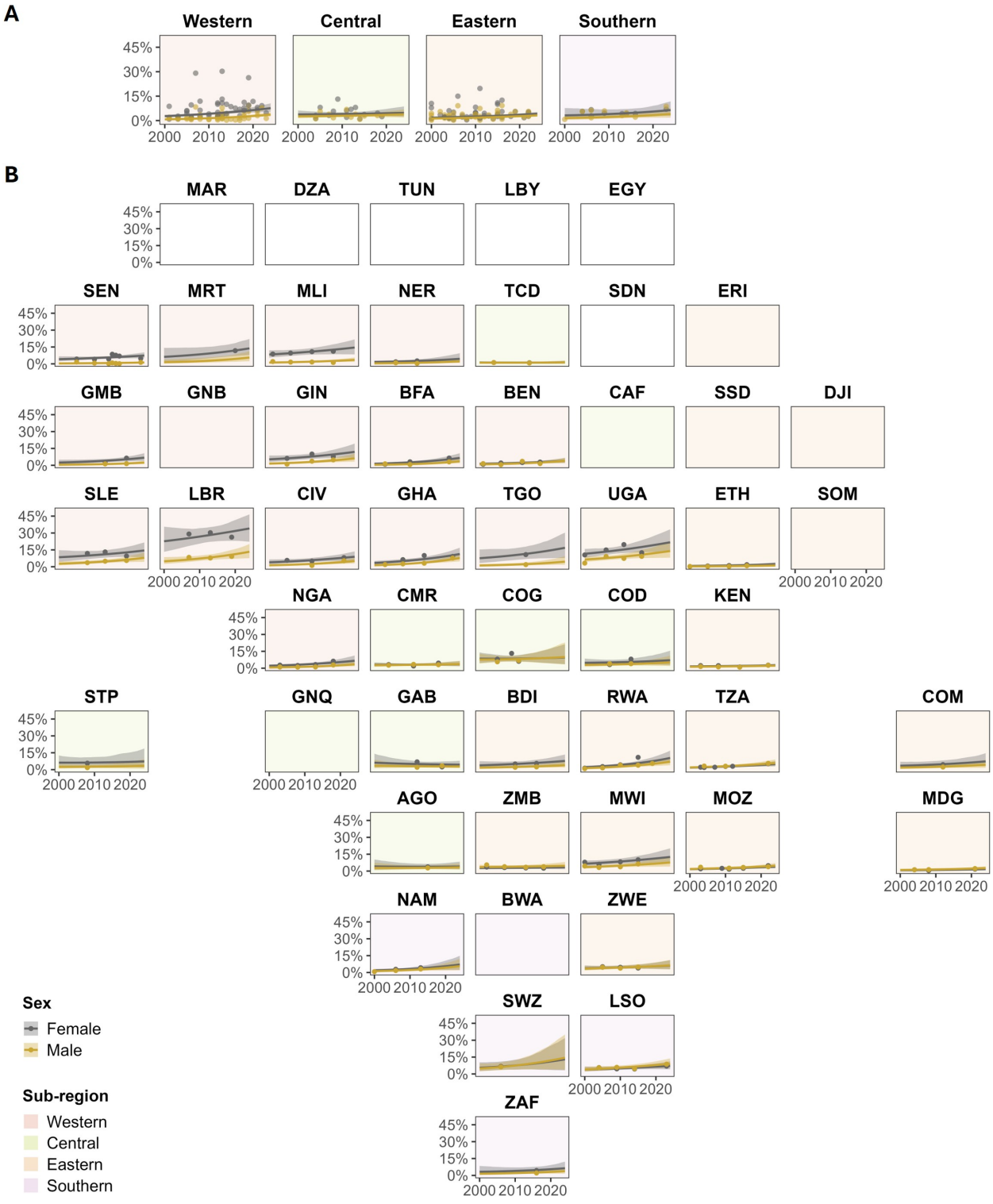


Figure C3: Prevalence of genital ulcer in sub-Saharan Africa, 2000-2024.

Estimated prevalence of self-reported genital ulcer at (A) sub-regional and (B) national levels, among ever sexually active 15-49 year-old males and females in sub-Saharan Africa between 2000 and 2024. National estimates generated using generalised linear mixed-effects model. Sub-regional estimates represent sex- and year-matched population-weighted means. Points depict survey observations. Lines and shaded areas depict mean estimate with 95% confidence interval. Y-axes truncated at 45%. ISO3 codes defined in Table C3.

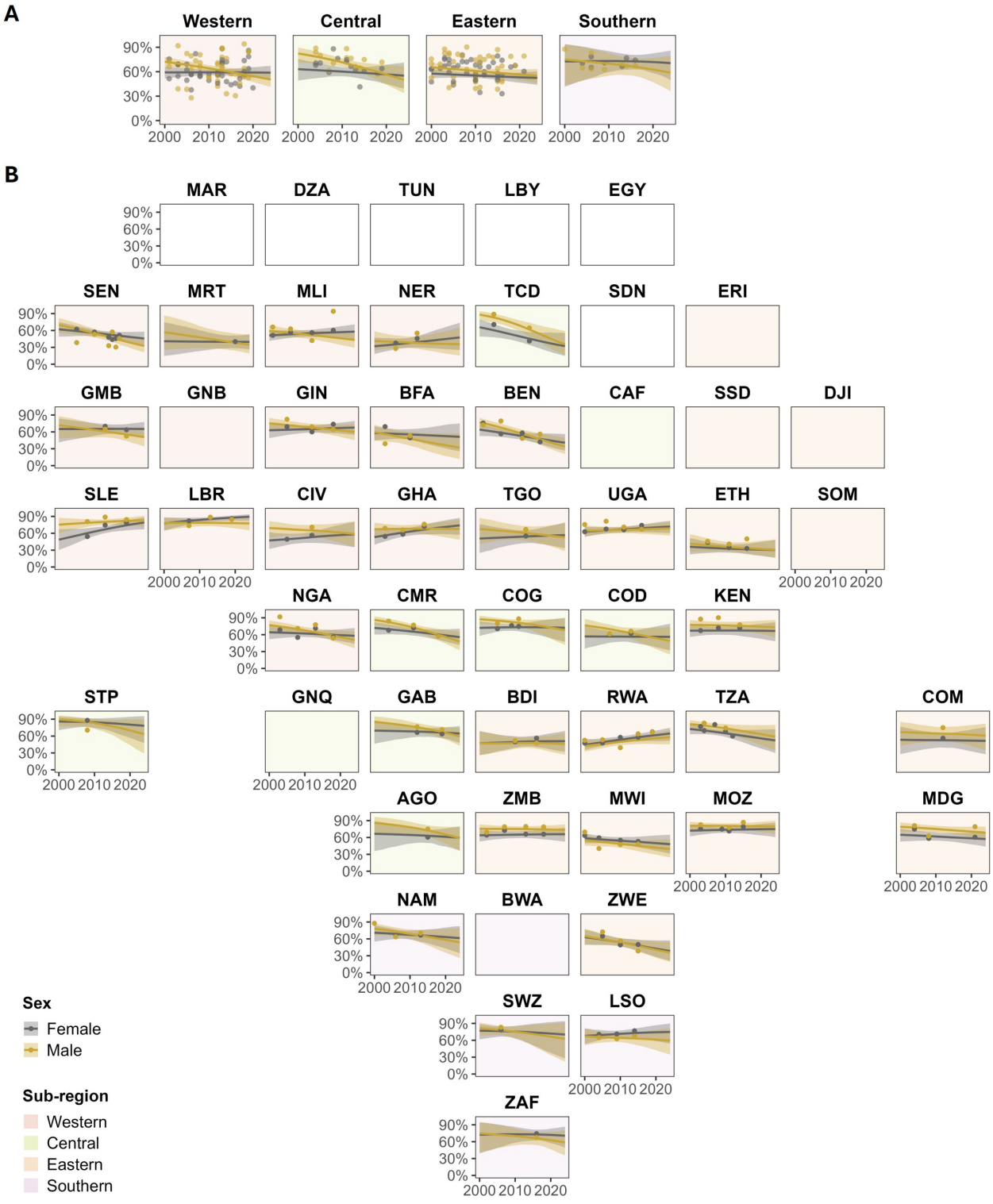


Figure C4: Proportion treated for genital symptoms in sub-Saharan Africa, 2000-2024.

Estimated proportion self-reporting care or treatment for genital symptoms at (A) sub-regional and (B) national levels, among symptomatic ever sexually active 15-49 year-old males and females in sub-Saharan Africa between 2000 and 2024. National estimates generated using generalised linear mixed-effects model. Sub-regional estimates represent sex- and year-matched population-weighted means. Points depict survey observations. Lines and shaded areas depict mean estimate with 95% confidence interval. ISO3 codes defined in Table C3.

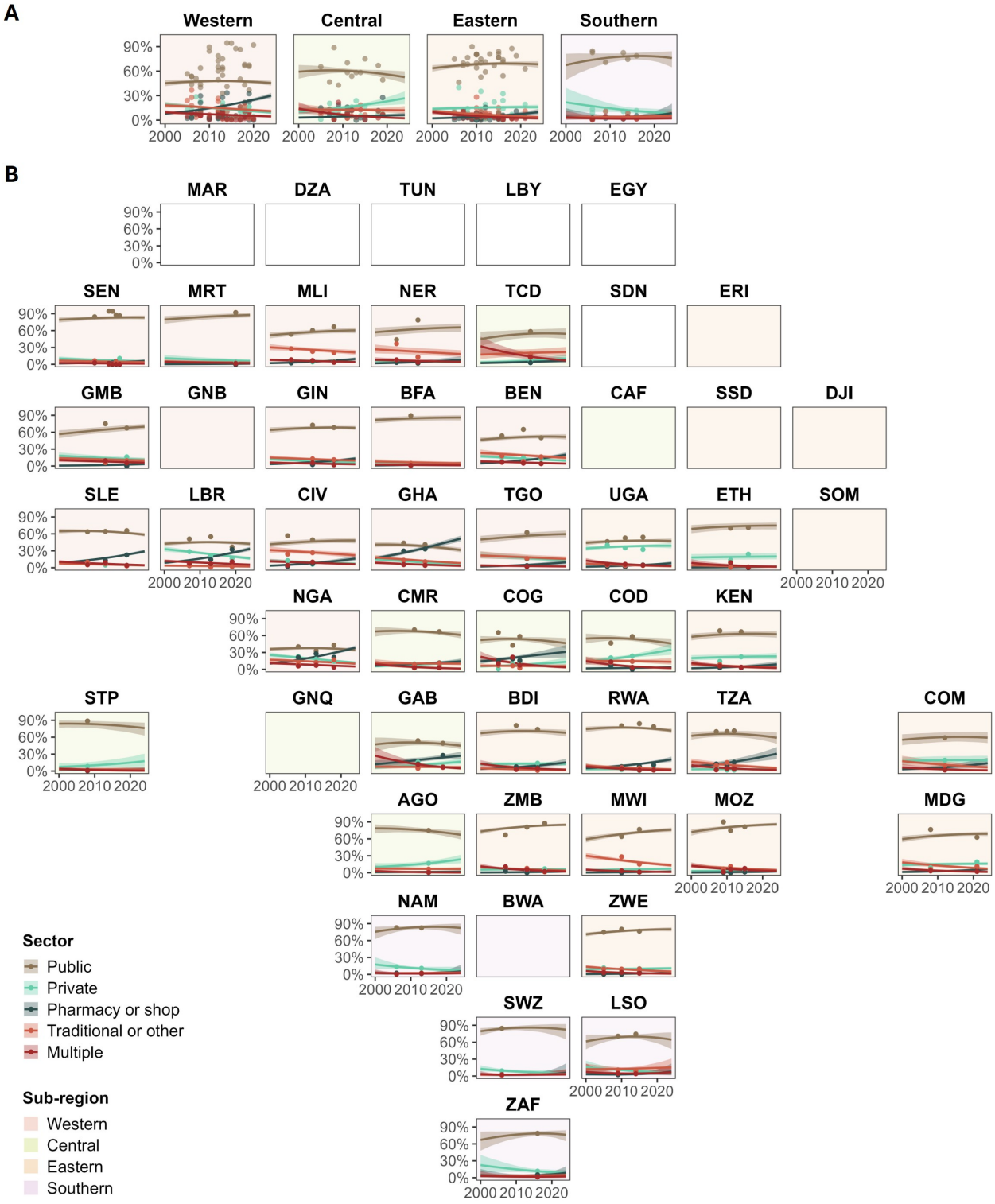


Figure C5: Health sectors utilised for treatment in sub-Saharan Africa, 2000-2024.

Distribution of health sectors utilised for care or treatment of genital symptoms at (A) sub-regional and (B) national levels, among symptomatic and treated ever sexually active 15-49 year-olds in sub-Saharan Africa between 2000 and 2024. National estimates generated using generalised mixed-effects multinomial model with the multinomial-Poisson transformation. Sub-regional estimates represent sex- and year-matched population-weighted means. Points depict survey observations. Lines and shaded areas depict mean estimate with 95% confidence interval. ISO3 codes defined in Table C3.

Table C9: Estimated national prevalence of genital discharge in 2000 and 2020

ISO3	Female (%)		Male (%)		Overall (%)	
	2000	2020	2000	2020	2000	2020
Western Africa						
BEN	2.6 (1.8-3.7)	7.3 (4.9-10.7)	1.8 (1.2-2.7)	4.2 (2.7-6.2)	2.2 (1.5-3.1)	5.5 (3.5-8.3)
BFA	3.9 (2.4-6.2)	14.2 (9.7-19.9)	1.1 (0.6-1.8)	3.4 (2.2-4.9)	2.1 (1.3-3.3)	7.1 (4.8-10.1)
CIV	14.1 (8.8-21.9)	25.2 (18.7-33.2)	4.2 (2.5-6.9)	6.4 (4.4-9.3)	7.8 (4.7-12.5)	12.9 (9.0-17.9)
GMB	2.3 (0.7-4.7)	8.0 (5.2-11.7)	1.0 (0.3-2.0)	2.9 (1.8-4.4)	1.5 (0.5-2.9)	4.8 (3.1-7.0)
GHA	7.7 (5.1-11.1)	22.9 (17.3-29.4)	3.3 (2.1-4.8)	8.5 (6.0-11.6)	5.1 (3.2-7.6)	14.5 (10.4-19.3)
GIN	23.3 (15.8-33.7)	38.2 (28.4-48.0)	5.5 (3.3-8.8)	8.9 (5.6-12.7)	11.9 (6.9-18.1)	20.2 (13.8-27.8)
LBR	21.9 (13.7-33.3)	40.8 (31.4-50.3)	8.1 (4.7-13.3)	14.7 (10.2-20.6)	13.8 (8.2-22.0)	25.8 (19.0-34.2)
MLI	16.4 (11.4-22.4)	29.3 (21.0-38.6)	3.7 (2.4-5.3)	5.8 (3.7-8.7)	7.8 (5.2-11.1)	13.5 (8.9-19.3)
MRT	9.8 (2.7-23.5)	22.7 (14.6-31.7)	3.0 (0.8-7.7)	6.3 (2.9-11.4)	5.7 (1.7-14.9)	12.4 (7.3-18.7)
NER	2.0 (1.0-3.3)	7.0 (3.8-12.3)	0.6 (0.3-1.0)	1.7 (0.8-3.3)	1.0 (0.5-1.8)	3.4 (1.7-6.5)
NGA	2.4 (1.4-3.6)	10.2 (6.9-14.7)	1.3 (0.7-1.9)	4.3 (2.9-6.5)	1.7 (1.0-2.8)	6.7 (4.3-9.7)
SEN	7.3 (4.6-10.9)	16.1 (12.7-20.0)	0.9 (0.6-1.4)	1.8 (1.3-2.4)	2.7 (1.7-4.1)	5.5 (4.3-6.9)
SLE	10.0 (5.5-16.5)	20.1 (14.1-27.6)	6.5 (3.4-10.8)	11.1 (7.5-16.0)	8.1 (4.4-13.7)	15.0 (10.5-20.3)
TGO	8.5 (3.5-17.2)	20.4 (11.9-32.4)	1.6 (0.6-3.4)	3.5 (1.8-6.4)	3.9 (1.7-8.2)	8.7 (4.7-15.2)
AVG	4.2 (3.2-5.5)	13.8 (11.1-17.0)	1.6 (1.2-2.2)	4.5 (3.6-5.7)	2.6 (2.0-3.5)	8.0 (6.2-10.0)
Central Africa						
AGO	7.9 (2.4-18.8)	8.7 (4.9-14.1)	3.6 (1.0-8.7)	4.1 (2.2-6.9)	5.3 (1.7-12.3)	6.0 (3.2-9.9)
CMR	8.0 (4.6-12.9)	11.2 (7.1-16.5)	3.8 (2.1-6.7)	5.7 (3.5-8.7)	5.6 (3.2-8.7)	8.0 (5.0-12.1)
TCD	5.0 (2.7-8.3)	4.3 (2.1-7.7)	2.2 (1.1-3.9)	2.1 (0.9-3.8)	3.4 (1.9-5.7)	3.0 (1.4-5.5)
COG	15.5 (9.5-24.0)	16.6 (8.3-28.0)	5.3 (2.9-9.0)	6.0 (2.7-11.3)	9.2 (5.2-15.0)	10.0 (4.9-17.4)
COD	6.2 (2.8-10.7)	10.8 (5.4-20.1)	2.7 (1.1-5.0)	5.3 (2.5-10.5)	4.3 (1.9-7.9)	7.6 (3.8-14.1)
GAB	17.8 (7.2-36.4)	13.9 (9.2-19.7)	5.5 (2.0-13.0)	4.2 (2.6-6.3)	10.2 (3.8-22.5)	7.6 (4.6-11.3)
STP	13.3 (6.4-23.9)	16.8 (6.8-31.1)	2.4 (1.0-5.1)	3.2 (1.1-7.1)	5.9 (2.9-10.8)	7.3 (2.6-14.8)
AVG	6.7 (3.8-10.8)	9.6 (6.3-14.4)	3.0 (1.6-4.8)	4.6 (2.8-7.3)	4.6 (2.7-7.3)	6.7 (4.3-10.0)
Eastern Africa						
BDI	5.3 (2.6-10.4)	9.2 (5.3-14.1)	2.1 (1.0-4.2)	3.3 (1.8-5.5)	3.4 (1.7-6.6)	5.6 (3.4-8.6)
COM	5.3 (2.3-10.5)	10.3 (5.4-18.5)	2.4 (1.0-4.9)	4.2 (2.0-8.1)	3.7 (1.5-7.4)	6.6 (3.3-11.7)
ETH	1.4 (0.9-2.0)	3.3 (2.1-5.1)	0.9 (0.5-1.3)	1.9 (1.2-3.0)	1.1 (0.7-1.6)	2.5 (1.5-3.8)
KEN	2.5 (1.6-3.8)	4.1 (2.8-5.7)	1.4 (0.9-2.2)	2.0 (1.4-2.8)	1.9 (1.2-2.9)	2.9 (2.0-4.0)
MDG	1.9 (1.2-3.0)	4.6 (3.1-6.6)	2.2 (1.3-3.5)	4.7 (3.1-6.8)	2.1 (1.3-3.1)	4.7 (3.1-6.8)
MWI	3.5 (2.5-4.8)	6.9 (4.2-10.6)	2.6 (1.9-3.6)	4.6 (2.7-7.4)	3.1 (2.2-4.3)	5.7 (3.5-8.7)
MOZ	4.2 (2.7-6.1)	8.4 (6.2-11.0)	3.3 (2.1-4.9)	5.8 (4.2-7.8)	3.7 (2.4-5.5)	7.1 (5.3-9.5)
RWA	3.2 (2.0-4.7)	11.0 (7.6-15.5)	1.2 (0.8-1.8)	4.0 (2.6-5.8)	2.1 (1.4-3.0)	6.6 (4.5-9.4)
TZA	3.0 (2.1-4.0)	7.6 (5.4-10.5)	2.1 (1.4-2.9)	4.7 (3.2-6.6)	2.5 (1.8-3.4)	6.1 (4.4-8.2)
UGA	10.8 (7.8-14.5)	21.9 (15.7-29.8)	4.2 (2.9-5.8)	8.2 (5.4-11.5)	6.9 (4.8-9.5)	13.6 (9.4-18.9)
ZMB	2.0 (1.3-3.0)	2.7 (1.7-4.1)	3.1 (2.0-4.7)	3.6 (2.3-5.4)	2.4 (1.5-3.6)	3.1 (1.9-4.7)
ZWE	4.9 (2.9-8.2)	7.0 (4.0-10.6)	3.1 (1.9-5.3)	4.1 (2.2-6.3)	4.0 (2.4-6.7)	5.5 (2.8-8.6)
AVG	2.7 (2.3-3.2)	5.8 (4.9-6.9)	1.8 (1.4-2.1)	3.3 (2.7-4.0)	2.2 (1.8-2.7)	4.4 (3.7-5.3)
Southern Africa						
SWZ	5.0 (2.6-9.0)	12.2 (3.8-27.3)	3.8 (1.9-6.8)	11.0 (3.4-24.5)	4.4 (2.2-7.7)	11.2 (3.9-24.4)
LSO	9.1 (5.4-13.7)	16.3 (11.8-21.8)	5.2 (3.0-8.0)	11.2 (8.0-15.4)	7.0 (4.1-10.7)	13.4 (9.6-18.0)
NAM	2.7 (1.6-4.2)	9.8 (4.8-17.6)	1.1 (0.7-1.7)	5.0 (2.4-9.2)	1.8 (1.1-2.7)	6.9 (3.3-13.1)
ZAF	5.5 (1.4-14.9)	11.1 (6.4-18.2)	2.0 (0.5-5.7)	4.9 (2.7-8.6)	3.5 (1.0-9.2)	7.5 (4.2-12.4)
AVG	5.4 (1.6-13.7)	11.2 (6.8-17.7)	2.0 (0.6-5.4)	5.1 (2.9-8.6)	3.5 (1.1-8.6)	7.6 (4.4-12.3)

Results presented as mean with 95% confidence interval. AVG represents sex- and year-matched population weighted mean estimate for sub-region. ISO3 codes defined in Table C3.

Table C10: Estimated national prevalence of genital ulcer in 2000 and 2020

ISO3	Female (%)		Male (%)		Overall (%)	
	2000	2020	2000	2020	2000	2020
Western Africa						
BEN	1.4 (0.9-2.0)	3.3 (2.0-5.0)	0.8 (0.5-1.2)	3.0 (1.8-4.6)	1.0 (0.6-1.5)	3.2 (2.0-4.8)
BFA	1.4 (0.8-2.4)	5.0 (3.2-7.6)	0.4 (0.2-0.7)	2.5 (1.5-3.9)	0.8 (0.4-1.3)	3.6 (2.3-5.4)
CIV	3.9 (2.2-6.5)	7.7 (5.2-11.0)	1.3 (0.7-2.2)	4.0 (2.6-5.9)	2.3 (1.3-4.0)	5.5 (3.6-8.0)
GMB	2.4 (0.9-5.0)	5.6 (3.6-8.2)	0.5 (0.2-1.0)	1.8 (1.1-2.7)	1.1 (0.4-2.4)	3.2 (2.0-4.8)
GHA	3.4 (2.0-5.1)	9.3 (6.4-12.8)	1.3 (0.8-2.1)	5.8 (3.9-8.2)	2.1 (1.3-3.3)	7.4 (5.1-10.5)
GIN	5.3 (2.9-8.7)	10.4 (6.7-15.4)	1.5 (0.8-2.6)	4.9 (3.0-7.6)	2.9 (1.6-4.8)	7.2 (4.5-10.6)
LBR	22.7 (13.3-35.6)	31.8 (23.1-41.5)	4.8 (2.5-8.6)	11.2 (7.1-15.8)	11.0 (5.9-18.2)	19.5 (13.4-27.0)
MLI	8.3 (5.6-12.1)	13.2 (8.5-18.7)	1.1 (0.7-1.8)	2.9 (1.7-4.3)	3.1 (2.0-4.6)	6.3 (3.9-9.3)
MRT	6.2 (1.8-14.2)	11.9 (7.1-18.4)	1.4 (0.4-3.8)	4.4 (1.9-8.4)	2.9 (0.9-7.3)	7.4 (4.3-12.0)
NER	1.7 (0.8-2.9)	3.7 (1.8-7.1)	0.5 (0.2-0.8)	1.7 (0.8-3.2)	0.9 (0.5-1.5)	2.5 (1.2-4.2)
NGA	2.1 (1.2-3.3)	5.4 (3.4-8.3)	0.6 (0.4-1.0)	2.5 (1.5-4.0)	1.2 (0.7-1.8)	3.7 (2.3-5.4)
SEN	4.1 (2.4-6.7)	6.6 (4.9-8.6)	0.4 (0.2-0.7)	1.0 (0.7-1.4)	1.3 (0.7-2.1)	2.6 (1.9-3.4)
SLE	8.4 (4.4-14.7)	13.1 (8.9-18.2)	2.6 (1.3-4.8)	6.5 (4.2-9.5)	4.7 (2.4-8.4)	9.2 (6.0-13.2)
TGO	7.6 (3.3-15.4)	14.6 (7.6-24.3)	1.1 (0.4-2.4)	3.5 (1.6-6.6)	3.0 (1.2-6.6)	7.3 (3.9-12.7)
AVG	2.7 (2.0-3.6)	6.4 (4.9-8.3)	0.8 (0.5-1.0)	2.8 (2.1-3.7)	1.4 (1.1-1.9)	4.2 (3.3-5.3)
Central Africa						
AGO	4.1 (1.2-10.3)	3.9 (2.0-7.1)	2.8 (0.8-7.5)	3.1 (1.5-5.8)	3.4 (1.0-8.5)	3.5 (1.8-6.3)
CMR	2.9 (1.6-5.1)	3.4 (2.0-5.4)	2.6 (1.3-4.7)	3.4 (1.9-5.5)	2.7 (1.4-4.8)	3.4 (2.0-5.3)
TCD	1.2 (0.6-2.2)	1.2 (0.5-2.3)	1.1 (0.5-1.9)	1.1 (0.5-2.4)	1.1 (0.6-2.0)	1.2 (0.6-2.3)
COG	8.5 (4.6-14.0)	9.1 (4.3-17.5)	7.7 (4.2-13.0)	9.2 (4.3-17.8)	8.2 (4.6-13.8)	9.3 (3.9-17.9)
COD	4.8 (2.2-8.5)	6.5 (3.2-12.2)	2.9 (1.3-5.5)	4.5 (2.1-8.5)	3.7 (1.7-6.8)	5.5 (2.6-10.4)
GAB	6.2 (2.1-14.2)	4.6 (2.7-7.4)	3.5 (1.2-8.8)	2.8 (1.6-4.7)	4.5 (1.6-10.7)	3.5 (2.1-5.6)
STP	6.2 (2.5-12.6)	6.9 (2.4-15.3)	2.5 (1.0-5.1)	3.1 (1.0-7.0)	3.8 (1.7-7.5)	4.5 (1.6-10.1)
AVG	3.8 (2.2-6.2)	4.5 (2.7-7.1)	2.6 (1.4-4.3)	3.5 (2.1-5.5)	3.1 (1.8-5.2)	4.0 (2.4-6.3)
Eastern Africa						
BDI	3.8 (1.8-7.5)	6.8 (4.0-10.7)	1.7 (0.8-3.3)	3.3 (1.9-5.5)	2.6 (1.2-5.3)	4.8 (2.8-7.7)
COM	3.4 (1.3-7.0)	6.4 (3.0-11.5)	1.8 (0.6-3.8)	3.7 (1.6-6.9)	2.6 (1.0-5.3)	4.9 (2.4-9.0)
ETH	0.7 (0.4-1.1)	1.9 (1.1-3.0)	0.3 (0.2-0.5)	0.9 (0.5-1.5)	0.5 (0.3-0.7)	1.3 (0.7-2.2)
KEN	1.6 (0.9-2.5)	2.3 (1.5-3.3)	1.3 (0.7-2.0)	2.0 (1.3-2.8)	1.4 (0.9-2.2)	2.2 (1.4-3.1)
MDG	0.7 (0.4-1.1)	1.4 (0.9-2.2)	1.0 (0.6-1.6)	2.2 (1.4-3.3)	0.9 (0.5-1.3)	1.8 (1.1-2.7)
MWI	6.5 (4.4-9.4)	11.2 (6.9-16.7)	3.5 (2.3-5.1)	6.7 (3.9-10.3)	4.8 (3.3-6.7)	8.7 (4.8-13.4)
MOZ	1.7 (1.1-2.6)	3.4 (2.3-4.7)	1.9 (1.1-2.8)	4.0 (2.8-5.6)	1.8 (1.1-2.7)	3.7 (2.6-5.2)
RWA	2.2 (1.4-3.4)	8.0 (5.1-12.0)	1.3 (0.8-2.0)	5.4 (3.3-8.1)	1.7 (1.1-2.6)	6.7 (4.2-9.9)
TZA	1.8 (1.2-2.5)	4.2 (2.7-5.9)	2.0 (1.3-2.9)	4.9 (3.3-6.9)	1.9 (1.3-2.8)	4.5 (3.1-6.4)
UGA	11.5 (8.1-15.8)	19.6 (13.1-28.1)	6.4 (4.3-9.0)	12.3 (7.9-18.1)	8.7 (6.0-12.1)	15.6 (10.2-21.5)
ZMB	2.7 (1.6-4.2)	3.2 (1.9-5.0)	3.5 (2.2-5.6)	4.4 (2.6-6.8)	3.1 (1.8-4.9)	3.8 (2.3-5.9)
ZWE	3.7 (2.1-6.3)	5.6 (3.1-8.9)	3.4 (1.9-5.9)	5.7 (3.2-9.0)	3.6 (2.0-6.1)	5.6 (3.2-9.0)
AVG	1.8 (1.4-2.1)	3.6 (2.9-4.4)	1.2 (1.0-1.5)	2.7 (2.2-3.3)	1.5 (1.2-1.8)	3.1 (2.5-3.8)
Southern Africa						
SWZ	5.4 (2.6-10.3)	11.1 (3.5-25.1)	4.8 (2.3-9.2)	12.0 (3.9-26.1)	5.1 (2.4-9.4)	11.8 (3.7-26.5)
LSO	4.0 (2.2-6.5)	6.5 (4.3-9.3)	4.0 (2.2-6.6)	7.9 (5.2-11.2)	4.0 (2.2-6.4)	7.2 (4.9-10.1)
NAM	1.9 (1.1-3.2)	5.6 (2.4-10.5)	1.2 (0.7-1.9)	4.3 (1.8-8.2)	1.5 (0.8-2.5)	5.0 (2.1-9.9)
ZAF	3.2 (0.8-8.4)	5.5 (2.9-9.2)	1.5 (0.4-4.0)	3.1 (1.6-5.4)	2.2 (0.6-5.7)	4.1 (2.2-7.1)
AVG	3.2 (0.9-7.7)	5.6 (3.1-9.1)	1.5 (0.4-3.9)	3.3 (1.8-5.6)	2.2 (0.6-5.4)	4.3 (2.3-7.0)

Results presented as mean with 95% confidence interval. AVG represents sex- and year-matched population weighted mean estimate for sub-region. ISO3 codes defined in Table C3.

Table C11: Estimated national proportion treated for genital symptoms in 2000 and 2020

ISO3	Female (%)		Male (%)		Overall (%)	
	2000	2020	2000	2020	2000	2020
Western Africa						
BEN	64.1 (52.7-74.3)	44.6 (33.1-57.1)	76.9 (67.0-84.7)	41.2 (29.6-54.2)	70.5 (61.4-79.1)	43.4 (31.6-56.6)
BFA	58.0 (44.8-69.7)	52.5 (30.7-72.5)	59.5 (44.2-72.7)	36.2 (17.1-56.7)	58.4 (45.2-70.8)	44.1 (23.9-65.3)
CIV	47.2 (32.3-62.1)	56.8 (38.9-75.2)	69.3 (52.0-81.8)	61.2 (42.7-78.3)	59.0 (43.3-73.8)	58.8 (40.2-76.5)
GMB	65.1 (41.2-83.8)	65.4 (54.9-74.6)	71.7 (48.6-87.9)	54.5 (41.9-66.3)	68.1 (44.7-87.0)	60.0 (48.2-70.4)
GHA	53.2 (39.8-66.3)	71.3 (58.4-83.1)	67.3 (53.9-78.9)	68.0 (52.8-81.2)	60.2 (46.7-72.7)	69.8 (54.3-82.2)
GIN	62.9 (49.0-75.0)	66.9 (57.2-76.3)	75.4 (62.7-84.8)	62.7 (51.1-73.1)	69.2 (57.7-79.6)	65.2 (54.4-74.6)
LBR	77.7 (64.6-88.0)	88.0 (82.6-92.0)	78.3 (65.0-88.5)	78.0 (68.8-84.6)	78.3 (64.6-87.9)	83.6 (76.8-89.0)
MLI	51.2 (41.6-61.3)	57.0 (46.2-67.4)	59.2 (48.4-69.3)	46.1 (34.5-57.9)	55.4 (45.5-64.7)	51.5 (40.7-62.2)
MRT	40.9 (14.5-74.7)	39.8 (28.5-51.0)	56.9 (25.6-86.7)	38.3 (24.3-54.5)	49.1 (18.3-80.8)	39.3 (28.1-51.4)
NER	32.0 (18.2-47.5)	44.8 (26.9-65.9)	41.3 (24.9-58.7)	36.1 (19.0-58.4)	36.8 (21.5-53.5)	40.3 (23.6-59.0)
NGA	64.3 (52.0-75.0)	59.2 (48.6-69.6)	77.0 (66.1-85.2)	55.7 (44.4-67.5)	70.8 (60.6-79.9)	57.4 (47.0-67.1)
SEN	62.1 (49.0-74.8)	48.5 (39.7-57.5)	69.8 (56.2-82.0)	38.5 (29.1-49.4)	65.3 (51.6-77.5)	43.6 (34.8-53.4)
SLE	48.7 (30.5-67.2)	75.3 (65.7-83.1)	75.3 (60.1-87.5)	82.3 (74.0-88.5)	63.1 (45.4-79.5)	79.1 (70.5-85.7)
TGO	50.7 (24.9-76.2)	56.0 (38.5-73.3)	67.5 (40.5-87.5)	55.6 (36.8-74.3)	59.7 (32.4-82.1)	55.6 (37.7-72.8)
AVG	59.1 (51.6-65.8)	59.0 (52.7-65.5)	72.3 (65.0-78.4)	54.5 (47.5-61.5)	65.8 (59.1-72.5)	56.8 (50.6-62.9)
Central Africa						
AGO	66.5 (35.8-89.2)	61.2 (43.9-75.9)	86.0 (64.7-96.7)	65.3 (48.9-79.0)	78.4 (52.5-93.9)	63.1 (46.9-76.8)
CMR	72.1 (60.2-82.2)	58.8 (47.8-69.7)	86.3 (77.0-92.5)	56.0 (44.1-68.4)	80.3 (69.8-87.9)	57.1 (45.7-68.2)
TCD	65.8 (49.3-79.4)	37.0 (21.9-56.5)	88.2 (78.0-94.4)	45.4 (27.5-64.6)	79.4 (66.3-88.9)	40.7 (24.2-58.4)
COG	72.1 (57.7-83.6)	72.9 (53.7-87.1)	87.4 (78.2-93.7)	72.6 (52.9-87.6)	81.0 (68.1-89.8)	72.5 (53.9-88.2)
COD	56.9 (35.2-75.0)	56.3 (37.9-74.6)	76.6 (57.4-88.1)	54.0 (34.5-72.7)	68.2 (49.4-83.4)	54.4 (35.6-73.4)
GAB	69.7 (46.5-87.5)	66.0 (55.1-75.4)	85.4 (68.4-95.4)	65.7 (53.9-75.9)	78.5 (57.4-91.9)	65.6 (54.2-76.0)
STP	85.9 (70.3-94.4)	80.4 (57.0-94.2)	89.7 (76.8-96.7)	70.1 (44.0-89.6)	88.1 (76.5-95.1)	75.7 (53.0-91.3)
AVG	63.1 (49.1-75.6)	56.7 (44.9-68.5)	82.4 (71.3-89.7)	56.6 (44.1-68.4)	74.4 (62.3-84.7)	56.1 (43.8-68.2)
Eastern Africa						
BDI	47.3 (26.0-68.7)	50.8 (36.3-65.0)	47.2 (25.8-69.0)	46.9 (32.1-61.5)	47.0 (26.2-66.7)	48.9 (35.7-62.9)
COM	53.5 (27.9-76.9)	51.9 (32.7-71.9)	67.1 (42.5-85.3)	62.3 (42.5-79.5)	60.2 (34.6-80.4)	57.2 (37.7-75.4)
ETH	35.9 (22.4-51.2)	31.0 (19.9-44.7)	41.5 (26.6-58.4)	32.1 (20.5-46.7)	38.7 (24.4-54.4)	31.2 (20.5-43.7)
KEN	67.1 (55.9-77.6)	67.0 (52.7-79.5)	77.5 (67.0-86.3)	74.3 (61.2-85.0)	72.2 (60.0-82.4)	71.2 (56.6-83.0)
MDG	64.9 (52.5-75.5)	58.5 (47.6-68.3)	79.0 (69.6-86.9)	70.5 (60.5-79.2)	72.2 (61.3-81.4)	64.9 (55.7-73.5)
MWI	58.7 (49.7-67.4)	49.9 (36.4-63.6)	55.3 (45.7-64.7)	42.0 (29.4-55.8)	56.7 (47.2-65.4)	46.7 (34.0-58.8)
MOZ	72.1 (61.8-81.2)	74.7 (63.7-83.9)	80.9 (71.6-88.0)	80.0 (69.7-88.0)	76.4 (66.1-84.3)	77.4 (67.3-86.1)
RWA	44.5 (35.3-54.3)	61.7 (51.3-69.9)	42.4 (32.4-53.5)	56.1 (45.8-65.6)	43.4 (34.2-53.8)	59.1 (49.8-67.9)
TZA	72.6 (63.4-81.1)	55.9 (37.7-71.5)	81.3 (72.5-88.4)	63.4 (44.0-79.5)	77.2 (68.3-84.9)	59.4 (41.5-75.2)
UGA	62.9 (53.5-71.7)	70.5 (61.4-79.1)	64.6 (54.8-73.6)	68.0 (57.7-77.6)	64.0 (55.2-72.7)	68.9 (59.5-77.5)
ZMB	63.9 (52.9-73.3)	65.7 (55.5-74.7)	75.4 (65.9-83.2)	74.0 (65.2-81.7)	69.7 (59.7-78.2)	70.3 (60.2-78.7)
ZWE	63.2 (49.1-76.8)	42.6 (28.0-57.3)	65.1 (50.4-78.2)	40.7 (26.5-55.4)	63.7 (48.9-78.0)	41.9 (27.4-57.8)
AVG	57.7 (52.0-63.4)	53.1 (46.5-58.9)	65.3 (59.2-71.3)	56.6 (49.6-62.9)	61.4 (55.6-66.9)	54.8 (48.5-61.4)
Southern Africa						
SWZ	77.0 (61.4-88.7)	72.0 (37.7-92.3)	81.4 (67.3-91.4)	66.5 (33.7-89.5)	78.8 (63.0-89.5)	70.6 (39.8-91.9)
LSO	67.9 (53.2-81.0)	74.2 (58.7-86.7)	67.1 (51.5-81.2)	61.2 (42.4-78.2)	67.7 (52.7-79.8)	68.4 (50.6-81.6)
NAM	71.0 (55.2-83.8)	63.3 (43.1-80.1)	78.2 (65.8-87.2)	58.5 (35.4-77.9)	74.3 (62.3-84.3)	61.3 (41.8-77.6)
ZAF	72.5 (39.2-94.6)	72.0 (57.1-83.1)	74.8 (40.6-94.4)	62.7 (46.5-77.4)	72.5 (34.8-94.1)	68.3 (53.3-81.0)
AVG	72.6 (41.4-93.9)	71.9 (58.3-82.5)	75.0 (43.5-93.9)	62.6 (47.2-76.8)	72.6 (37.4-93.2)	68.2 (53.9-80.2)

Results presented as mean with 95% confidence interval. AVG represents sex- and year-matched population weighted mean estimate for sub-region. ISO3 codes defined in Table C3.

Table C12: Estimated national proportion utilising public sector in 2000 and 2020

ISO3	Female (%)		Male (%)		Overall (%)	
	2000	2020	2000	2020	2000	2020
Western Africa						
BEN	57.3 (53.2-61.0)	60.4 (56.9-63.8)	36.8 (29.7-44.7)	43.6 (37.5-50.0)	46.6 (41.9-51.4)	52.3 (48.3-56.2)
BFA	87.0 (82.7-90.3)	90.2 (86.8-92.7)	75.8 (65.0-84.8)	80.9 (72.2-87.9)	81.7 (75.9-86.6)	85.9 (80.9-89.9)
CIV	48.9 (44.9-53.1)	54.4 (50.4-58.6)	34.7 (26.9-43.9)	42.0 (34.8-50.0)	41.4 (36.3-47.0)	48.2 (43.9-52.7)
GMB	61.5 (55.6-67.5)	73.7 (70.1-76.9)	51.3 (39.7-62.9)	62.3 (52.4-70.6)	56.5 (49.5-63.2)	68.1 (63.0-72.9)
GHA	47.9 (43.9-51.9)	38.2 (34.8-41.6)	34.6 (28.2-42.0)	30.5 (24.4-36.8)	40.9 (36.6-45.4)	34.4 (30.9-38.2)
GIN	67.3 (63.1-71.1)	72.0 (69.5-74.3)	60.5 (51.8-68.5)	64.5 (58.7-70.0)	63.8 (58.9-68.6)	68.3 (65.2-71.2)
LBR	48.3 (43.9-52.6)	51.0 (48.0-54.1)	36.5 (29.9-43.6)	37.1 (32.1-42.0)	42.4 (38.4-46.7)	44.1 (40.8-47.2)
MLI	56.9 (53.1-60.6)	63.1 (60.0-66.1)	47.2 (38.1-56.3)	56.5 (48.6-64.2)	51.7 (46.1-57.0)	59.8 (55.5-64.0)
MRT	85.5 (81.1-88.9)	91.3 (88.9-93.2)	72.2 (57.2-84.2)	80.2 (68.6-88.7)	79.5 (72.1-85.6)	86.8 (82.4-90.5)
NER	66.2 (59.7-72.1)	71.9 (66.5-76.8)	48.3 (35.9-61.1)	57.3 (45.2-68.7)	57.0 (48.7-64.9)	65.0 (57.5-71.2)
NGA	40.4 (36.4-44.1)	40.1 (37.9-42.2)	31.7 (25.0-38.5)	32.1 (27.9-36.5)	35.9 (31.9-40.0)	36.1 (33.7-38.6)
SEN	87.7 (85.3-89.8)	89.9 (88.3-91.4)	70.2 (60.9-78.8)	76.0 (69.2-82.1)	79.1 (74.1-83.6)	83.3 (80.0-86.3)
SLE	69.3 (65.8-72.7)	67.8 (65.5-70.0)	59.8 (52.6-67.0)	54.8 (50.8-58.6)	64.6 (60.5-68.8)	61.2 (58.7-63.5)
TGO	57.4 (52.0-62.5)	65.6 (61.4-69.7)	43.0 (32.2-54.5)	51.9 (42.6-62.1)	50.0 (43.4-56.6)	58.9 (53.5-64.7)
AVG	50.9 (47.3-54.0)	51.7 (49.9-53.5)	39.6 (33.1-46.0)	41.9 (38.0-45.7)	45.1 (41.5-49.0)	46.9 (44.6-49.1)
Central Africa						
AGO	85.7 (78.5-90.7)	70.6 (63.9-76.7)	72.1 (56.9-83.6)	70.7 (61.2-78.8)	78.7 (69.8-85.7)	70.7 (64.5-76.3)
CMR	76.3 (68.1-83.0)	67.0 (63.2-70.8)	59.4 (45.9-71.8)	60.4 (52.8-67.5)	67.2 (58.2-75.1)	63.8 (59.5-67.7)
TCD	59.5 (44.2-72.6)	62.1 (54.2-69.9)	33.0 (18.5-51.6)	46.6 (33.9-59.9)	44.9 (32.8-57.8)	55.0 (47.1-63.1)
COG	67.8 (61.4-73.5)	58.9 (51.0-66.0)	38.6 (28.4-50.2)	39.7 (28.9-50.7)	51.9 (45.0-59.1)	49.4 (42.2-56.5)
COD	65.5 (56.7-73.4)	49.2 (42.5-55.6)	45.7 (33.7-58.4)	49.5 (39.5-59.5)	54.4 (46.0-63.3)	49.3 (43.0-55.9)
GAB	58.1 (43.9-69.8)	53.6 (48.4-59.0)	38.5 (23.9-55.9)	41.9 (34.7-49.2)	47.2 (35.1-59.5)	47.6 (42.8-52.7)
STP	92.4 (87.9-95.7)	83.2 (72.9-90.6)	74.0 (60.1-85.0)	72.9 (56.9-84.5)	83.9 (76.4-89.9)	78.8 (67.9-86.6)
AVG	70.1 (61.2-76.8)	57.2 (52.0-62.3)	50.3 (37.9-62.9)	54.5 (46.6-62.4)	59.3 (50.7-67.6)	55.9 (50.9-60.9)
Eastern Africa						
BDI	78.5 (74.0-82.4)	79.6 (76.4-82.5)	55.2 (45.8-65.0)	59.5 (51.2-67.1)	67.0 (60.7-72.8)	69.6 (64.6-74.3)
COM	59.2 (50.3-68.2)	63.0 (54.5-71.5)	51.4 (37.8-65.6)	57.6 (45.0-70.1)	55.4 (45.7-65.2)	60.2 (51.4-69.4)
ETH	71.1 (62.1-78.7)	74.5 (67.1-80.8)	66.4 (55.1-76.4)	74.5 (66.2-81.3)	68.7 (61.0-75.6)	74.6 (68.1-79.7)
KEN	68.8 (63.3-73.6)	72.7 (68.4-76.9)	46.7 (37.1-56.5)	53.3 (44.2-62.0)	57.6 (51.4-63.3)	63.0 (57.4-68.2)
MDG	66.6 (60.0-72.0)	72.6 (68.4-76.3)	52.8 (43.9-61.2)	65.0 (59.8-69.6)	59.4 (53.8-65.2)	68.8 (65.2-72.1)
MWI	60.1 (53.1-66.4)	75.9 (72.6-79.1)	58.2 (49.3-66.4)	73.4 (67.1-78.7)	59.2 (53.5-64.7)	74.7 (71.3-77.8)
MOZ	80.6 (74.7-85.2)	88.1 (85.1-90.8)	63.3 (54.3-71.5)	81.4 (77.2-85.0)	71.9 (65.7-77.1)	84.9 (82.1-87.5)
RWA	83.4 (79.4-86.8)	83.7 (81.6-85.6)	63.8 (54.6-71.9)	64.8 (59.6-70.1)	74.3 (68.9-78.8)	74.3 (71.4-77.2)
TZA	70.8 (65.3-75.5)	69.3 (62.8-75.2)	54.5 (43.4-65.8)	56.0 (43.4-68.1)	62.4 (55.7-69.0)	62.4 (54.5-69.9)
UGA	51.5 (47.5-55.5)	55.2 (52.3-57.8)	36.8 (30.3-43.5)	41.1 (35.9-46.7)	44.1 (39.7-48.1)	48.0 (44.9-51.3)
ZMB	84.5 (80.4-88.0)	89.6 (87.2-91.6)	61.6 (54.0-68.8)	78.7 (75.2-82.0)	73.0 (68.0-77.1)	84.3 (82.2-86.3)
ZWE	80.2 (76.6-83.4)	85.1 (82.5-87.5)	60.5 (54.6-66.4)	73.1 (68.1-78.1)	70.9 (67.3-74.3)	79.6 (76.7-82.4)
AVG	70.4 (66.5-74.1)	73.9 (71.1-76.6)	56.5 (49.6-62.9)	64.4 (59.8-68.7)	63.4 (59.4-67.3)	69.1 (66.2-71.8)
Southern Africa						
SWZ	88.9 (83.1-93.1)	86.5 (72.7-94.0)	69.4 (56.6-79.9)	82.1 (65.9-91.7)	79.4 (71.4-85.0)	84.6 (73.6-91.9)
LSO	70.5 (57.7-81.1)	72.2 (59.8-81.2)	53.2 (34.2-70.8)	63.5 (47.4-76.4)	61.3 (47.4-73.4)	67.9 (58.0-76.3)
NAM	79.4 (68.7-87.6)	81.9 (72.6-89.1)	72.0 (52.8-85.4)	86.0 (75.8-92.4)	75.5 (62.5-84.4)	84.0 (76.4-89.4)
ZAF	75.6 (57.4-88.3)	78.0 (70.5-83.8)	59.1 (33.9-80.4)	78.1 (67.7-86.5)	67.0 (48.1-81.6)	78.0 (71.8-83.3)
AVG	75.8 (58.1-88.0)	78.1 (70.6-83.9)	59.4 (34.9-79.9)	78.0 (68.2-86.3)	67.3 (49.3-81.2)	78.0 (72.0-83.2)

Proportion accessing sector for treatment, among those reporting treatment for genital symptoms. Results presented as mean with 95% confidence interval. AVG represents sex- and year-matched population weighted mean estimate for sub-region. ISO3 codes defined in Table C3.

Table C13: Estimated national proportion utilising private sector in 2000 and 2020

ISO3	Female (%)		Male (%)		Overall (%)	
	2000	2020	2000	2020	2000	2020
Western Africa						
BEN	20.5 (17.0-24.2)	10.7 (9.0-12.7)	14.4 (9.2-20.5)	10.5 (7.1-14.3)	17.3 (14.0-21.2)	10.6 (8.5-12.8)
BFA	5.6 (3.5-8.6)	2.9 (1.7-4.6)	8.6 (4.0-15.2)	5.7 (2.7-9.9)	7.1 (4.1-10.9)	4.2 (2.4-6.5)
CIV	9.2 (7.1-11.6)	5.1 (3.9-6.4)	14.5 (9.3-21.5)	10.8 (7.2-16.0)	12.0 (8.9-15.8)	8.0 (5.9-10.6)
GMB	18.3 (13.8-23.4)	10.8 (8.5-13.3)	18.2 (10.8-27.7)	13.6 (8.7-20.1)	18.2 (13.6-24.2)	12.1 (9.1-15.8)
GHA	16.7 (13.4-20.4)	6.6 (5.3-8.0)	13.5 (9.1-18.9)	7.3 (4.8-10.4)	15.1 (12.1-18.7)	6.9 (5.5-8.6)
GIN	11.1 (8.6-13.9)	5.9 (4.8-7.1)	12.5 (7.9-18.4)	8.2 (5.5-11.2)	11.8 (9.2-15.1)	7.0 (5.5-8.8)
LBR	28.9 (24.5-33.7)	15.1 (13.1-17.1)	37.3 (29.7-45.1)	23.4 (19.1-28.5)	33.1 (28.5-37.8)	19.2 (16.7-22.1)
MLI	7.0 (5.5-8.7)	3.8 (3.0-4.7)	9.0 (5.7-13.7)	6.6 (4.2-9.5)	8.0 (6.0-10.6)	5.2 (3.9-6.8)
MRT	8.8 (5.8-12.8)	4.6 (3.3-6.3)	13.6 (6.2-25.1)	9.3 (4.4-17.3)	10.9 (6.6-17.2)	6.5 (4.1-10.0)
NER	5.1 (3.0-8.1)	2.7 (1.6-4.2)	7.7 (4.1-13.4)	5.6 (2.9-9.6)	6.4 (3.9-9.9)	4.1 (2.5-6.3)
NGA	27.2 (23.0-31.7)	13.3 (12.0-14.8)	24.0 (18.2-30.7)	15.0 (11.8-18.5)	25.6 (21.6-29.9)	14.2 (12.4-16.0)
SEN	7.2 (5.4-9.4)	3.6 (2.9-4.5)	13.5 (7.9-20.7)	8.9 (5.8-12.8)	10.3 (7.3-14.0)	6.2 (4.6-8.1)
SLE	8.7 (6.9-11.0)	4.2 (3.4-5.1)	8.8 (6.1-12.7)	4.9 (3.8-6.4)	8.8 (6.9-11.0)	4.6 (3.9-5.4)
TGO	20.9 (16.1-26.6)	11.8 (9.2-14.7)	23.4 (15.0-33.7)	17.4 (11.6-24.7)	22.2 (16.9-28.6)	14.6 (11.0-18.9)
AVG	19.7 (16.7-22.9)	9.6 (8.7-10.6)	18.8 (14.3-24.0)	12.0 (9.8-14.5)	19.3 (16.3-22.5)	10.8 (9.6-12.2)
Central Africa						
AGO	6.8 (3.6-11.8)	21.8 (15.7-28.7)	13.2 (5.6-25.4)	18.7 (11.3-28.3)	10.1 (5.4-17.0)	20.3 (15.4-26.2)
CMR	3.1 (1.7-5.1)	10.6 (8.3-13.5)	8.0 (3.7-14.9)	11.8 (7.5-17.5)	5.7 (3.1-9.6)	11.2 (8.6-14.6)
TCD	2.1 (0.9-4.1)	8.4 (4.5-14.6)	4.5 (1.5-9.9)	9.3 (4.3-17.5)	3.3 (1.4-6.7)	8.8 (4.8-14.3)
COG	3.7 (2.6-5.0)	12.9 (8.3-18.7)	6.4 (3.4-10.9)	10.0 (5.0-17.4)	5.1 (3.4-7.8)	11.5 (7.5-16.6)
COD	11.6 (7.5-16.9)	34.4 (26.9-41.9)	15.9 (8.3-26.1)	25.6 (16.1-37.6)	14.0 (9.2-20.5)	30.5 (24.0-37.8)
GAB	4.7 (2.5-8.0)	16.8 (12.8-21.7)	7.6 (2.9-16.0)	11.9 (7.3-18.2)	6.2 (3.1-11.3)	14.3 (10.9-18.4)
STP	3.7 (1.7-7.2)	13.4 (6.2-23.9)	11.8 (4.5-23.7)	17.1 (7.7-32.0)	7.4 (3.4-14.1)	15.0 (7.8-25.3)
AVG	8.0 (5.0-12.2)	25.5 (20.2-30.9)	12.6 (6.5-21.4)	20.0 (13.4-28.5)	10.5 (6.6-16.0)	23.0 (18.3-28.1)
Eastern Africa						
BDI	11.0 (8.4-14.0)	10.3 (8.3-12.7)	12.8 (8.2-19.5)	15.5 (10.7-21.7)	11.9 (8.9-15.8)	12.9 (10.1-16.4)
COM	20.7 (13.7-29.2)	20.5 (13.4-28.4)	14.5 (8.5-22.6)	18.4 (11.2-28.5)	17.6 (12.1-24.4)	19.4 (13.0-26.9)
ETH	21.2 (14.4-29.4)	20.7 (14.9-28.0)	14.9 (8.7-23.0)	18.9 (12.3-26.9)	18.0 (12.7-24.2)	19.7 (14.7-26.0)
KEN	17.1 (13.2-21.3)	16.8 (13.4-20.6)	22.5 (15.3-31.4)	29.0 (20.6-37.6)	19.8 (15.5-25.2)	22.9 (18.1-28.0)
MDG	17.5 (13.3-22.5)	17.7 (14.4-21.5)	10.2 (6.7-15.1)	14.0 (10.9-17.5)	13.7 (10.5-17.4)	15.9 (13.3-18.5)
MWI	3.2 (2.3-4.2)	3.7 (2.9-4.8)	6.8 (4.1-10.5)	9.6 (6.4-14.0)	4.9 (3.5-6.7)	6.6 (4.8-9.0)
MOZ	2.4 (1.5-3.8)	2.5 (1.6-3.7)	3.7 (2.2-5.8)	5.4 (3.6-7.9)	3.1 (2.0-4.4)	3.9 (2.8-5.4)
RWA	2.5 (1.8-3.4)	2.3 (1.8-3.0)	3.7 (2.2-5.9)	4.3 (2.9-6.0)	3.1 (2.2-4.3)	3.3 (2.5-4.3)
TZA	2.7 (1.6-4.1)	2.4 (1.5-3.8)	3.3 (1.7-5.6)	3.8 (2.0-6.6)	3.0 (1.8-4.4)	3.1 (2.0-4.8)
UGA	33.4 (29.2-37.8)	33.3 (30.5-36.5)	35.0 (27.6-42.6)	44.2 (38.5-50.2)	34.2 (30.0-38.4)	38.9 (35.5-42.4)
ZMB	4.7 (3.3-6.4)	4.6 (3.3-6.3)	5.9 (4.0-8.5)	8.6 (6.3-11.1)	5.3 (4.0-6.9)	6.6 (5.1-8.0)
ZWE	8.7 (6.7-11.0)	8.6 (6.8-10.9)	9.2 (6.6-12.3)	12.6 (9.1-16.6)	8.9 (7.3-10.8)	10.4 (8.3-12.7)
AVG	14.1 (11.3-17.0)	14.3 (12.3-16.7)	13.4 (9.9-17.9)	17.6 (14.3-21.3)	13.7 (11.3-16.4)	16.0 (13.9-18.3)
Southern Africa						
SWZ	8.4 (4.6-13.4)	4.6 (1.7-10.1)	17.3 (8.7-29.2)	6.8 (1.8-16.6)	12.7 (7.8-19.1)	5.5 (2.1-11.1)
LSO	13.7 (6.8-23.1)	7.3 (4.0-12.2)	17.6 (6.6-35.8)	6.2 (2.6-12.6)	15.8 (8.0-27.3)	6.7 (3.9-10.7)
NAM	17.0 (9.4-27.5)	9.3 (4.8-16.6)	18.0 (6.7-37.7)	6.4 (2.5-13.8)	17.6 (9.7-30.3)	7.9 (4.3-13.5)
ZAF	19.6 (7.6-37.9)	10.0 (6.8-14.4)	24.8 (7.9-51.6)	9.4 (4.1-17.8)	22.4 (9.5-40.6)	9.7 (6.4-14.3)
AVG	19.1 (7.6-36.6)	9.8 (6.7-14.3)	24.2 (7.8-49.8)	9.1 (4.0-17.3)	21.8 (9.6-39.4)	9.5 (6.2-13.8)

Proportion accessing sector for treatment, among those reporting treatment for genital symptoms. Results presented as mean with 95% confidence interval. AVG represents sex- and year-matched population weighted mean estimate for sub-region. ISO3 codes defined in Table C3.

Table C14: Estimated national proportion utilising pharmacy or shop in 2000 and 2020

ISO3	Female (%)		Male (%)		Overall (%)	
	2000	2020	2000	2020	2000	2020
Western Africa						
BEN	4.4 (3.4-5.6)	16.6 (13.6-19.8)	4.8 (3.2-7.0)	15.8 (11.4-21.1)	4.6 (3.6-5.8)	16.2 (13.3-19.2)
BFA	0.5 (0.2-1.0)	2.0 (0.9-3.9)	1.6 (0.7-3.4)	4.8 (2.0-9.5)	1.1 (0.5-2.0)	3.3 (1.5-6.2)
CIV	2.8 (2.0-3.6)	11.1 (8.4-14.0)	4.4 (2.5-7.0)	14.8 (9.8-21.1)	3.6 (2.5-5.1)	12.9 (9.7-16.8)
GMB	0.4 (0.2-0.7)	1.5 (0.7-2.9)	1.1 (0.5-2.1)	3.7 (1.8-6.8)	0.7 (0.3-1.3)	2.6 (1.3-4.6)
GHA	15.6 (13.0-18.3)	44.7 (40.9-48.6)	18.5 (13.5-24.2)	45.2 (37.4-52.7)	17.1 (14.1-20.3)	45.0 (40.6-49.5)
GIN	2.0 (1.6-2.6)	7.9 (6.6-9.3)	4.6 (3.0-6.7)	13.7 (10.1-17.7)	3.3 (2.5-4.4)	10.7 (8.8-13.0)
LBR	6.7 (5.4-8.1)	25.2 (22.7-27.9)	10.8 (7.8-14.4)	30.4 (25.4-35.3)	8.7 (7.1-10.7)	27.8 (25.1-30.7)
MLI	1.7 (1.2-2.1)	6.6 (5.3-8.0)	2.5 (1.5-4.1)	8.4 (5.3-12.6)	2.1 (1.5-3.0)	7.5 (5.7-9.7)
MRT	0.1 (0.0-0.3)	0.4 (0.1-1.0)	0.6 (0.2-1.4)	1.8 (0.5-4.1)	0.3 (0.1-0.7)	1.0 (0.3-2.2)
NER	1.2 (0.5-2.2)	4.5 (2.1-8.5)	2.6 (1.1-5.1)	8.6 (4.1-15.6)	1.9 (0.9-3.5)	6.4 (3.3-11.5)
NGA	8.5 (7.0-10.1)	30.5 (28.4-32.8)	12.0 (8.7-16.0)	33.9 (29.2-39.1)	10.3 (8.4-12.4)	32.2 (29.7-35.1)
SEN	1.0 (0.7-1.4)	3.6 (2.5-5.0)	2.1 (1.1-3.7)	6.3 (3.5-10.0)	1.5 (1.0-2.4)	4.9 (3.2-7.0)
SLE	5.2 (4.1-6.4)	18.3 (16.4-20.4)	11.9 (8.2-16.3)	30.1 (26.5-34.0)	8.5 (6.7-10.8)	24.3 (22.2-26.5)
TGO	1.5 (0.9-2.3)	6.0 (3.9-8.7)	2.8 (1.4-5.1)	9.3 (5.1-15.2)	2.1 (1.3-3.4)	7.6 (4.9-11.1)
AVG	6.5 (5.5-7.6)	23.1 (21.6-24.9)	9.2 (6.9-12.2)	26.6 (22.7-30.2)	7.9 (6.6-9.5)	24.8 (22.9-26.9)
Central Africa						
AGO	0.4 (0.2-0.9)	0.7 (0.4-1.3)	1.4 (0.5-3.0)	2.9 (1.4-5.3)	0.9 (0.4-1.8)	1.7 (0.9-3.0)
CMR	6.2 (3.6-10.0)	10.7 (8.4-13.4)	7.0 (3.1-12.8)	14.8 (10.1-20.5)	6.6 (3.9-10.3)	12.7 (10.0-15.9)
TCD	1.4 (0.5-3.2)	2.9 (1.2-5.8)	3.0 (1.0-6.7)	8.9 (4.0-16.4)	2.2 (0.9-4.9)	5.6 (2.7-10.0)
COG	11.4 (8.5-15.2)	20.2 (13.7-27.4)	16.7 (9.9-25.6)	36.8 (24.9-50.5)	14.3 (10.1-19.1)	28.4 (20.7-37.2)
COD	1.1 (0.6-2.1)	1.7 (0.9-2.8)	2.2 (0.9-4.2)	5.0 (2.7-8.4)	1.7 (0.8-3.0)	3.2 (1.9-5.0)
GAB	10.0 (5.7-15.8)	18.3 (14.1-22.8)	13.6 (5.7-25.6)	30.8 (23.7-39.3)	11.9 (6.8-19.1)	24.7 (20.5-29.9)
STP	0.7 (0.2-1.9)	1.3 (0.3-3.5)	2.2 (0.5-5.8)	4.6 (1.3-10.8)	1.4 (0.4-3.4)	2.7 (0.8-6.4)
AVG	2.4 (1.5-3.7)	3.7 (2.8-4.9)	3.6 (1.7-6.2)	8.0 (5.4-11.5)	3.0 (1.8-4.6)	5.7 (4.3-7.6)
Eastern Africa						
BDI	1.8 (0.9-3.2)	5.6 (3.8-7.9)	5.4 (2.8-8.9)	18.9 (12.5-26.8)	3.6 (2.1-5.7)	12.3 (8.8-16.6)
COM	2.5 (1.0-5.0)	8.3 (3.8-15.5)	3.7 (1.4-7.3)	13.3 (6.3-22.7)	3.1 (1.4-5.4)	10.9 (5.6-18.3)
ETH	0.3 (0.1-0.7)	1.0 (0.4-2.0)	0.6 (0.2-1.2)	2.1 (1.1-3.5)	0.4 (0.2-0.8)	1.6 (0.8-2.6)
KEN	1.2 (0.6-2.1)	4.1 (2.4-6.9)	2.7 (1.2-5.2)	9.9 (5.4-16.4)	1.9 (1.1-3.3)	7.0 (4.2-11.0)
MDG	0.7 (0.3-1.4)	2.3 (1.4-3.5)	1.9 (0.8-3.6)	7.5 (5.0-10.6)	1.3 (0.6-2.3)	4.9 (3.5-6.7)
MWI	0.2 (0.1-0.4)	0.7 (0.3-1.5)	0.7 (0.3-1.4)	2.8 (1.4-5.1)	0.4 (0.2-0.8)	1.7 (0.9-3.1)
MOZ	0.2 (0.1-0.6)	0.8 (0.3-1.8)	0.6 (0.2-1.3)	2.6 (1.2-4.8)	0.4 (0.2-0.8)	1.7 (0.8-3.1)
RWA	2.9 (1.5-5.0)	9.0 (7.3-10.8)	7.4 (3.8-12.4)	24.4 (19.3-29.8)	5.0 (2.9-7.7)	16.6 (13.8-19.7)
TZA	6.2 (4.1-9.1)	19.4 (13.2-26.7)	9.1 (4.9-15.1)	30.8 (20.3-44.6)	7.7 (5.0-11.0)	25.4 (18.2-34.0)
UGA	1.3 (0.8-2.0)	4.3 (3.1-5.9)	2.2 (1.1-3.8)	8.2 (5.5-11.6)	1.8 (1.1-2.7)	6.3 (4.6-8.2)
ZMB	0.2 (0.1-0.3)	0.6 (0.3-0.9)	0.7 (0.3-1.3)	2.8 (1.7-4.2)	0.4 (0.2-0.7)	1.7 (1.1-2.4)
ZWE	0.4 (0.2-0.7)	1.2 (0.6-2.3)	0.8 (0.4-1.5)	3.3 (1.7-5.7)	0.6 (0.3-1.0)	2.2 (1.2-3.6)
AVG	1.5 (1.0-2.3)	5.0 (3.6-6.9)	2.8 (1.6-4.5)	10.1 (7.1-14.0)	2.1 (1.4-3.1)	7.6 (5.8-10.0)
Southern Africa						
SWZ	0.7 (0.2-1.9)	5.5 (0.8-18.0)	4.2 (0.9-12.2)	1.8 (0.2-6.8)	2.3 (0.7-6.5)	3.8 (0.7-11.6)
LSO	1.3 (0.2-4.0)	8.5 (3.0-18.9)	5.1 (0.8-16.7)	1.8 (0.3-5.3)	3.3 (0.7-9.9)	5.3 (1.9-11.9)
NAM	0.9 (0.1-2.9)	5.8 (1.6-14.0)	3.1 (0.5-11.1)	1.1 (0.2-3.3)	2.0 (0.4-6.5)	3.5 (1.1-8.3)
ZAF	1.8 (0.2-7.2)	9.4 (4.9-17.0)	6.7 (0.7-26.1)	2.3 (0.5-5.9)	4.3 (0.6-15.4)	6.0 (3.2-10.7)
AVG	1.7 (0.2-6.8)	9.1 (4.7-16.8)	6.5 (0.8-25.1)	2.2 (0.5-5.8)	4.2 (0.6-14.6)	5.9 (3.1-10.5)

Proportion accessing sector for treatment, among those reporting treatment for genital symptoms. Results presented as mean with 95% confidence interval. AVG represents sex- and year-matched population weighted mean estimate for sub-region. ISO3 codes defined in Table C3.

Table C15: Estimated national proportion utilising traditional sector in 2000 and 2020

ISO3	Female (%)		Male (%)		Overall (%)	
	2000	2020	2000	2020	2000	2020
Western Africa						
BEN	11.5 (9.3-14.2)	9.2 (7.6-11.1)	34.0 (25.7-43.8)	23.3 (17.9-29.1)	23.3 (18.5-29.3)	16.0 (13.2-19.2)
BFA	5.2 (3.1-8.2)	4.1 (2.4-6.4)	10.6 (5.5-18.2)	6.5 (3.2-11.2)	7.8 (4.6-12.2)	5.2 (3.1-8.1)
CIV	27.1 (23.2-31.5)	23.0 (19.6-26.9)	35.2 (25.5-45.9)	24.6 (18.0-31.9)	31.4 (25.7-37.7)	23.8 (19.6-28.4)
GMB	8.0 (5.6-10.8)	7.2 (5.5-9.3)	20.2 (11.4-31.4)	14.1 (8.9-21.3)	14.0 (9.1-20.4)	10.6 (7.7-14.4)
GHA	13.2 (10.5-16.2)	8.0 (6.4-9.7)	22.4 (16.0-29.9)	11.4 (8.1-15.6)	18.0 (14.3-22.3)	9.6 (7.7-11.7)
GIN	14.3 (11.2-17.9)	11.6 (9.9-13.5)	15.4 (9.9-22.5)	9.4 (7.0-12.7)	14.9 (11.3-18.9)	10.5 (8.8-12.4)
LBR	2.1 (1.6-2.7)	1.7 (1.3-2.1)	5.5 (3.6-7.9)	3.2 (2.3-4.4)	3.8 (2.7-5.1)	2.4 (1.9-3.1)
MLI	26.0 (22.7-29.3)	22.0 (19.2-24.8)	33.9 (24.6-43.8)	23.4 (17.3-30.7)	30.3 (25.0-36.0)	22.7 (19.1-26.8)
MRT	2.6 (1.4-4.2)	2.1 (1.3-3.3)	7.9 (3.4-15.7)	5.0 (2.1-9.7)	5.0 (2.6-8.7)	3.3 (1.8-5.5)
NER	21.3 (16.0-27.4)	17.6 (13.2-22.8)	31.9 (20.3-45.4)	22.0 (13.3-32.8)	26.8 (19.5-35.2)	19.7 (14.4-26.5)
NGA	17.0 (14.0-20.4)	12.8 (11.3-14.4)	17.0 (12.1-23.5)	9.9 (7.6-12.7)	17.0 (13.8-20.9)	11.4 (9.9-13.0)
SEN	2.8 (2.1-3.8)	2.2 (1.7-2.8)	10.6 (5.6-17.7)	6.6 (3.8-10.3)	6.7 (4.1-10.5)	4.3 (2.8-6.0)
SLE	6.8 (5.2-8.8)	5.1 (4.2-6.1)	8.2 (5.3-12.2)	4.3 (3.2-5.6)	7.5 (5.7-9.9)	4.7 (3.9-5.5)
TGO	17.1 (13.4-21.6)	14.9 (12.2-17.6)	26.0 (16.8-37.5)	18.1 (11.6-26.7)	21.8 (16.4-29.3)	16.5 (12.7-21.2)
AVG	16.1 (13.8-18.8)	12.3 (11.0-13.5)	20.3 (15.0-26.4)	12.3 (9.8-14.8)	18.3 (15.2-21.7)	12.3 (10.8-13.8)
Central Africa						
AGO	5.2 (2.4-10.4)	6.4 (4.0-9.7)	9.5 (4.0-18.7)	7.0 (4.1-11.0)	7.4 (3.7-13.0)	6.7 (4.6-9.2)
CMR	7.7 (3.8-13.4)	10.2 (7.7-13.2)	13.7 (6.3-25.9)	10.5 (7.0-15.2)	10.9 (6.2-18.1)	10.3 (8.0-13.2)
TCD	12.4 (6.4-21.5)	19.6 (13.8-26.6)	21.5 (8.8-39.6)	23.4 (13.5-36.4)	17.3 (9.4-28.8)	21.4 (15.0-29.0)
COG	3.5 (2.2-5.3)	4.7 (2.8-7.6)	8.6 (4.2-16.1)	6.9 (3.4-11.8)	6.3 (3.7-10.6)	5.7 (3.6-8.7)
COD	10.8 (6.1-17.8)	12.4 (8.7-17.0)	18.8 (10.6-31.2)	15.8 (9.5-23.9)	15.3 (9.8-22.9)	14.0 (9.9-18.7)
GAB	3.9 (1.8-7.7)	5.4 (3.5-7.9)	10.4 (4.2-20.5)	8.5 (5.0-13.1)	7.4 (3.6-13.4)	7.0 (4.8-9.7)
STP	1.2 (0.3-3.0)	1.6 (0.5-4.2)	5.3 (1.5-13.0)	4.0 (1.3-9.5)	3.0 (1.0-7.0)	2.6 (0.9-6.1)
AVG	9.1 (5.0-15.2)	11.3 (8.2-14.7)	16.3 (8.8-27.4)	13.5 (8.7-19.2)	13.1 (8.2-20.0)	12.3 (9.3-15.6)
Eastern Africa						
BDI	3.0 (1.5-5.1)	1.3 (0.7-2.1)	6.3 (3.1-10.8)	2.6 (1.3-4.6)	4.6 (2.6-7.5)	1.9 (1.1-3.2)
COM	14.8 (8.2-23.4)	6.5 (3.8-10.4)	20.4 (10.7-34.1)	9.0 (4.6-15.4)	17.5 (10.4-27.1)	7.8 (4.5-12.3)
ETH	3.2 (0.9-7.8)	1.4 (0.4-3.3)	5.2 (2.0-11.3)	2.3 (0.9-4.8)	4.2 (1.6-8.9)	1.8 (0.7-4.0)
KEN	7.9 (5.2-11.5)	3.5 (2.3-5.0)	10.8 (6.4-17.6)	4.8 (2.9-7.3)	9.4 (6.5-13.4)	4.1 (2.8-5.6)
MDG	12.0 (7.6-17.3)	5.4 (3.8-7.5)	24.2 (15.7-34.2)	11.5 (8.2-15.3)	18.3 (13.0-24.7)	8.4 (6.4-10.7)
MWI	33.9 (27.5-40.9)	17.8 (14.7-21.3)	25.9 (18.3-35.3)	12.6 (9.2-17.0)	30.2 (24.8-35.8)	15.2 (12.6-18.2)
MOZ	10.5 (6.7-15.9)	4.8 (3.1-7.2)	13.9 (8.8-20.2)	6.9 (4.8-10.0)	12.2 (8.7-16.4)	5.8 (4.2-7.9)
RWA	8.2 (5.6-11.9)	3.4 (2.6-4.3)	11.2 (6.6-17.4)	4.4 (3.0-6.3)	9.6 (6.8-13.4)	3.9 (3.0-5.0)
TZA	16.2 (12.2-20.9)	6.6 (4.8-8.8)	17.0 (10.1-26.5)	6.8 (3.9-11.1)	16.6 (12.1-22.3)	6.7 (4.7-9.3)
UGA	6.5 (4.8-8.7)	2.9 (2.3-3.6)	8.0 (4.8-12.3)	3.4 (2.2-5.0)	7.2 (5.3-9.6)	3.2 (2.4-4.1)
ZMB	6.9 (4.5-10.3)	3.0 (2.1-4.2)	12.7 (8.4-18.1)	6.3 (4.5-8.3)	9.8 (7.2-13.0)	4.6 (3.6-5.9)
ZWE	8.3 (6.0-11.4)	3.7 (2.5-5.3)	19.5 (14.5-25.3)	9.2 (6.3-12.6)	13.6 (10.9-16.8)	6.2 (4.6-8.0)
AVG	9.5 (7.2-12.2)	4.1 (3.3-5.1)	12.4 (8.6-17.5)	5.3 (4.0-7.0)	11.0 (8.5-13.8)	4.7 (3.9-5.7)
Southern Africa						
SWZ	1.2 (0.4-2.9)	0.5 (0.1-1.3)	3.1 (1.1-6.5)	7.0 (1.9-16.7)	2.1 (0.9-4.1)	3.4 (1.0-8.2)
LSO	12.1 (4.6-23.1)	4.6 (2.2-8.9)	12.4 (4.4-25.8)	25.1 (13.5-41.3)	12.2 (5.9-22.2)	14.6 (8.1-23.7)
NAM	2.1 (0.6-4.9)	0.8 (0.3-1.8)	2.5 (0.8-5.9)	5.2 (1.9-11.4)	2.3 (0.9-4.7)	3.0 (1.2-6.1)
ZAF	2.2 (0.5-6.3)	0.7 (0.3-1.5)	4.1 (0.9-10.8)	8.7 (3.9-15.5)	3.1 (0.9-7.7)	4.5 (2.2-7.9)
AVG	2.5 (0.6-6.8)	0.9 (0.4-1.6)	4.4 (1.0-10.9)	9.1 (4.3-16.3)	3.4 (1.1-7.9)	4.7 (2.4-8.0)

Proportion accessing sector for treatment, among those reporting treatment for genital symptoms. Results presented as mean with 95% confidence interval. AVG represents sex- and year-matched population weighted mean estimate for sub-region. ISO3 codes defined in Table C3.

Table C16: Estimated national proportion utilising multiple sectors in 2000 and 2020

ISO3	Female (%)		Male (%)		Overall (%)	
	2000	2020	2000	2020	2000	2020
Western Africa						
BEN	6.3 (4.5-8.6)	3.2 (2.4-4.2)	10.0 (6.0-15.1)	6.8 (4.6-9.9)	8.2 (5.9-11.3)	4.9 (3.7-6.6)
BFA	1.7 (0.7-3.5)	0.8 (0.3-1.7)	3.3 (1.3-7.0)	2.0 (0.8-4.1)	2.4 (1.1-4.8)	1.4 (0.6-2.7)
CIV	12.0 (9.2-15.3)	6.4 (4.8-8.2)	11.2 (6.3-18.0)	7.8 (4.9-11.7)	11.6 (8.6-15.5)	7.1 (5.3-9.2)
GMB	11.9 (8.1-16.6)	6.8 (5.1-9.0)	9.2 (4.8-15.6)	6.4 (3.8-9.6)	10.6 (7.1-14.6)	6.6 (4.8-8.7)
GHA	6.6 (4.7-9.2)	2.5 (1.8-3.4)	10.9 (6.7-16.6)	5.6 (3.5-8.3)	8.9 (6.3-12.2)	4.0 (2.9-5.5)
GIN	5.3 (3.7-7.2)	2.7 (2.1-3.3)	7.1 (4.1-11.4)	4.3 (2.7-6.9)	6.2 (4.3-8.7)	3.5 (2.6-4.8)
LBR	14.0 (10.9-17.6)	7.0 (5.7-8.4)	10.0 (6.5-14.4)	5.9 (4.1-8.2)	12.0 (9.6-14.9)	6.4 (5.3-7.7)
MLI	8.5 (6.5-10.8)	4.5 (3.6-5.5)	7.3 (4.3-11.4)	5.0 (3.2-7.7)	7.9 (5.8-10.5)	4.8 (3.6-6.1)
MRT	3.0 (1.5-5.4)	1.5 (0.8-2.5)	5.8 (2.3-11.9)	3.8 (1.5-7.3)	4.3 (2.1-7.5)	2.4 (1.2-4.2)
NER	6.3 (3.7-9.9)	3.3 (1.9-5.2)	9.5 (4.4-17.2)	6.6 (3.1-11.4)	8.0 (4.6-12.7)	4.8 (2.8-7.6)
NGA	6.9 (5.2-8.8)	3.3 (2.7-3.9)	15.3 (10.1-22.0)	9.0 (6.6-11.8)	11.2 (8.4-14.6)	6.1 (4.9-7.6)
SEN	1.2 (0.7-2.0)	0.6 (0.4-0.9)	3.7 (1.7-6.7)	2.3 (1.2-4.1)	2.4 (1.3-4.1)	1.4 (0.8-2.3)
SLE	9.9 (7.4-12.7)	4.6 (3.7-5.7)	11.4 (6.9-16.7)	6.0 (4.6-7.7)	10.6 (8.3-13.7)	5.3 (4.4-6.3)
TGO	3.1 (1.8-4.9)	1.7 (1.1-2.6)	4.8 (2.3-8.5)	3.3 (1.7-5.7)	4.0 (2.3-6.2)	2.5 (1.5-3.8)
AVG	6.8 (5.4-8.5)	3.2 (2.8-3.8)	12.0 (8.0-17.0)	7.2 (5.5-9.2)	9.5 (7.3-12.1)	5.2 (4.3-6.2)
Central Africa						
AGO	1.9 (0.6-4.5)	0.4 (0.2-0.9)	3.8 (1.2-9.0)	0.8 (0.3-1.5)	2.9 (1.1-5.8)	0.6 (0.3-1.0)
CMR	6.7 (3.1-12.4)	1.6 (1.0-2.4)	11.9 (5.0-22.8)	2.5 (1.3-4.2)	9.5 (4.7-16.0)	2.0 (1.3-3.0)
TCD	24.7 (11.4-42.5)	7.0 (4.0-11.4)	37.9 (18.4-60.4)	11.8 (5.6-20.7)	32.2 (17.6-49.1)	9.2 (5.5-14.1)
COG	13.6 (8.2-20.2)	3.3 (1.8-5.4)	29.7 (17.0-43.7)	6.7 (3.2-12.4)	22.4 (14.5-31.9)	5.0 (3.0-7.9)
COD	10.9 (5.7-19.4)	2.2 (1.3-3.6)	17.3 (8.5-30.2)	4.0 (2.1-7.2)	14.5 (8.6-22.2)	3.0 (1.9-4.7)
GAB	23.3 (11.8-39.3)	5.8 (3.7-9.0)	30.0 (13.4-51.4)	6.9 (4.0-10.9)	27.3 (15.0-42.7)	6.4 (4.5-9.0)
STP	2.1 (0.7-5.3)	0.5 (0.2-1.3)	6.7 (2.3-15.0)	1.4 (0.5-3.5)	4.2 (1.6-8.2)	0.9 (0.3-2.1)
AVG	10.4 (5.4-18.7)	2.3 (1.5-3.3)	17.2 (8.5-29.6)	3.9 (2.2-6.7)	14.2 (8.2-22.2)	3.1 (2.1-4.4)
Eastern Africa						
BDI	5.7 (3.4-8.8)	3.2 (2.2-4.5)	20.3 (11.6-31.1)	3.4 (1.9-5.6)	13.0 (8.1-19.5)	3.3 (2.3-4.6)
COM	2.8 (1.0-6.3)	1.7 (0.6-3.7)	10.0 (3.8-20.1)	1.7 (0.6-3.5)	6.3 (2.6-12.6)	1.7 (0.7-3.4)
ETH	4.2 (1.7-8.5)	2.4 (1.0-4.9)	12.8 (5.9-23.0)	2.2 (1.1-4.0)	8.6 (4.3-15.2)	2.3 (1.2-4.0)
KEN	5.0 (3.1-8.0)	2.9 (1.9-4.5)	17.4 (9.7-27.1)	3.0 (1.7-4.9)	11.3 (7.0-16.8)	3.0 (2.0-4.2)
MDG	3.3 (1.6-5.9)	2.0 (1.1-3.3)	10.9 (5.8-18.7)	2.0 (1.2-3.2)	7.2 (4.2-11.7)	2.0 (1.3-2.9)
MWI	2.6 (1.6-3.9)	1.8 (1.2-2.7)	8.5 (4.6-14.4)	1.6 (0.9-2.5)	5.4 (3.4-8.2)	1.7 (1.2-2.4)
MOZ	6.3 (3.9-9.6)	3.8 (2.4-5.6)	18.5 (11.3-28.6)	3.6 (2.3-5.4)	12.4 (8.4-18.3)	3.7 (2.6-5.1)
RWA	2.9 (1.7-4.6)	1.6 (1.1-2.2)	13.9 (7.5-23.5)	2.1 (1.3-3.4)	8.0 (4.7-13.1)	1.9 (1.3-2.6)
TZA	4.2 (2.5-6.5)	2.3 (1.4-3.6)	16.1 (7.7-27.0)	2.5 (1.2-4.7)	10.3 (5.7-16.7)	2.4 (1.4-3.8)
UGA	7.3 (5.3-9.8)	4.3 (3.4-5.4)	18.0 (11.6-26.3)	3.1 (1.9-4.7)	12.6 (9.1-17.2)	3.7 (2.8-4.6)
ZMB	3.8 (2.2-6.2)	2.2 (1.4-3.4)	19.0 (12.3-28.4)	3.7 (2.5-5.4)	11.4 (7.6-16.3)	2.9 (2.1-3.9)
ZWE	2.4 (1.5-3.8)	1.4 (0.8-2.2)	10.0 (6.4-14.7)	1.9 (1.1-3.2)	6.0 (4.1-8.6)	1.6 (1.1-2.4)
AVG	4.5 (3.1-6.4)	2.7 (2.0-3.5)	14.9 (9.2-21.7)	2.6 (1.8-3.6)	9.7 (6.6-13.5)	2.6 (2.0-3.3)
Southern Africa						
SWZ	0.9 (0.2-2.6)	3.0 (0.4-10.0)	6.1 (1.6-15.8)	2.3 (0.2-8.8)	3.4 (1.1-8.7)	2.7 (0.4-8.4)
LSO	2.4 (0.6-6.9)	7.4 (2.4-17.2)	11.7 (2.1-34.8)	3.3 (0.6-9.8)	7.4 (1.7-22.0)	5.5 (1.9-12.1)
NAM	0.7 (0.1-1.9)	2.2 (0.5-6.0)	4.4 (0.6-15.3)	1.3 (0.2-4.2)	2.7 (0.6-8.8)	1.7 (0.4-4.3)
ZAF	0.8 (0.1-3.1)	1.9 (0.9-3.6)	5.3 (0.6-19.5)	1.5 (0.2-5.5)	3.1 (0.4-11.9)	1.7 (0.8-3.6)
AVG	0.9 (0.1-3.2)	2.1 (0.9-4.1)	5.5 (0.7-20.2)	1.6 (0.3-5.7)	3.3 (0.5-12.1)	1.9 (0.9-3.8)

Proportion accessing multiple sectors for treatment, among those reporting treatment for genital symptoms. Results presented as mean with 95% confidence interval. AVG represents sex- and year-matched population weighted mean estimate for sub-region. ISO3 codes defined in Table C3.

Appendix D

Supplement to Chapter 5

D.1 Supplementary methods

D.1.1 Variable definitions

Table D1: Definition of outcome variables from survey questions

Variable	Identifier (Questionnaire)	Question
STI identified in past year	v763a (IR) mv763a (MR)	Now I would like to ask you some questions about your health in the past year. During the past year, have you had a disease which you got through sexual contact?
Genital ulcer in past year	v763b (IR)	Sometimes women have a genital sore or ulcer. During the past year, have you had a genital sore or ulcer?
	mv763b (MR)	Sometimes men have a sore or ulcer near their penis. During the past year, have you had a sore or ulcer on or near your penis?
Genital discharge in past year	v763c (IR)	Sometimes women experience a bad-smelling abnormal genital discharge. During the past year, have you had a bad-smelling abnormal genital discharge?
	mv763c (MR)	Sometimes men experience an abnormal discharge from their penis. During the past year, have you had an abnormal discharge from your penis?
Treatment seeking for STI or STI-related symptom in past year	v770 (IR) mv770 (MR)	The last time you had (PROBLEM), did you seek any kind of advice or treatment?
Sector for treatment of STI or STI-related symptom in past year	v770 a-x (IR) mv770 a-x (MR)	Where did you go? Public sector • Government hospital • Government health centre • Stand along HTC centre • Family planning clinic • Mobile HTC services • Other public sector Private medical sector • Private hospital/clinic/private doctor • Stand-alone HTC centre • Pharmacy • Mobile HTC services • Other private medical sector Other source • Shop • Other

IR: Individual women's recode file, MR: Men's recode file.

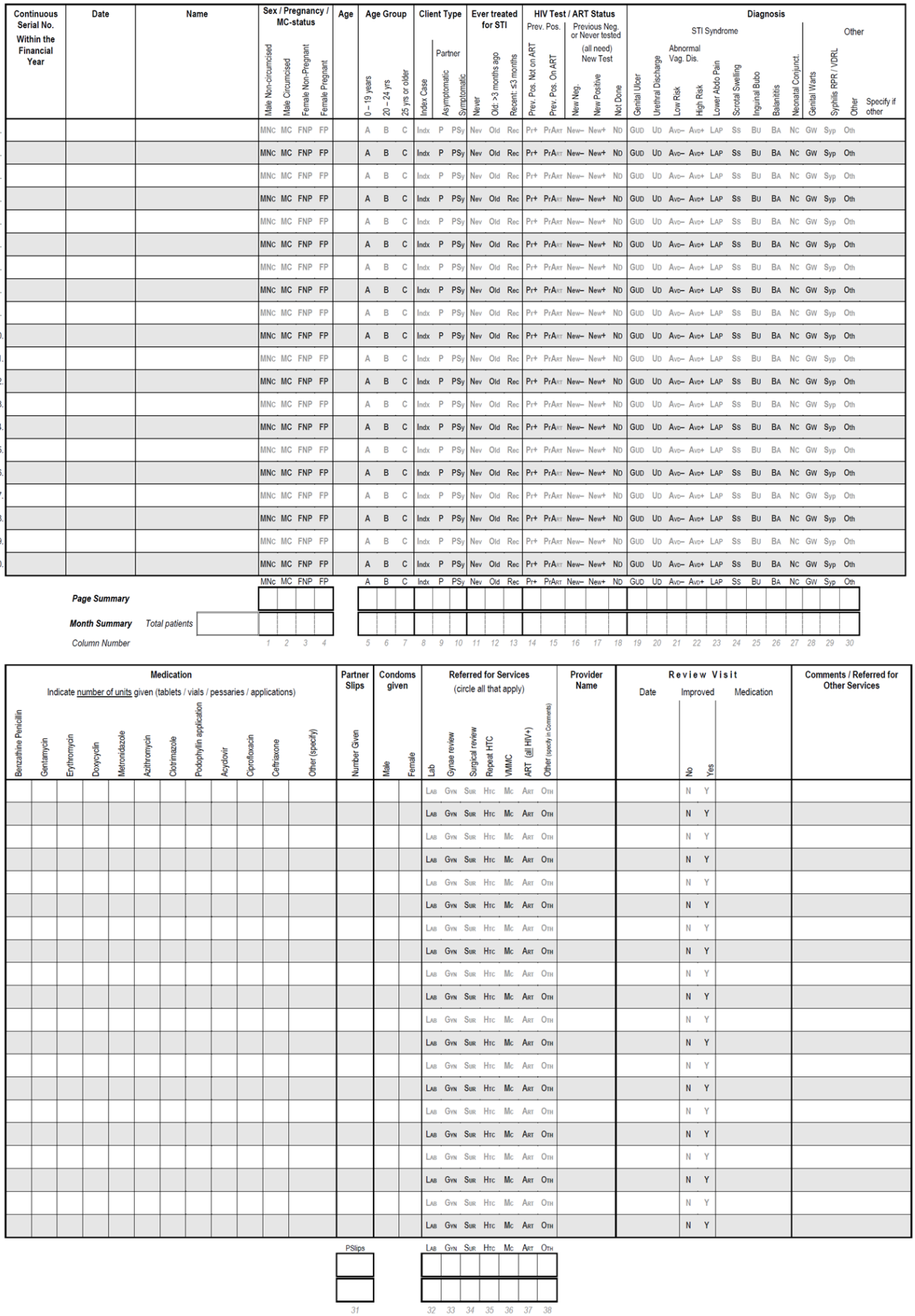


Figure D1: Paper STI client register in Malawi

Paper client register used by Malawi Department of HIV & AIDS, STIs, and Viral Hepatitis

D.2 Supplementary results

Table D2: Characteristics and behaviours of sexually active respondents in Malawi Demographic and Health Surveys

	DHS 2000		DHS 2004		DHS 2010		DHS 2015-16	
	Male	**Female**	**Male**	**Female**	**Male**	**Female**	**Male**	**Female**
	N = 2 758	N = 11 451	N = 2 816	N = 9 581	N = 5 852	N = 18 201	N = 6 277	N = 20 806
Age group								
15-19	403 (15%)	1 676 (15%)	358 (13%)	1 277 (13%)	894 (15%)	2 109 (12%)	930 (15%)	2 630 (13%)
20-24	537 (19%)	2 797 (24%)	517 (18%)	2 520 (26%)	1 007 (17%)	3 827 (21%)	1 217 (19%)	4 639 (22%)
25-29	537 (19%)	2 250 (20%)	592 (21%)	1 936 (20%)	1 008 (17%)	3 909 (21%)	997 (16%)	3 747 (18%)
30-34	331 (12%)	1 504 (13%)	458 (16%)	1 331 (14%)	877 (15%)	2 966 (16%)	917 (15%)	3 488 (17%)
35-39	332 (12%)	1 353 (12%)	274 (10%)	996 (10%)	736 (13%)	2 314 (13%)	844 (13%)	2 870 (14%)
40+	618 (22%)	1 871 (16%)	617 (22%)	1 521 (16%)	1 330 (23%)	3 076 (17%)	1 372 (22%)	3 432 (16%)
Region of residence								
Northern	460 (17%)	1896 (17%)	365 (13%)	1261 (13%)	993 (17%)	3355 (18%)	1300 (21%)	4001 (19%)
Central	992 (36%)	3830 (33%)	1080 (38%)	3299 (34%)	2186 (37%)	6232 (34%)	2239 (36%)	7064 (34%)
Southern	1306 (47%)	5725 (50%)	1371 (49%)	5021 (52%)	2673 (46%)	8614 (47%)	2738 (44%)	9741 (47%)
Residence type								
Rural	2 130 (77%)	9 012 (79%)	2 397 (85%)	8 304 (87%)	5 057 (86%)	15 864 (87%)	4 896 (78%)	16 451 (79%)
Urban	628 (23%)	2 439 (21%)	419 (15%)	1 277 (13%)	795 (14%)	2 337 (13%)	1 381 (22%)	4 355 (21%)
Marital status								
Never married	738 (27%)	905 (7.9%)	670 (24%)	653 (6.8%)	1 611 (28%)	1 313 (7.2%)	1 821 (29%)	2 322 (11%)
Formerly married	124 (4.5%)	1 500 (13%)	105 (3.7%)	1 291 (13%)	247 (4.2%)	2 761 (15%)	258 (4.1%)	3 139 (15%)
Married or living together	1 896 (69%)	9 046 (79%)	2 041 (72%)	7 637 (80%)	3 994 (68%)	14 127 (78%)	4 198 (67%)	15 345 (74%)
Highest education								
None	292 (11%)	3 166 (28%)	331 (12%)	2 425 (25%)	396 (6.8%)	2 955 (16%)	373 (5.9%)	2 581 (12%)
Primary	1 839 (67%)	6 986 (61%)	1 778 (63%)	5 906 (62%)	3 697 (63%)	12 109 (67%)	3 469 (55%)	12 776 (61%)
Secondary or higher	627 (23%)	1 299 (11%)	707 (25%)	1 250 (13%)	1 759 (30%)	3 137 (17%)	2 435 (39%)	5 449 (26%)
Employment								
No or unknown	972 (35%)	4 650 (41%)	1 035 (37%)	3 893 (41%)	809 (14%)	7 287 (40%)	878 (14%)	7 120 (34%)
Yes	1 786 (65%)	6 801 (59%)	1 781 (63%)	5 688 (59%)	5 043 (86%)	10 914 (60%)	5 399 (86%)	13 686 (66%)

Continued...

	DHS 2000		DHS 2004		DHS 2010		DHS 2015-16	
	Male	**Female**	**Male**	**Female**	**Male**	**Female**	**Male**	**Female**
	N = 2 758	N = 11 451	N = 2 816	N = 9 581	N = 5 852	N = 18 201	N = 6 277	N = 20 806
Age at first sex								
<15	541 (20%)	2 706 (24%)	371 (13%)	2 080 (22%)	1 108 (19%)	3 733 (21%)	1 016 (16%)	3 908 (19%)
15-19	1 530 (55%)	7 550 (66%)	1 744 (62%)	6 545 (68%)	3 177 (54%)	12 528 (69%)	3 675 (59%)	14 875 (71%)
20-24	577 (21%)	1 088 (9.5%)	591 (21%)	884 (9.2%)	1 296 (22%)	1 776 (9.8%)	1 299 (21%)	1 892 (9.1%)
25+	110 (4.0%)	107 (0.9%)	110 (3.9%)	72 (0.8%)	271 (4.6%)	164 (0.9%)	287 (4.6%)	131 (0.6%)
Number of sex partners in last 12 months[1]								
0	291 (11%)	1 330 (12%)	299 (11%)	1 188 (12%)	799 (14%)	2609 (14%)	632 (10%)	3 080 (15%)
1	2 029 (74%)	10 004 (87%)	2 211 (79%)	8 302 (87%)	4 390 (75%)	15 472 (85%)	4 658 (74%)	17 455 (84%)
2+	438 (16%)	117 (1%)	306 (11%)	91 (1%)	663 (11%)	120 (1%)	987 (16%)	271 (1%)
Condom use at last sex in past 12 months[1]								
No or unknown	2 108 (76%)	9 609 (84%)	2 143 (76%)	7 983 (83%)	4 025 (69%)	14 187 (78%)	4 021 (64%)	15 641 (75%)
Yes	359 (13%)	512 (4.5%)	374 (13%)	410 (4.3%)	1 028 (18%)	1 405 (7.7%)	1 624 (26%)	2 085 (10%)
Not applicable	291 (11%)	1 330 (12%)	299 (11%)	1 188 (12%)	799 (14%)	2 609 (14%)	632 (10%)	3 080 (15%)
Ever tested for HIV								
No or unknown	2 293 (83%)	10 364 (91%)	2 331 (83%)	8 085 (84%)	2 487 (42%)	3 460 (19%)	1 428 (23%)	1 807 (9%)
Yes	465 (17%)	1 087 (9%)	485 (17%)	1 496 (16%)	3 365 (58%)	14 741 (81%)	4 849 (77%)	18 999 (91%)
Knowledge of STIs								
No or unknown	99 (3.6%)	970 (8.5%)	236 (8.4%)	1 175 (12%)	315 (5.4%)	1 310 (7.2%)	373 (5.9%)	2 431 (12%)
Yes	2 659 (96%)	10 481 (92%)	2 580 (92%)	8 406 (88%)	5 537 (95%)	16 891 (93%)	5 904 (94%)	18 375 (88%)

[1]Measured among those who reported sex in the past 12 months. Frequencies are unweighted; proportions are survey weighted.

Table D3: Adjusted odds of treatment-seeking among ever sexually active adults who reported a genital symptom or known STI in the last year, estimated via generalised linear mixed-effects models

	Female	Male
	aOR (95% CrI)	aOR (95% CrI)
Intercept	0.25 (0.16-0.39)	0.61 (0.19-1.94)
Year (centred at 2010)	0.95 (0.93-0.97)	0.96 (0.92-1.00)
Age group		
15-19	0.64 (0.52-0.79)	0.47 (0.25-0.86)
20-24	0.95 (0.81-1.11)	0.72 (0.44-1.16)
25-29	Ref	Ref
30-34	1.06 (0.90-1.25)	1.03 (0.65-1.66)
35-39	0.94 (0.79-1.12)	1.24 (0.76-2.00)
40+	1.04 (0.88-1.24)	1.13 (0.72-1.78)
Residence type		
Rural	Ref	Ref
Urban	1.43 (1.21-1.69)	1.69 (1.19-2.43)
Region		
Northern	Ref	Ref
Central	1.01 (0.75-1.35)	0.42 (0.24-0.70)
Southern	1.26 (0.95-1.68)	0.54 (0.31-0.88)
Marital status		
Never married	Ref	Ref
Formerly married	1.36 (1.04-1.79)	1.87 (0.96-3.70)
Married or living together	1.19 (0.92-1.53)	1.28 (0.79-2.17)
Highest education		
None	Ref	Ref
Primary	1.16 (1.01-1.34)	1.24 (0.72-2.18)
Secondary or higher	1.37 (1.13-1.68)	1.13 (0.62-2.11)
Employment		
No or unknown	Ref	Ref
Yes	1.01 (0.90-1.13)	1.05 (0.72-1.57)
Age at first sex		
<15	1.09 (0.96-1.23)	1.27 (0.89-1.78)
15-19	Ref	Ref
20-24	0.87 (0.71-1.05)	1.20 (0.81-1.80)
25+	0.95 (0.49-1.86)	1.65 (0.73-3.82)
Number of sex partners in past year[1]		
None	Ref	Ref
1	1.07 (0.88-1.30)	1.42 (0.85-2.37)
2+	1.28 (0.86-1.89)	2.16 (1.23-3.76)
Condom use at last sex in past year[1]		
No or unknown	Ref	Ref
Yes	1.23 (1.03-1.50)	0.90 (0.62-1.32)
Ever tested for HIV		
No or unknown	Ref	Ref
Yes	1.48 (1.26-1.73)	1.53 (1.12-2.08)

Continued...

	Female	Male
	aOR (95% CrI)	aOR (95% CrI)
Knowledge of STIs		
No or unknown	Ref	Ref
Yes	1.50 (1.25-1.79)	0.82 (0.42-1.62)
Symptom		
None	1.18 (0.94-1.47)	1.19 (0.70-2.04)
Discharge	Ref	Ref
Ulcer	1.70 (1.50-1.94)	1.14 (0.82-1.59)
Discharge and ulcer	2.39 (2.04-2.80)	2.65 (1.74-4.06)
Random effects		
τ_{00} geographic area	0.23	0.11
τ_{00} cluster	0.21	0.82
τ_{11} geographic area	0.03	0.07
ρ_{01} geographic area	0.22	0.16

[1]Measured among those who reported sex in the past 12 months. aOR: adjusted odds ratio, CrI: credible interval, Ref: Reference, τ_{00}: random intercept variance, τ_{11}: random slope variance, ρ_{01}: correlation between intercept and slope.

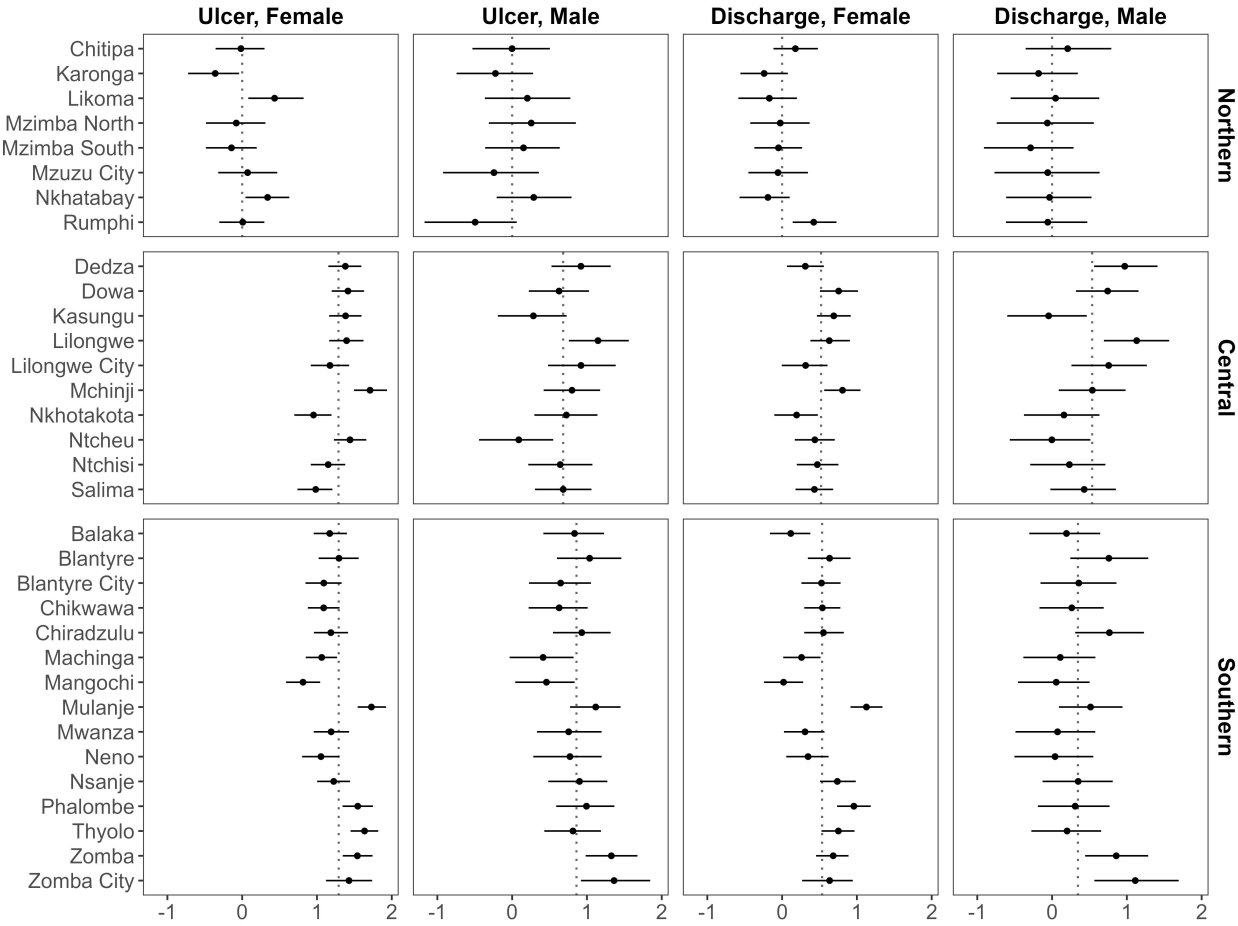


Figure D2: Random intercepts for models estimating the odds of self-reported symptoms

Random effect intercepts on the logit scale for models estimating the odds of self-reported symptoms in the last year among ever sexually active adults in Malawi. Random intercepts offset by regional fixed effects. Error bars represent 95% credible intervals.

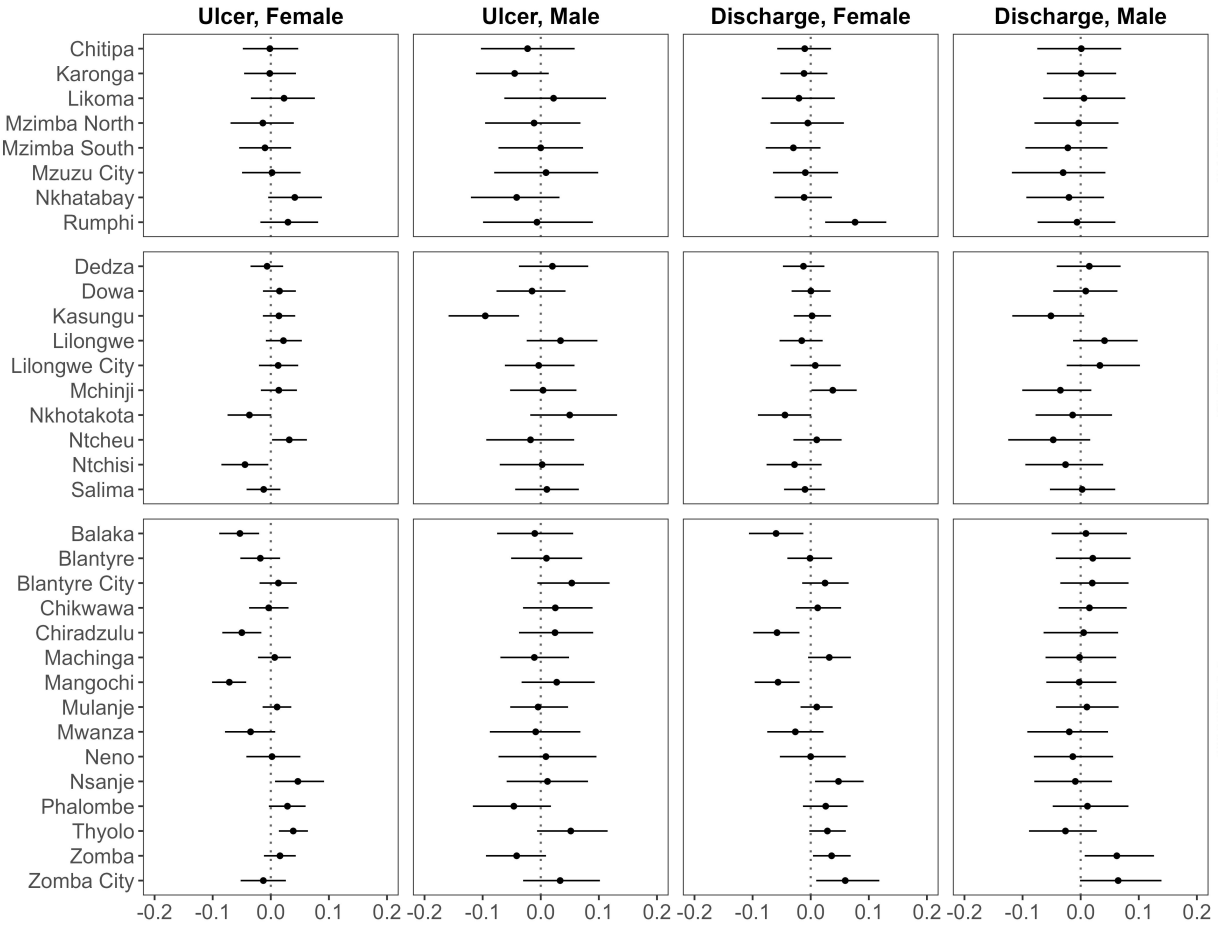


Figure D3: Random slopes for models estimating the odds of self-reported symptoms

Random effect slopes on the logit scale for models estimating the odds of self-reported symptoms in the last year among ever sexually active adults in Malawi. Error bars represent 95% credible intervals.

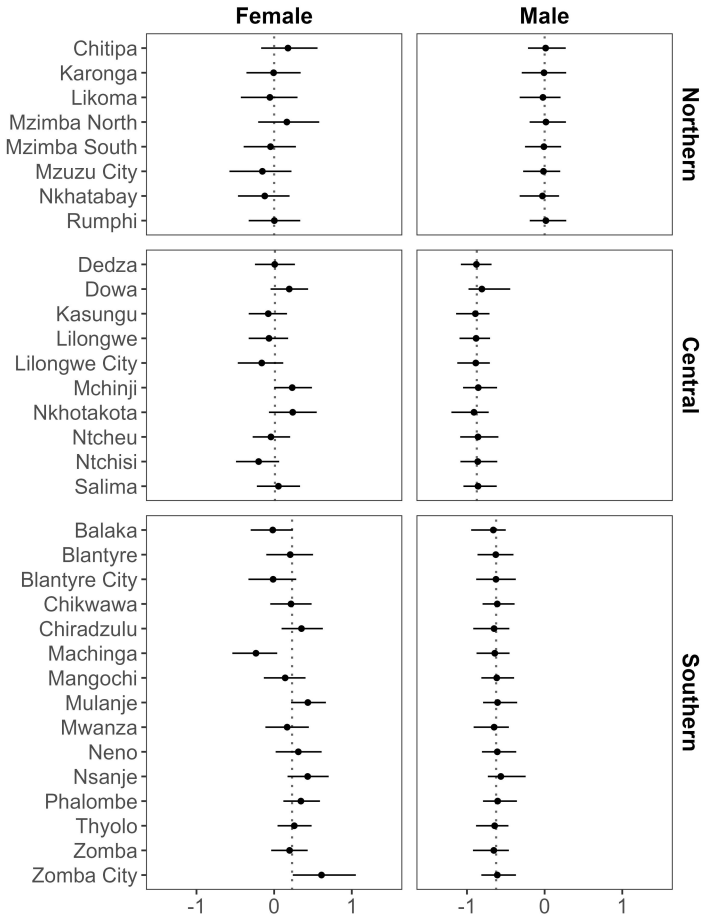


Figure D4: Random intercepts for models estimating the odds of treatment-seeking

Random effect intercepts on the logit scale for models estimating the odds of treatment-seeking, among ever sexually active adults in Malawi who reported an STI or STI symptom in the last year. Random intercepts offset by regional fixed effects. Error bars represent 95% credible intervals.

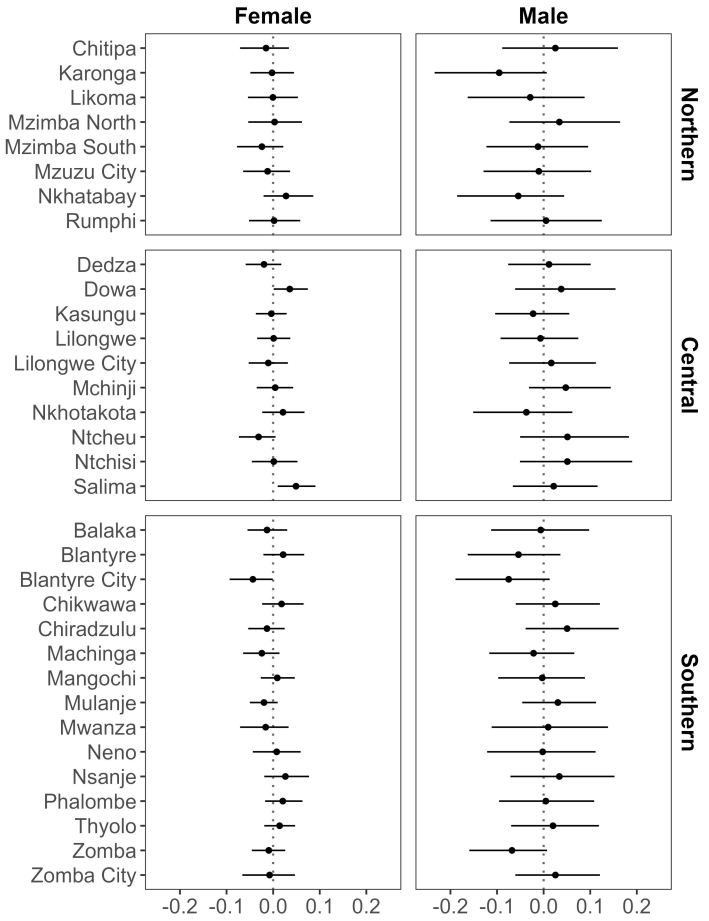


Figure D5: Random slopes for models estimating the odds of treatment-seeking

Random effect slopes on the logit scale for models estimating the odds of treatment-seeking, among ever sexually active adults in Malawi who reported an STI or STI symptom in the last year. Error bars represent 95% credible intervals.

Table D4: Characteristics of case reports in the Malawi Department of HIV and AIDS Management Information System

	2014 N = 212 404	2015 N = 227 144	2016 N = 264 493	2017 N = 304 341	2018 N = 355 236	2019 N = 413 142	2020 N = 386 346	2021 N = 384 631	Total N = 2 547 737
Sex									
Male	85 670 (40%)	91 239 (40%)	107 431 (41%)	124 046 (41%)	142 020 (40%)	164 432 (40%)	157 834 (41%)	159 721 (42%)	1 032 393 (41%)
Female	126 734 (60%)	135 905 (60%)	157 062 (59%)	180 295 (59%)	213 216 (60%)	248 710 (60%)	228 512 (59%)	224 910 (58%)	1 515 344 (59%)
Age group									
0 - 19	18 696 (8.7%)	20 314 (8.9%)	23 960 (9.1%)	27 857 (9.2%)	31 091 (8.8%)	37 104 (9.0%)	31 341 (8.1%)	32 616 (8.5%)	222 979 (8.8%)
20 - 24	50 908 (24%)	54 436 (24%)	63 756 (24%)	72 578 (24%)	84 780 (24%)	92 849 (22%)	89 163 (23%)	91 298 (24%)	599 768 (24%)
25+	142 800 (67%)	152 394 (67%)	176 777 (67%)	203 906 (67%)	239 365 (67%)	283 189 (69%)	265 842 (69%)	260 717 (68%)	1 724 990 (68%)
Region									
Central	71 067 (33%)	78 300 (34%)	88 426 (33%)	105 197 (35%)	122 507 (34%)	143 486 (35%)	136 333 (35%)	141 688 (37%)	887 004 (35%)
Northern	27 614 (13%)	31 395 (14%)	35 424 (13%)	40 793 (13%)	48 862 (14%)	54 558 (13%)	48 661 (13%)	51 462 (13%)	338 769 (13%)
Southern	113 723 (54%)	117 449 (52%)	140 643 (53%)	158 351 (52%)	183 867 (52%)	215 098 (52%)	201 352 (52%)	191 481 (50%)	1 321 964 (52%)
HIV status									
HIV-positive	27 098 (13%)	31 327 (14%)	42 568 (16%)	48 406 (16%)	56 638 (16%)	67 204 (16%)	64 617 (17%)	62 844 (16%)	400 702 (16%)
HIV-negative	77 484 (36%)	93 963 (41%)	152 706 (58%)	205 193 (67%)	257 466 (72%)	301 261 (73%)	283 883 (73%)	280 812 (73%)	1 652 768 (65%)
Unknown	107 822 (51%)	101 854 (45%)	69 219 (26%)	50 742 (17%)	41 132 (12%)	44 677 (11%)	37 846 (10%)	40 975 (11%)	494 267 (19%)
Client type									
Symptomatic index	169 686 (80%)	183 836 (81%)	214 733 (81%)	249 298 (82%)	291 509 (82%)	336 625 (81%)	316 069 (82%)	316 910 (82%)	2 078 666 (82%)
Symptomatic partner	17 253 (8.1%)	17 788 (7.8%)	21 319 (8.1%)	22 036 (7.2%)	23 414 (6.6%)	27 343 (6.6%)	27 626 (7.2%)	27 556 (7.2%)	184 335 (7%)
Asymptomatic partner	25 465 (12%)	25 520 (11%)	28 441 (11%)	33 007 (11%)	40 313 (11%)	49 174 (12%)	42 651 (11%)	40 165 (10%)	284 736 (11%)
STI treatment history									
$\leq$ 3 months	18 232 (8.6%)	17 996 (7.9%)	18 763 (7.1%)	20 964 (6.9%)	23 197 (6.5%)	28 165 (6.8%)	25 287 (6.5%)	23 956 (6.2%)	176 560 (7.2%)
> 3 months	34 589 (16%)	40 735 (18%)	47 856 (18%)	52 887 (17%)	65 988 (19%)	77 254 (19%)	74 946 (19%)	76 558 (20%)	470 813 (18%)
Never treated	159 583 (75%)	168 413 (74%)	197 874 (75%)	230 490 (76%)	266 051 (75%)	307 723 (74%)	286 113 (74%)	284 117 (74%)	1 900 364 (75%)
Diagnosis[1]									
UDS	52 276 (23%)	55 586 (23%)	68 718 (25%)	79 165 (25%)	91 163 (25%)	106 724 (25%)	109 412 (27%)	112 790 (28%)	675 834 (25%)
VDS high risk	41 117 (18%)	44 350 (19%)	51 371 (19%)	62 160 (20%)	79 740 (22%)	92 707 (22%)	87 657 (22%)	86 834 (22%)	545 936 (21%)
VDS low risk	25 090 (11%)	28 496 (12%)	30 802 (11%)	32 325 (10%)	35 375 (9.6%)	37 633 (8.8%)	32 655 (8.2%)	32 903 (8.2%)	255 279 (10%)
GUD	37 158 (17%)	40 625 (17%)	44 073 (16%)	47 748 (15%)	50 686 (14%)	55 480 (13%)	48 309 (12%)	48 877 (12%)	372 956 (14%)
LAP	36 316 (16%)	36 796 (15%)	41 164 (15%)	47 077 (15%)	54 571 (15%)	56 732 (13%)	49 842 (12%)	46 986 (12%)	369 484 (14%)
Other[2]	30 943 (14%)	33 930 (14%)	40 075 (15%)	48 415 (15%)	57 656 (16%)	80 863 (19%)	71 842 (18%)	73 861 (18%)	437 585 (16%)
Total	222 900 (100%)	239 784 (100%)	276 203 (100%)	316 890 (100%)	369 191 (100%)	430 139 (100%)	399 718 (100%)	402 251 (100%)	2 657 076 (100%)
Health sector									
Public	207 872 (98%)	218 911 (96%)	254 844 (96%)	292 670 (96%)	342 965 (97%)	400 826 (97%)	375 488 (97%)	371 050 (96%)	2 464 626 (97%)
Private	4 532 (2.1%)	8 233 (3.6%)	9 649 (3.6%)	11 671 (3.8%)	12 271 (3.5%)	12 316 (3%)	10 858 (2.8%)	13 581 (3.5%)	83 111 (3.3%)

[1]Multiple diagnoses possible per STI case. [2]Other diagnosis includes balanitis, genital warts, inguinal bubo, neonatal conjunctivitis, scrotal swelling, syphilis, among others.
UDS: urethral discharge syndrome, VDS: vaginal discharge syndrome, GUD: genital ulcer disease, LAP: lower abdominal pain.

Appendix E

Supplement to Chapter 6

E.1 Supplementary methods

E.1.1 Transmission model details

The modelled population was stratified by sex (male and female), sexual risk (low, intermediate, and high, with high representing female sex workers [FSWs] and clients of female sex workers [CFSWs]), age group (15-24 years [younger] and 25-49 years [older]), and gestational status (non-pregnant and pregnant), denoted as $s \in \{m, f\}$, $r \in \{l, i, h\}$, $a \in \{y, o\}$, $g \in \{n, p\}$ throughout this supplement.

Equations

For an individual of a given sex (s), sexual risk (r), age (a), and gestational status (g), the model is described by the following system of differential equations. In these equations, stratification indices are represented numerically as: $s \in \{0, 1\}$ for male and female; $r \in \{0, 1, 2\}$ for low, intermediate, and high sexual risk; $a \in \{0, 1\}$ for 15–24 and 25–49 years; and $g \in \{0, 1\}$ for non-pregnant and pregnant.

Parameters are defined in Table 6.1, with further detail on data sources and assumptions informing parameters values in Table E2. In Equation E.1, there are no terms relating to pregnancy (f_{ra} and α) as individuals have not yet become sexually active. In Equations E.6 - E.10, intervention-related parameters (δ_{srag} or τ^h_{srag}) are set to zero in scenarios where diagnostic or screening interventions are not active. This allows all compartments and transitions to be retained in the model system while isolating intervention effects analytically. Intervention scenarios are detailed in Text E.1.1.

In this chapter, $\mathbb{1}_A$ is used as an indicator function such that:

$$\mathbb{1}_A := \begin{cases} 1 & \text{if } A \text{ is true} \\ 0 & \text{if } A \text{ is false} \end{cases}$$

$$\frac{dU_{s,r,a,g}(t)}{dt} = ent_{s,r,a,g} - \psi_{s,r,a,g}\, U_{s,r,a,g} - \omega_a\, U_{s,r,a,g} + \mathbb{1}_{\{a=1\}}\, \omega_{a-1}\, U_{s,r,a-1,g} - \mu_{s,a}\, U_{s,r,a,g} \tag{E.1}$$

$$\begin{aligned} \frac{dS_{s,r,a,g}(t)}{dt} &= \psi_{s,r,a,g}\, U_{s,r,a,g} - \lambda_{s,r,a,g}\, S_{s,r,a,g} \\ &+ \pi(X_{s,r,a,g} + Y_{s,r,a,g} + Z_{s,r,a,g} + R^y_{s,r,a,g} + R^h_{s,r,a,g}) + \sigma(M^y_{s,r,a,g} + T^y_{s,r,a,g} + T^h_{s,r,a,g}) \\ &- \mathbb{1}_{\{s=1,g=0\}}\, f_{ra}\, S_{s,r,a,g} + \mathbb{1}_{\{s=1,g=1\}}\, f_{ra}\, S_{s,r,a,g-1} + \mathbb{1}_{\{s=1,g=0\}}\, \alpha\, S_{s,r,a,g+1} - \mathbb{1}_{\{s=1,g=1\}}\, \alpha\, S_{s,r,a,g} \\ &- \omega_a\, S_{s,r,a,g} + \mathbb{1}_{\{a=1\}}\, \omega_{a-1}\, S_{s,r,a-1,g} - \mu_{s,a}\, S_{s,r,a,g} \end{aligned} \tag{E.2}$$

$$\frac{dX_{s,r,a,g}(t)}{dt} = \lambda_{s,r,a,g}\ (1-\phi_s)\ S_{s,r,a,g} - \pi\ X_{s,r,a,g} - \tau^h_{s,r,a,g}\ X_{s,r,a,g}$$
$$- \mathbb{1}_{\{s=1,g=0\}}\ f_{ra}\ X_{s,r,a,g} + \mathbb{1}_{\{s=1,g=1\}}\ f_{ra}\ X_{s,r,a,g-1} + \mathbb{1}_{\{s=1,g=0\}}\ \alpha\ X_{s,r,a,g+1} - \mathbb{1}_{\{s=1,g=1\}}\ \alpha\ X_{s,r,a,g}$$
$$- \omega_a\ X_{s,r,a,g} + \mathbb{1}_{\{a=1\}}\ \omega_{a-1}\ X_{s,r,a-1,g} - \mu_{s,a}\ X_{s,r,a,g} \tag{E.3}$$

$$\frac{dY_{s,r,a,g}(t)}{dt} = \lambda_{s,r,a,g}\ \phi_s\ \gamma_{s,r,a,g}\ S_{s,r,a,g} - \pi\ Y_{s,r,a,g} - \tau^y_{s,r,a,g}\ Y_{s,r,a,g} - \tau^h_{s,r,a,g}\ Y_{s,r,a,g}$$
$$- \mathbb{1}_{\{s=1,g=0\}}\ f_{ra}\ Y_{s,r,a,g} + \mathbb{1}_{\{s=1,g=1\}}\ f_{ra}\ Y_{s,r,a,g-1} + \mathbb{1}_{\{s=1,g=0\}}\ \alpha\ Y_{s,r,a,g+1} - \mathbb{1}_{\{s=1,g=1\}}\ \alpha\ Y_{s,r,a,g}$$
$$- \omega_a\ Y_{s,r,a,g} + \mathbb{1}_{\{a=1\}}\ \omega_{a-1}\ Y_{s,r,a-1,g} - \mu_{s,a}\ Y_{s,r,a,g} \tag{E.4}$$

$$\frac{dZ_{s,r,a,g}(t)}{dt} = \lambda_{s,r,a,g}\ \phi_s\ (1-\gamma_{s,r,a,g})\ S_{s,r,a,g} - \pi\ Z_{s,r,a,g} - \tau^h_{s,r,a,g}\ Z_{s,r,a,g}$$
$$- \mathbb{1}_{\{s=1,g=0\}}\ f_{ra}\ Z_{s,r,a,g} + \mathbb{1}_{\{s=1,g=1\}}\ f_{ra}\ Z_{s,r,a,g-1} + \mathbb{1}_{\{s=1,g=0\}}\ \alpha\ Z_{s,r,a,g+1} - \mathbb{1}_{\{s=1,g=1\}}\ \alpha\ Z_{s,r,a,g}$$
$$- \omega_a\ Z_{s,r,a,g} + \mathbb{1}_{\{a=1\}}\ \omega_{a-1}\ Z_{s,r,a-1,g} - \mu_{s,a}\ Z_{s,r,a,g} \tag{E.5}$$

$$\frac{dM^y_{s,r,a,g}(t)}{dt} = \tau^y_{s,r,a,g}\ \zeta_s\ (1-\delta_{s,r,a,g})\ Y_{s,r,a,g} - \sigma\ M^y_{s,r,a,g}$$
$$- \mathbb{1}_{\{s=1,g=0\}}\ f_{ra}\ M^y_{s,r,a,g} + \mathbb{1}_{\{s=1,g=1\}}\ f_{ra}\ M^y_{s,r,a,g-1} + \mathbb{1}_{\{s=1,g=0\}}\ \alpha\ M^y_{s,r,a,g+1} - \mathbb{1}_{\{s=1,g=1\}}\ \alpha\ M^y_{s,r,a,g}$$
$$- \omega_a\ M^y_{s,r,a,g} + \mathbb{1}_{\{a=1\}}\ \omega_{a-1}\ M^y_{s,r,a-1,g} - \mu_{s,a}\ M^y_{s,r,a,g} \tag{E.6}$$

$$\frac{dT^y_{s,r,a,g}(t)}{dt} = \tau^y_{s,r,a,g}\ \eta_s\ \delta_{s,r,a,g}\ Y_{s,r,a,g} - \sigma\ T^y_{s,r,a,g}$$
$$- \mathbb{1}_{\{s=1,g=0\}}\ f_{ra}\ T^y_{s,r,a,g} + \mathbb{1}_{\{s=1,g=1\}}\ f_{ra}\ T^y_{s,r,a,g-1} + \mathbb{1}_{\{s=1,g=0\}}\ \alpha\ T^y_{s,r,a,g+1} - \mathbb{1}_{\{s=1,g=1\}}\ \alpha\ T^y_{s,r,a,g}$$
$$- \omega_a\ T^y_{s,r,a,g} + \mathbb{1}_{\{a=1\}}\ \omega_{a-1}\ T^y_{s,r,a-1,g} - \mu_{s,a}\ T^y_{s,r,a,g} \tag{E.7}$$

$$\frac{dR^y_{s,r,a,g}(t)}{dt} = \tau^y_{s,r,a,g}\ [1-\zeta_s(1-\delta_{s,r,a,g}) - \eta_s\delta_{s,r,a,g}]\ Y_{s,r,a,g} - \pi\ R^y_{s,r,a,g}$$
$$- \mathbb{1}_{\{s=1,g=0\}}\ f_{ra}\ R^y_{s,r,a,g} + \mathbb{1}_{\{s=1,g=1\}}\ f_{ra}\ R^y_{s,r,a,g-1} + \mathbb{1}_{\{s=1,g=0\}}\ \alpha\ R^y_{s,r,a,g+1} - \mathbb{1}_{\{s=1,g=1\}}\ \alpha\ R^y_{s,r,a,g}$$
$$- \omega_a\ R^y_{s,r,a,g} + \mathbb{1}_{\{a=1\}}\ \omega_{a-1}\ R^y_{s,r,a-1,g} - \mu_{s,a}\ R^y_{s,r,a,g} \tag{E.8}$$

$$\frac{dT^h_{s,r,a,g}(t)}{dt} = \tau^h_{s,r,a,g}\ \eta_s\ (X_{s,r,a,g} + Y_{s,r,a,g} + Z_{s,r,a,g}) - \sigma\ T^h_{s,r,a,g}$$
$$- \mathbb{1}_{\{s=1,g=0\}}\ f_{ra}\ T^h_{s,r,a,g} + \mathbb{1}_{\{s=1,g=1\}}\ f_{ra}\ T^h_{s,r,a,g-1} + \mathbb{1}_{\{s=1,g=0\}}\ \alpha\ T^h_{s,r,a,g+1} - \mathbb{1}_{\{s=1,g=1\}}\ \alpha\ T^h_{s,r,a,g}$$
$$- \omega_a\ T^h_{s,r,a,g} + \mathbb{1}_{\{a=1\}}\ \omega_{a-1}\ T^h_{s,r,a-1,g} - \mu_{s,a}\ T^h_{s,r,a,g} \tag{E.9}$$

$$\begin{aligned}\frac{dR^h_{s,r,a,g}(t)}{dt} &= \tau^h_{s,r,a,g}\ (1-\eta_s)\ (X_{s,r,a,g}+Y_{s,r,a,g}+Z_{s,r,a,g}) - \pi\ R^h_{s,r,a,g} \\ &- \mathbb{1}_{\{s=1,g=0\}}\ f_{ra}\ R^h_{s,r,a,g} + \mathbb{1}_{\{s=1,g=1\}}\ f_{ra}\ R^h_{s,r,a,g-1} + \mathbb{1}_{\{s=1,g=0\}}\ \alpha\ R^h_{s,r,a,g+1} - \mathbb{1}_{\{s=1,g=1\}}\ \alpha\ R^h_{s,r,a,g} \\ &- \omega_a\ R^h_{s,r,a,g} + \mathbb{1}_{\{a=1\}}\ \omega_{a-1}\ R^h_{s,r,a-1,g} - \mu_{s,a}\ R^h_{s,r,a,g}\end{aligned} \tag{E.10}$$

The total (N), sexually active ($\hat{N}$), and infected (I) population are calculated as:

$$N_{s,r,a,g} = U_{s,r,a,g} + S_{s,r,a,g} + I_{s,r,a,g} \tag{E.11}$$

$$\hat{N}_{s,r,a,g} = S_{s,r,a,g} + I_{s,r,a,g} \tag{E.12}$$

$$I_{s,r,a,g} = X_{s,r,a,g} + Y_{s,r,a,g} + Z_{s,r,a,g} + M^y_{s,r,a,g} + T^y_{s,r,a,g} + R^y_{s,r,a,g} + T^h_{s,r,a,g} + R^h_{s,r,a,g} \tag{E.13}$$

Sexual mixing

Sexual mixing was implemented as fully proportionate. The mixing matrix ($\rho_{srags'r'a'g'}$) represented the probability that an individual of type s, r, a, g selects a sexual partner of type s', r', a', g', conditional on forming a new partnership. This probability was proportional to the total number of partnerships offered by the opposite sex, as determined by their sexually active population size ($\hat{N}_{s'r'a'g'}$) and their average annual rate of partner acquisition ($c_{s'r'a'g'}$). The mixing matrix also incorporated structural constraints in the sexual network ($\theta_{srags'r'a'g'}$): (1) partnerships were only possible between individuals of the opposite sex, and (2) FSW formed partnerships exclusively with their clients (high-risk males), while clients could form partnerships with females from all risk groups.

$$\rho_{srags'r'a'g'} = \frac{\theta_{srags'r'a'g'}\ c_{s'r'a'g'}\ \hat{N}_{s'r'a'g'}}{\sum^h_{r''=l}\sum^o_{a''=y}\sum^p_{g''=n}\theta_{srags''r''a''g''}\ c_{s''r''a''g''}\ \hat{N}_{s''r''a''g''}} \tag{E.14}$$

$$\theta_{srags'r'a'g'} = \begin{cases} 0 & \text{Same-sex partnerships} \\ & s = s' \\ 0 & \text{FSW and non-client partnerships} \\ & s = f,\ r = h,\ s' = m,\ r' \in \{l, i\} \\ & s = m,\ r \in \{l, i\},\ s' = f,\ r' = h \\ 1 & \text{Other partnerships} \end{cases} \tag{E.15}$$

To ensure consistency in the number of partnerships formed between sexes, a balancing procedure was applied to the annual rates of partner acquisition.[897] This adjustment was necessary because reported sexual partner numbers often differ between men and women. The balancing factor ($B_{srags'r'a'g'}$) quantified the degree of imbalance in the total number of partnerships formed by individuals of type s, r, a, g with those of type $s'r'a'g$:

$$B_{srags'r'a'g'} = \frac{c_{srag}\ \rho_{srags'r'a'g'}\ \hat{N}_{srag}}{c_{s'r'a'g'}\ \rho_{s'r'a'g'srag}\ \hat{N}_{s'r'a'g'}} \tag{E.16}$$

A value of $B_{srags'r'a'g'} = 1$ indicated perfect balance, while values greater or less than 1 indicated that individuals of sex s are forming more or fewer partnerships with individuals of sex s', respectively. To preserve relative differences in partner acquisition across groups while ensuring symmetry in the total number of partnerships formed between any two strata, the initial rates were adjusted as follows:

$$c^*_{srags'r'a'g'} = c_{srag}\ B^{-0.5}_{srags'r'a'g'} \tag{E.17}$$

$$c^*_{s'r'a'g'srag} = c_{s'r'a'g'}\ B^{0.5}_{srags'r'a'g'} \tag{E.18}$$

The adjusted rate ($c^*_{srags'r'a'g'}$) represented the effective annual rate at which individuals of type s, r, a, g acquired new partners of type s', r', a', g', after accounting for imbalances in partnership numbers between sexes. The product $c^*_{srags'r'a'g'}\rho_{srags'r'a'g'}$ therefore reflected the annual number of new partners of type s', r', a', g' acquired by individuals of type s, r, a, g.

Force of infection

The force of infection (λ_{srag}) determined the rate per year at which a susceptible individual of type s, r, a, g acquired infection through the formation of new sexual partnerships. This depended on the per-partnership probability of transmission ($\kappa^p_{srags'r'a'g'}$), the adjusted rate of acquiring new partners ($c^*_{srags'r'a'g'}$), the probability of selecting such a partner conditional on partnership formation ($\rho_{srags'r'a'g'}$), and the prevalence of infection among potential partners ($I_{s'r'a'g'}/\hat{N}_{s'r'a'g'}$):

$$\lambda_{srag} = \sum_{r'=l}^{h} \sum_{a'=y}^{o} \sum_{g'=n}^{p} \kappa^p_{srags'r'a'g'}\ c^*_{srags'r'a'g'}\ \rho_{srags'r'a'g'}\ \frac{I_{s'r'a'g'}}{\hat{N}_{s'r'a'g'}} \tag{E.19}$$

The per-partnership transmission probability ($\kappa^p_{srags'r'a'g'}$) was determined based on the transmission probability per sex act (κ^a_s), the number of sex acts per partnership ($n^p_{srags'r'a'g'}$), the proportion of sex acts within the partnership where a condom is used ($\chi^p_{srags'r'a'g'}$), and the efficacy of condoms in

preventing transmission (e). The formulation considered the probability of avoiding infection across all sex acts within the partnership, accounting separately for acts with and without condom use:

$$\kappa^{p}_{srags'r'a'g'} = 1 \; - \; (1-(1-e)\kappa^{a}_{s})^{n^{p}_{srags'r'a'g'}\chi^{p}_{srags'r'a'g'}} \; (1-\kappa^{a}_{s})^{n^{p}_{srags'r'a'g'}(1-\chi^{p}_{srags'r'a'g'})} \tag{E.20}$$

Since data were lacking to quantify the number of sex acts within each partnership type, it was assumed that the annual number of sex acts per individual (n) was the same across all strata, and that sex acts were distributed equally across partners. FSW and their clients were assumed to have only one sex act per partnership. The number of sex acts per partnership was therefore defined as:

$$n^{p}_{srags'r'a'g'} = \begin{cases} 1 & \text{FSW and client partnerships} \\ \frac{n}{c^{*}_{srags'r'a'g'}\rho_{srags'r'a'g'}} & \text{Other partnerships} \end{cases} \tag{E.21}$$

To ensure consistency in condom use assumptions across partner types, the proportion of sex acts within each partnership that used a condom ($\chi^{p}_{srags'r'a'g'}$) was calculated by equally weighting each partner's reported condom use at last sex:

$$\chi^{p}_{srags'r'a'g'} = (\chi_{srag} + \chi_{s'r'a'g'})^{0.5} \tag{E.22}$$

Intervention scenarios

Point-of-care testing for gonorrhoea was implemented from 2025 to 2030. Tests were allocated to one of five target population groups: pregnant women (all ages and risk groups); sexually active adolescent girls and young women (AGYW; non-pregnant 15-24-year-old females who had ever had sex); FSW (all ages and gestational status); the general male population (all ages and risk groups); and CFSW (high-risk males of all ages). For each target population, tests were used as either a diagnostic or screening intervention.

Diagnostic testing: In the diagnostic testing scenario, POCTs were implemented among individuals accessing care for urethral discharge and vaginal discharge syndromes. The eligible population was determined as the annual number of symptomatic cases caused by gonorrhoea (NG-case$_{srag}$) and other reproductive tract infections (RTI-case$_{srag}$), such that the same individual could be tested multiple times if they experienced repeat infections.

The number of symptomatic gonorrhoea cases accessing treatment per year (y) was defined as the total

number of individuals leaving the Y compartment, where Y_{srag} represented symptomatic gonorrhoea infections in individuals planning to access treatment. This was calculated as:

$$\text{NG-cases}_{srag}(y) = \int_{t=y}^{y+1} \tau_{srag}^{y}\, Y_{srag}(t)\, dt \tag{E.23}$$

Annual symptomatic cases due to other RTIs were calculated using the gonorrhoea aetiologic proportion (p_s), defined as the proportion of individuals with a given symptom who have gonorrhoea as the underlying cause. This allowed estimation of total symptomatic cases attributable to causes other than gonorrhoea by scaling observed symptomatic gonorrhoea cases accordingly. These RTI case numbers were then fixed at pre-intervention levels to ensure they remained independent of changes in gonorrhoea incidence during intervention simulations:

$$\text{RTI-case}_{srag}(y) = \begin{cases} \text{NG-case}_{srag}(y)\ \frac{(1-p_s)}{p_s} & y < 2025 \\ \text{NG-case}_{srag}(y)\ \frac{(1-p_s)}{p_s}\big|_{y=2024} & y \geq 2025 \end{cases} \tag{E.24}$$

The annual probability of receiving a diagnostic test within a selected target population (δ_T) was calculated as the ratio of available tests to the total number of eligible symptomatic cases in that population. Each target population T comprised a subset of strata defined by sex, risk, age, and gestational status, such that $(srag) \in T$ denoted the included combinations. A maximum probability of 1 was imposed to ensure that testing coverage did not exceed the number of eligible individuals:

$$\delta_T = \min\left(1, \frac{\text{annual tests available for } T}{\sum_{(srag)\in T} \text{NG-case}_{srag}(y) + \text{RTI-case}_{srag}(y)}\right) \tag{E.25}$$

Screening: In the screening scenario, POCTs were implemented among individuals attending either primary healthcare or antenatal care services. The eligible population was defined as the number of individuals accessing these health services annually. Since individuals may attend care more than once per year, the annual probability of healthcare attendance (hc_{srag}) was converted to a rate, assuming visits follow a Poisson process with exponential spacing over time. The effective number of individuals attending healthcare services annually and eligible for screening was calculated as:

$$N_{srag}^{\text{h}} = -\log(1 - \text{hc}_{srag})(U_{srag} + S_{srag} + X_{srag} + Y_{srag} + Z_{srag}) \tag{E.26}$$

For each targeted population T, the annual screening rate (τ_T^{h}) was calculated as the ratio of available

tests to the total number of eligible individuals in the target population:

$$\tau_T^{\mathrm{h}} = \frac{\text{annual tests available for } T}{\sum_{(srag)\in T} N_{srag}^{\mathrm{h}}} \tag{E.27}$$

The same annual screening rate was applied to each stratum $srag \in T$.

E.1.2 Transmission model parametrisation

Demographic parameters

Population size was derived using age- and sex-stratified demographic estimates for the 15-49-year-old Kenyan population between 1970 and 2030 from UN World Population Prospects (WPP) 2024.[287] The female population was further stratified by gestational status. The number of pregnant women in year y was estimated according to the number of births by maternal age group in the following year (B_{fa}) and the annual rate of delivery (α), corresponding to a mean gestational period of 9 months (i.e. $1/\alpha = 0.75$ year):

$$N_{fap}(y) = \alpha B_{fa}(y+1) \tag{E.28}$$

Annual pregnancy rates for females were then calculated per age group as the ratio of pregnant to non-pregnant women in that group, scaled by α to account for pregnancy duration:

$$f_a = \alpha \frac{N_{fap}}{N_{fan}} \tag{E.29}$$

To estimate annual pregnancy rates among sexually active females, population-level rates were adjusted by the proportion sexually active in each non-pregnant female stratum (q_{fran}), which varied by risk group, such that:

$$f_{ra}^{model} = \frac{f_a}{q_{fran}} \tag{E.30}$$

This adjustment ensured that the total number of pregnancies was preserved. If all non-pregnant females in a stratum were sexually active ($q_{fran} = 1$), no adjustment was needed. If only a subset were sexually active ($q_{fran} < 1$), the per-capita pregnancy rate among those individuals was increased proportionally.

Each sex-age-gestational stratum was divided into sexual risk groups according to the sex-specific

probability of belonging to a risk group (υ_{sr}):

$$N_{srag}(y) = \upsilon_{sr} N_{sag}(y) \tag{E.31}$$

New male and female entrants to the population were aged 15-24 years and allocated to the U compartment. The annual number of entrants was calculated using sex-stratified births 15 years prior (B_s) and deaths in corresponding sex and age cohorts (D_s^{age}) at the midpoint of exposure.[287] All entrants were non-pregnant and were stratified by risk group using the same method as for the total population.

$$ent_{srag}(y) = \mathbb{1}_{\{a=15-24, g=n\}}\, \upsilon_{sr} \left[B_s(y-15) - D_s^{0-4}(y-13) - D_s^{5-9}(y-8) - D_s^{10-14}(y-3)\right] \tag{E.32}$$

Values were calculated for 1970 and 2030, with intermediate years interpolated linearly.

Individuals aged from 15–24 to 25–49 years and exited the 25-49 year-old group at the annual rate ω_a. Sex and risk group were assumed to remain fixed over time, while gestational status changed dynamically for females based on the age- and risk-specific pregnancy rates described above.

Mortality rates (μ_{sa}) from all causes were calculated by dividing total deaths by population size per sex and age in 1970 and 2030, with values interpolated linearly for intermediate years.[287]

Sexual behaviour, diagnosis, and treatment parameters

Parameter values related to sexual behaviour and care-seeking for symptoms were derived from Kenya DHS (KDHS) in 2003, 2008-2009, 2014 and 2022,[372–375] and Kenya Population-based HIV Impact Assessment (KenPHIA) in 2018.[376] For the parameters listed below, values were primarily estimated by pooling KDHS and summarising data as survey-weighted proportions with design-based standard errors and 95% CIs. KenPHIA covered fewer relevant indicators; data were extracted from the published report as supplementary input. Variables included:

- Percentage of males who had ever paid for sex.[372,374]
- Percentage of adults who had ever had sex, stratified by sex and age group.[372–375]
- Number of sexual partners reported among sexually active adults in the past year, stratified by sex, age group, and gestational status.[372–375]
- Percentage of sexually active adults reporting condom use at last sex, stratified by sex, age group,[372–376] gestational status,[372–375] marital status,[372–376] and higher-risk sex.[372–375]

- Percentage of sexually active adults with genital discharge who reported accessing care in the past year, stratified by sex, age group, and pregnancy status.[372–375]
- Percentage of women who attended ANC at least once for their most recent pregnancy.[372–375]

Table E1: Demographic parameter values

Stratum	Population		Entrants		Pregnancy rate		Mortality rate	
	1970	2030	1970	2030	1970	2030	1970	2030
m.l.y.n	730 072	4 089 571	96 749	485 610	0	0	0.0042	0.0020
m.l.y.p	0	0	0	0	0	0	0	0
m.l.o.n	729 196	6 017 108	0	0	0	0	0.0073	0.0060
m.l.o.p	0	0	0	0	0	0	0	0
m.i.y.n	157 253	880 867	20 839	104 597	0	0	0.0042	0.0020
m.i.y.p	0	0	0	0	0	0	0	0
m.i.o.n	157 064	1 296 046	0	0	0	0	0.0073	0.0060
m.i.o.p	0	0	0	0	0	0	0	0
m.h.y.n	154 455	865 197	20 468	102 736	0	0	0.0042	0.0020
m.h.y.p	0	0	0	0	0	0	0	0
m.h.o.n	154 270	1 272 990	0	0	0	0	0.0073	0.0060
m.h.o.p	0	0	0	0	0	0	0	0
f.l.y.n	607 808	3 870 978	101 486	497 481	0.49	0.19	0.0036	0.0015
f.l.y.p	132 960	339 634	0	0	0	0	0.0036	0.0015
f.l.o.n	735 096	5 828 462	0	0	0.32	0.11	0.0062	0.0043
f.l.o.p	177 531	487 227	0	0	0	0	0.0062	0.0043
f.i.y.n	208 485	1 332 056	34 811	170 642	0.45	0.17	0.0036	0.0015
f.i.y.p	45 607	112 233	0	0	0	0	0.0036	0.0015
f.i.o.n	252 147	1 999 513	0	0	0.32	0.11	0.0062	0.0043
f.i.o.p	60 895	166 843	0	0	0	0	0.0062	0.0043
f.h.y.n	16 827	107 853	2 810	13 773	0.41	0.16	0.0036	0.0015
f.h.y.p	3 681	8 717	0	0	0	0	0.0036	0.0015
f.h.o.n	20 351	161 406	0	0	0.32	0.11	0.0062	0.0043
f.h.o.p	4 915	13 443	0	0	0	0	0.0062	0.0043

Parameters derived using UN WPP 2024 estimates.[287] Values shown use median posterior estimates for the probability of being in a sexual risk group (υ_{sr}) and the probability of being sexually active (q_{srag}).

Table E2: Detailed parameter definitions, rationale, and assumptions for gonorrhoea transmission model

Description	Symbol	Prior or value	Evidence and rationale
Demography			
Initial population size in 1970	N^0_{srag}	Fixed: Table E1	Derived from UN WPP 2024 (Text E.1.2).[287]
Annual entrants into population	ent_{srag}	Fixed: Table E1	Annual entrants into 15-24-year-old non-pregnant population, allocated to U compartment. Derived from UN WPP 2024 (Text E.1.2).[287]
Probability of being in sexual risk group			
Male, low risk	υ_{ml}	Calculated	$1 - \upsilon_{mi} - \upsilon_{mh}$
Male, intermediate risk	υ_{mi}	U(0.10, 0.20)	Assumption for intermediate risk such that both sexes have low risk probability: 0.65 - 0.80.
Male, high risk (CFSW)	υ_{mh}	U(0.10, 0.20)	Percentage of males who had ever paid for sex: 8.6-12.1% (KDHS).[372,374]
Female, low risk	υ_{fl}	Calculated	$1 - \upsilon_{fi} - \upsilon_{fh}$
Female, intermediate risk	υ_{fi}	U(0.20, 0.30)	Assumption for intermediate risk such that both sexes have low risk probability: 0.65 - 0.80.
Female, high risk (FSW)	υ_{fh}	U(0.01, 0.03)	Meta-analysis on Kenya's FSW population size: 1.62% (95% CI: 0.86-2.94%).[377]
Mean duration in age group (year)			
15-24 y	$1/\omega_y$	Fixed: 10	-
25-49 y	$1/\omega_o$	Fixed: 25	-
Rate of becoming pregnant among sexually active non-pregnant females ($year^{-1}$)	f_{ra}	Fixed: Table E1	Derived from UN WPP 2024 (Text E.1.2).[287]
Mean duration of pregnancy (year)	$1/\alpha$	Fixed: 0.75	-
Rate of mortality ($year^{-1}$)	μ_{sa}	Fixed: Table E1	Derived from UN WPP 2024 (Text E.1.2).[287]
Sexual behaviour			
Probability of being sexually active			
All sex, low risk, 15-24 y, non-pregnant	q_{slyn}	U(0.50, 0.70)	Percentage of 15-24-year-olds reporting sexual debut: 61-63% for males, 61-62% for females (KDHS).[372–375]
All sex, all risk, 25-49 y, non-pregnant	q_{sron}	Fixed: 1	Percentage of 25-49-year-olds reporting sexual debut: >98% for both sexes (KDHS).[372–375]
Female, all risk, all age, pregnant	q_{frap}	Fixed: 1	-
Relative probability of being sexually active among all sex, 15-24 y, non-pregnant			

Continued...

Description	Symbol	Prior or value	Evidence and rationale
Intermediate:low risk	$rq_{i:l}$	U(1.05, 1.15)	Assumption
High:intermediate risk	$rq_{h:i}$	U(1.05, 1.15)	Assumption
Rate of becoming sexually active (year^{-1})	ψ_{srag}	Calculated	-$log(1 - q_{srag})$
Number of partnerships per person (year^{-1})			
All sex, low risk, 15-24 y, non-pregnant	c_{slyn}	U(1.00, 1.50)	Number of sexual partners reported among sexually active 15-24-year-olds in the past year: 1.24-1.29 for males, 1.04-1.05 for females (KDHS).[372–375]
Relative number of partnerships per person			
Male, intermediate:low risk	$rc_{mi:ml}$	U(3.00, 6.00)	Assumption
Male, high:intermediate risk	$rc_{mh:mi}$	U(2.50, 5.00)	Assumption based on CFSW and FSW population sizes, and number of clients reported by FSW.
Female, intermediate:low risk	$rc_{fi:fl}$	U(3.00, 6.00)	Assumption
Female, high:intermediate risk	$rc_{fh:fi}$	U(10.0, 25.0)	Number of clients reported by FSW: 1-7 week^{-1} (∼50-360 year^{-1}).[378–391]
25-49:15-24 y	$rc_{o:y}$	U(0.85, 1.00)	Number of sexual partners reported among sexually active individuals in the past year: 1.08-1.11 for 25-49-year-olds, 1.12-1.14 for 15-24-year-olds (KDHS).[372–375]
Pregnant:non-pregnant	$rc_{p:n}$	U(0.85, 1.00)	Number of sexual partners reported among sexually active 15-49-year-old females in the past year: 1.01-1.02 for pregnant, 1.03-1.04 for non-pregnant (KDHS).[372–375]
Number of sex acts per person (year^{-1})	n	U(52.0, 104.0)	Assumption that applies to all strata except FSW/CFSW, where one sex act is assumed per partnership.
Probability of condom use per sex act			
All sex, low risk, 15-24 y, non-pregnant	χ_{slyn}	U(0.25, 0.35)	Percentage of sexually active 15-24-year-olds reporting condom use at last sex: 57-61% for males, 18-20% for females (KDHS).[372–375]
Relative probability of condom use per sex act			
Intermediate:low risk	$r\chi_{i:l}$	U(2.30, 2.50)	Percentage of individuals who had sex with a non-marital partner reporting condom use at last sex with that partner: ∼50-70% (KDHS and KenPHIA).[372–376]
High:intermediate risk	$r\chi_{h:i}$	U(1.00, 1.15)	Percentage of FSW and CFSW reporting condom use at last sex, in last week, or with other measures of consistency: ∼60-80%.[379–383,385,387–392]

Continued...

Description	Symbol	Prior or value	Evidence and rationale
25-49:15-24 y	$r\chi_{o:y}$	U(0.85, 1.00)	Percentage of sexually active individuals reporting condom use at last sex: 17-19% among 25-49-year-old and 57-61% among 15-24-year-old males, 8-9% among 25-49-year-old and 18-20% among 15-24-year-old females (KDHS).[372–375]
Pregnant:non-pregnant, low risk	$r\chi_{lp:ln}$	U(0.10, 0.30)	Percentage of sexually active females reporting condom use at last sex: 2-4% among pregnant, 11-13% among non-pregnant (KDHS).[372–375]
Pregnant:non-pregnant, intermediate risk	$r\chi_{ip:in}$	U(0.10, 0.30)	Assumption that equivalent to low risk population.
Pregnant:non-pregnant, high risk	$r\chi_{hp:hn}$	U(0.85, 1.00)	Assumption that condom use among pregnant and non-pregnant FSW is more similar than that of lower and intermediate risk females.
Efficacy of condoms per sex act	e	U(0.90, 1.00)	Poor quality evidence for association between condom use and reduction in risk of gonorrhoea.[98] Modelling studies assume >90% efficacy.[15,274,393]
Diagnosis and treatment			
Probability symptomatic individuals access care			
Male, low risk, 15-24 y, non-pregnant	γ_{mlyn}	U(0.65, 0.75)	Percentage of 15-24-year-old sexually active males with genital discharge who reported accessing treatment in the past year: 64-86% (KDHS).[372–375]
Relative probability symptomatics access care			
Female:male	$r\gamma_{f:m}$	U(0.70, 0.90)	Percentage of 15-49-year-old sexually active individuals with genital discharge who reported accessing treatment in the past year: 70-76% for females, 76-89% for males (KDHS).[372–375]
Intermediate:low risk	$r\gamma_{i:l}$	U(1.00, 1.10)	Assumption
High:intermediate risk	$r\gamma_{h:i}$	U(1.00, 1.10)	Percentage of FSW with symptoms who reported accessing treatment in the past year: 60-90%.[389–391,394,395] Percentage of sexually active males who had ever paid for sex with genital discharge, who reported accessing treatment in the past year: 60-96% (KDHS).[373,374]
25-49 y:15-24 y	$r\gamma_{o:y}$	U(1.00, 1.10)	Percentage of sexually active individuals with genital discharge who reported accessing treatment in the past year: 79-93% for 25-49-year-old and 64-86% for 15-24-year-old males, 74-81% for 25-49-year-old and 54-67% for 15-24-year-old females (KDHS).[372–375]
Pregnant:non-pregnant	$r\gamma_{p:n}$	U(1.00, 1.10)	Percentage of sexually active 15-49-year-old females with genital discharge who reported accessing treatment in the past year: 67-86% for pregnant, 69-76% for non-pregnant (KDHS).[372–375]

Continued...

Description	Symbol	Prior or value	Evidence and rationale
Rate of treatment among symptomatic males (year^{-1})	τ^{y}_{m}	U(20.0, 40.0)	Duration between infection and treatment incorporates incubation period and time to access care. Incubation period: 7-10 days.[10,12,26,27,32,61] Time to seek care among males once symptomatic: 3-8 days.[188,198,396–398]
Relative rate of treatment among symptomatics			
Female:male	$r\tau^{y}_{f:m}$	U(0.50, 1.00)	Duration between infection and treatment incorporates incubation period and time to access care. Incubation period: 7-10 days.[10,12,26,27,32,61] Time to seek care once symptomatic: 7-15 days for females, 3-8 days for males.[188,198,396–398]
Probability of POCT diagnosis among symptomatic	δ_{srag}	Fixed: scenario specific	Calculation detailed in Text E.1.1
Probability of primary healthcare access among all risk, 15-24 y, non-pregnant			
Male	h_{mryn}	Fixed: 0.70	Range based on literature review of primary healthcare service coverage and the unmet need for primary healthcare.[399–403]
Female	h_{fryn}	Fixed: 0.75	Range based on literature review of primary healthcare service coverage and the unmet need for primary healthcare.[399–403]
Relative probability of primary healthcare access among all sex and risk			
25-49 y:15-24 y	$rh_{o:y}$	Fixed: 1.05	Range based on literature review of unmet need for primary healthcare by age group.[403]
Pregnant:non-pregnant	$rh_{p:n}$	Fixed: 1.20	Percentage of 15-49-year-old pregnant women attending ANC at least once: 90-95% in general population,[372–375] 80-90% among FSW in other SSA countries.[404,405]
Rate of screening with POCT (year^{-1})	τ^{h}_{srag}	Fixed: scenario specific	Calculation detailed in Text E.1.1
Sensitivity of syndromic management			
Male	ζ_{m}	U(0.80, 0.90)	Meta-analysis on sensitivity of syndromic management for gonorrhoea and/or chlamydia diagnosis among males with urethral discharge syndrome: 85.2% (95% CI: 80.5-89.0).[125]
Female	ζ_{f}	U(0.40, 0.50)	Meta-analysis on sensitivity of syndromic management including speculum examination for gonorrhoea and/or chlamydia diagnosis among females with vaginal discharge syndrome: 44.9% (95% CI: 42.2-47.4).[116,120]
Sensitivity of POCT			

Continued...

Description	Symbol	Prior or value	Evidence and rationale
Male	η_m	Fixed: 0.95	WHO optimal target product profile for NG POCT.[175]
Female	η_f	Fixed: 0.95	WHO optimal target product profile for NG POCT.[175]
Natural history			
Probability of transmission per unprotected sex act			
Female-to-male	κ_m	U(0.15, 0.30)	Range based on literature review.[7–9,11–13]
Male-to-female	κ_f	U(0.10, 0.40)	Range based on literature review.[7,10–13]
Probability infection is symptomatic			
Male	ϕ_m	U(0.45, 0.90)	Range based on literature review.[7,11,12,30–32]
Female	ϕ_f	U(0.20, 0.45)	Range based on literature review.[7,11,12,30–33]
Rate of natural recovery (year^{-1})	π	U(1.80, 2.80)	Duration of untreated infection: 20-24 weeks.[7,12,14,32,40,41]
Rate of recovery with treatment (year^{-1})	σ	U(26.0, 52.0)	Duration of recovery after treatment: approximately 1 week.[117]
Probability syndrome is caused by gonorrhoea			
Male (urethral discharge)	p_m	U(0.65, 0.8)	Meta-analysis on proportion of urethral discharge cases due to gonorrhoea: 77.1% (95% CI: 68.1-84.1).[321]
Female (vaginal discharge)	p_f	U(0.10, 0.20)	Meta-analysis on proportion of vaginal discharge cases due to gonorrhoea: 6.6% (95% CI: 3.3-12.7), with range increased during calibration.[321]

Row shading indicates fixed parameters (light blue) and estimated parameters (white). CFSW: clients of female sex workers, FSW: female sex workers. U: Uniform distribution (min, max), y = years.

E.1.3 Transmission model calibration

Calibration targets

Calibration targets included: (i) gonorrhoea prevalence among sexually active females in the general population, (ii) gonorrhoea prevalence among FSW, (iii) male-to-female ratio for gonorrhoea prevalence, and (iv) male-to-female ratio for all-cause symptomatic case rates (Table E3).

Table E3: Calibration targets

Variable	Target	Estimate (95%CI) informing target	Source for estimate
Gonorrhoea prevalence			
Female, overall	U(1.0%, 3.5%)	2.2% (1.3 - 3.5%)	Meta-regression estimate in 2020 (Figure E1)
Female sex workers	U(2.0%, 6.5%)	3.7% (2.1 - 6.4%)	Meta-regression estimate in 2020 (Figure E1)
Male-to-female ratios			
Prevalence	U(0.60, 1.00)	0.81 (0.61 - 1.09)	Meta-regression estimate in 2020 (Chapter 2)
Symptomatic case rate	U(0.35, 0.60)	0.46 (0.36 - 0.58)	Analysis of Kenya DHS[372–374]

Symptomatic case rate: annual number of symptomatic urethral or vaginal discharge cases due to any cause, divided by the sexually active population size.

Gonorrhoea prevalence was estimated through systematic data collation and meta-regression. For the general population, prevalence observations (n = 24) with diagnostic test performance adjustment from Kenyan studies between 2000 and 2024 were extracted from the systematic review in Chapter 2. These studies primarily involved sexually active populations. For FSW, an additional PubMed search was conducted using the same inclusion criteria as Chapter 2, with an added search domain related to sex work (i.e. “sex work”, “sex worker”, “prostitute”, “commercial sex”). After de-duplication of identified articles, prevalence observations (n = 12) were extracted and adjusted for diagnostic test performance using the same approach as Chapter 2 (Table E4).

A generalised linear mixed-effects meta-regression model was then fit to the collated data. Fixed effects included year (midpoint of data collection), population group (general population, FSW), sex (female, male), age group (younger adults 12–25 years, all adults $\geq$ 12 years), and HIV status (HIV negative, non-stratified status). Interaction terms between year and sex, and year and population group, were not included due to limited observations. A study-level random intercept accounted for between-study heterogeneity. Model predictions were used to estimate prevalence for adults in the general and FSW populations, without stratifying by HIV status (Figure E1). Predictions were not stratified by age group, due to limited observations among younger adults (n = 6 general population, n = 0 FSW).

Due to limited prevalence observations, male prevalence was not directly used as a calibration target. Instead, calibration targets included the ratio of male-to-female gonorrhoea prevalence and male-to-female all-cause symptomatic case rates.

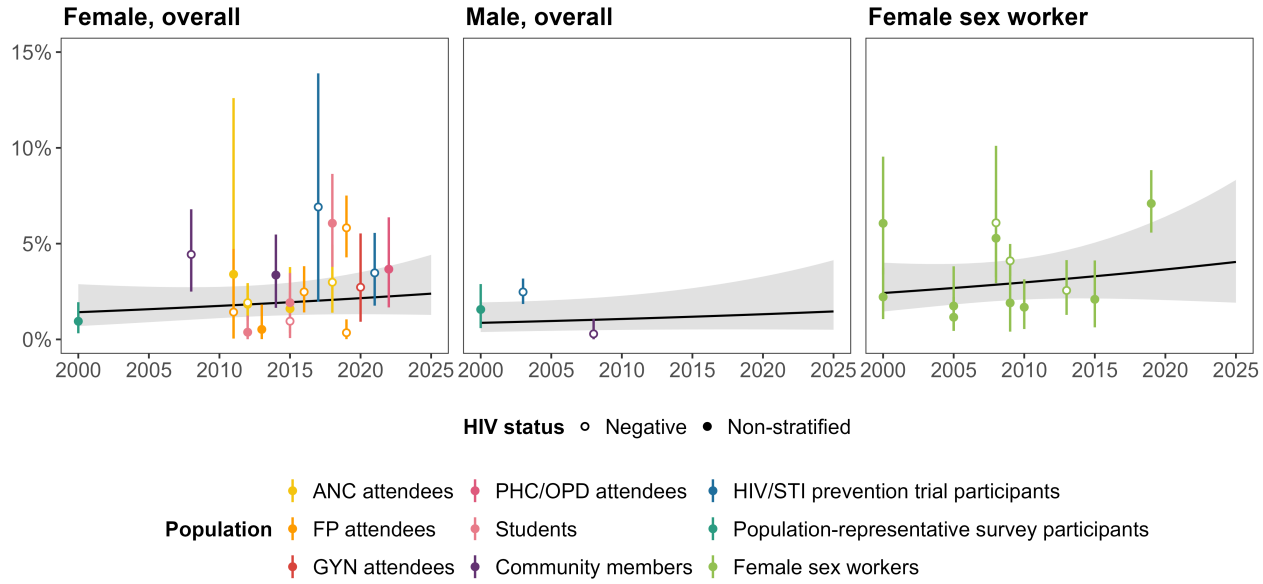


Figure E1: Estimated prevalence of gonorrhoea among the general population and female sex workers in Kenya, 2000-2025

Gonorrhoea prevalence among male and female general populations and FSW between 2000-2025. Lines and shading depict mean estimates with 95% CIs. Points represent study observations. ANC: antenatal care, FP: family planning, GYN: gyneacology, PHC/OPD: primary healthcare or outpatient department.

Table E4: Study observations of gonorrhoea prevalence in Kenya

Sex	Population	Year	Setting	Age	HIV status	Samp	Test	Sens (%)	Spec (%)	N	Prevalence Raw (%)	Prevalence Adjusted (%, 95%CI)	Reference
Population category: General													
F	Population-representative survey participants	2000	Mombasa	15 to 49	Non-stratified	UR	NAAT	91.6	100.0	571	0.7	0.9 (0.3-1.9)	Hawken 2002[538]
F	Community members	2008	Kisumu	18 to 34	Negative	GE	NAAT	93.3	99.2	424	4.7	4.4 (2.5-6.8)	Otieno 2015[564]
F	ANC attendees	2011	Mombasa	18 to 35	Non-stratified	GE	NAAT	93.3	99.2	30	0.0	3.4 (0.1-12.6)	Jespers 2014[572]
F	FP attendees	2011	Mombasa	18 to 35	Negative	GE	NAAT	93.3	99.2	110	0.9	1.4 (0.0-4.7)	Jespers 2014[572]
F	ANC attendees	2012	Western Region	14+	Negative	GE	NAAT	93.3	99.2	1276	2.5	1.9 (1.1-2.9)	Kinuthia 2015[579]
F	Students	2012	Western Region	14 to 17	Non-stratified	GE	NAAT	93.3	99.2	511	0.6	0.4 (0.0-1.2)	Kerubo 2016[578]
F	FP attendees	2013	Nairobi	20 to 49	Non-stratified	GE	Culture	75.7	100.0	249	0.0	0.5 (0.0-1.8)	Maina 2016[585]
F	Community members	2014	Kisumu	18 to 34	Non-stratified	GE	NAAT	93.3	99.2	457	3.7	3.4 (1.6-5.5)	Oliver 2018[591]
F	ANC attendees	2015	Kilifi	18 to 45	Non-stratified	UR	NAAT	91.6	100.0	202	1.0	1.6 (0.3-3.8)	Masha 2017[599]
F	Students	2015	Thika	16 to 20	Negative	GE	NAAT	93.3	99.2	373	1.3	1.0 (0.1-2.5)	Yuh 2020[600]
F	FP attendees	2016	Kisumu	16 to 35	Negative	GE	NAAT	93.3	99.2	901	3.0	2.5 (1.4-3.8)	Deese 2021[604]
F	Prevention trial participants	2017	Kisumu	18 to 50	Negative	GE/ UR	NAAT	91.6	99.2	82	6.1	6.9 (2.0-13.9)	Mgodi 2021[605]
F	ANC attendees	2018	NR	NR	Negative	GE	NAAT	93.3	99.2	474	3.4	3.0 (1.4-4.9)	Madanitsa 2023[610]
F	Students	2018	Siaya	14 to 22	Non-stratified	GE	NAAT	93.3	99.2	436	6.2	6.1 (3.8-8.6)	Mehta 2023[611]
F	FP attendees	2019	Nairobi, Mombasa	18 to 45	Negative	GE	NAAT	93.3	99.2	691	0.7	0.4 (0.0-1.0)	Lokken 2022[613]
F	FP attendees	2019	Kisumu	16 to 25	Negative	GE	NAAT	93.3	99.2	1000	6.1	5.8 (4.3-7.5)	Celum 2022[612]
F	GYN attendees	2020	Thika, Kisumu	15 to 30	Negative	UR	NAAT	91.6	100.0	198	2.0	2.7 (0.9-5.5)	Heffron 2021[618]

Continued...

Sex	Population	Year	Setting	Age	HIV status	Samp	Test	Sens (%)	Spec (%)	N	Prevalence Raw (%)	Prevalence Adjusted (%, 95%CI)	Reference
F	Prevention trial participants	2021	Kisumu	18 to 30	Negative	GE	NAAT	93.3	99.2	448	3.8	3.5 (1.8-5.6)	Oware 2023[622]
F	OPD attendees	2022	Kitui	15 to 44	Non-stratified	GE	Culture	75.7	100.0	322	2.5	3.7 (1.7-6.4)	Mbuvi 2024[625]
M	Population-representative survey participants	2000	Mombasa	15 to 49	Non-stratified	UR	NAAT	80.9	99.9	583	1.2	1.6 (0.6-2.9)	Hawken 2002[538]
M	Prevention trial participants	2003	Kisumu	18 to 24	Negative	UR	NAAT	80.9	99.9	2754	2.1	2.5 (1.8-3.2)	Bailey 2007[546]
M	Community members	2008	Kisumu	18 to 34	Negative	UR	NAAT	80.9	99.9	422	0.0	0.3 (0.0-1.1)	Otieno 2015[564]
Population category: Female sex workers													
F	Female sex workers	2000	Nairobi	18 to 35	Non-stratified	GE	NAAT	93.3	99.2	295	6.1	6.1 (3.4-9.3)	Cohen 2007[898]
F	Female sex workers	2000	Mombasa	15 to 65	Non-stratified	UR	NAAT	91.6	100.0	493	1.8	2.2 (1.1-3.8)	Hawken 2002[380]
F	Female sex workers	2005	Kisumu	NR	Non-stratified	UR	NAAT	91.6	100.0	250	1.2	1.7 (0.4-3.8)	Kwena 2010[899]
F	Female sex workers	2005	Mombasa	16+	Non-stratified	GE	Culture	75.7	100.0	677	0.7	1.2 (0.5-2.3)	Chersich 2007[900]
F	Female sex workers	2008	Nairobi	18 to 55	Negative	GE	NAAT	93.3	99.2	200	6.0	6.1 (2.8-10.3)	Priddy 2011[382]
F	Female sex workers	2008	Kisumu	NR	Non-stratified	GE	NAAT	93.3	99.2	474	5.5	5.3 (3.2-7.8)	Vandenhoudt 2013[383]
F	Female sex workers	2009	Nairobi	18 to 49	Non-stratified	GE	NAAT	93.3	99.2	348	2.3	1.9 (0.4-3.9)	Gomih-Alakija 2014[384]
F	Female sex workers	2009	Nairobi	NR	Negative	GE	Culture	75.7	100.0	2931	3.1	4.1 (3.3-5.0)	Preston 2013[901]
F	Female sex workers	2010	Nairobi	18+	Non-stratified	GE	NAAT	93.3	99.2	596	2.2	1.7 (0.5-3.1)	Musyoki 2015[385]
F	Female sex workers	2013	Mombasa	NR	Negative	GE	NAAT	93.3	99.2	660	3.0	2.6 (1.3-4.1)	Willcox 2021[387]
F	Female sex workers	2015	Mombasa	19 to 66	Non-stratified	GE	NAAT	93.3	99.2	399	2.5	2.1 (0.6-4.0)	Grabert 2022[902]
F	Female sex workers	2019	Nairobi	18 to 45	Non-stratified	UR	NAAT	91.6	100.0	1042	6.4	7.1 (5.6-8.8)	Beksinska 2021[388]

Study year is midpoint between start and end of data collection period. Sensitivity and specificity of diagnostic tests collated per approach in Chapter 2. ANC: antenatal care, FP: family planning, F: female, GE: genital fluid, GYN: gynaecology clinic M: male, OPD: outpatient department, samp: sample, sens: sensitivity, spec: specificity, UR: urine, NR: not reported.

E.1.4 Quality-adjusted life years lost

Equations for individual and combined sequelae

QALYs lost were calculated separately for individual and combined sequelae. In the notation used below, subscripts s and ℓ refer to shorter- and longer-term sequelae, respectively.

For individual sequelae (whether short- or long-term), QALYs lost (Q) were calculated as the product of the number of incident gonorrhoea cases (x), the probability of developing the sequela (p), the duration of utility loss (d), and the decrement in health utility ($1 - u$):

$$Q = x\,p\,d\,(1-u) \tag{E.33}$$

For combined sequelae following PID, QALYs lost were calculated in two parts, consistent with the approach used by Li *et al.*[407] covering: (i) the overlapping period, during which both sequelae occurred simultaneously, corresponding to the duration of the shorter-term sequela (d_s); and (ii) the extended period, during which only the longer-term sequela persisted, defined as the remaining duration of the longer-term sequela ($d_\ell - d_s$ where $d_\ell > d_s$). During the overlapping period, the joint utility was calculated as the product of the individual utility weights ($u_s\,u_\ell$). During the extended period, only the utility of the longer-term sequela (u_ℓ) was applied. The total QALY loss among individuals with combined sequelae was:

$$Q_{s\ell} = x\ p_s\ p_\ell\ [(1 - u_s\,u_\ell)\,d_s + (1-u_\ell)(d_\ell - d_s)] \tag{E.34}$$

Here, x denoted the estimated number of PID cases, p_s and p_ℓ were the probabilities of developing each sequela, u_s and u_ℓ were the respective utility weights, and d_s and d_ℓ were the corresponding durations.

For sequelae extending beyond the year of infection, durations were calculated using a life table approach and were discounted to the year of infection.[407]

Adverse birth outcomes

To quantify QALYs lost due to stillbirth and LBW, estimates were required for the probability of each outcome among women with untreated gonorrhoea. However, direct estimates of these probabilities are rarely available. Instead, most studies report associations using odds ratios (ORs), which quantify the relative odds of an outcome in exposed versus unexposed groups, i.e., among women with untreated

gonorrhoea compared to those without gonorrhoea or with treated infection. ORs alone cannot be used to estimate absolute risks, since they do not account for the underlying prevalence of the outcome in the general population. Therefore, the approach described by Nyemba et al.[272] was used to convert ORs into absolute probabilities suitable for QALY calculations, by linking relative and absolute measures through algebraic relationships.

The approach involved expressing the outcome probability among exposed individuals (p_1) and unexposed individuals (p_0) using both the OR and risk ratio (RR):

$$\text{OR} = \frac{p_1}{1-p_1} / \frac{p_0}{1-p_0} \tag{E.35}$$

$$\text{RR} = \frac{p_1}{p_0} \tag{E.36}$$

To express RR in terms of OR, Equation E.35 was substituted into E.36:

$$\text{RR} = \frac{\text{OR}}{1 - p_0(1 - \text{OR})} \tag{E.37}$$

The background prevalence of the adverse outcome in the general pregnant population (u) was calculated using the median equilibrium prevalence of untreated gonorrhoea at baseline (g):

$$u = g \cdot p_1 + (1 - g) \cdot p_0 \tag{E.38}$$

Substituting Equation E.37 and solving for p_0 yielded:

$$p_0 = \frac{u}{1 + g \cdot (\frac{\text{OR}}{1-p_0(1-\text{OR})} - 1)} \tag{E.39}$$

Equation E.39 was rearranged into a quadratic Equation:

$$a \cdot {p_0}^2 + b \cdot p_0 + c = 0 \tag{E.40}$$

Where the coefficients were defined as:

$a = (1 - g)(\text{OR} - 1)$

$b = 1 - (\text{OR} - 1)(u - g)$

$c = -u$

The value for p_0 was obtained using the quadratic formula: $p_0 = \frac{-b \pm \sqrt{b^2 - 4ac}}{2a}$

The value for p_1 was then calculated using Equation E.35.

The odds ratios, background prevalence, and resulting probability estimates (p_1) are in Table E5.

Table E5: Probability of adverse outcomes in pregnant women with untreated gonorrhoea.

Parameter	Stillbirth	Low birth weight
Odds ratio (effect of gonorrhoea on outcome)	2.16 (1.25-3.46)[71]	1.66 (1.12 - 2.48)[71]
Background prevalence of outcome in general pregnant population	0.023[415]	0.115[416]
Estimated probability of outcome in pregnant women with untreated gonorrhoea	0.047	0.176
Estimated probability of outcome in pregnant women with treated gonorrhoea or no gonorrhoea	0.023	0.114

Duration and discounting

Long-term durations were assigned to chronic sequelae of PID (CPP and TFI) in non-pregnant women and adverse birth outcomes (stillbirth and LBW) in pregnant women and their infants. For chronic sequelae, a 10-year duration was assumed, representing a conservative intermediate between the 5-year, 10-year, and lifetime durations considered by Li *et al.*[407] Stillbirth and LBW losses were valued using life expectancy at birth (65 years).[287,413,424,903,904] For mothers, stillbirth-associated losses were valued over 10 years based on maternal perinatal mortality surveys,[412] and LBW-associated maternal impacts were assumed to have the same 10-year duration.

Years lived with long-term sequelae were estimated assuming infections and sequelae onset at age group midpoints: age 20 for the 15-24 year group and age 37 for the 25-49 years group. For chronic sequelae, onset was modelled with a 5 year lag after infection, with health losses occurring from ages 25-35 years for the younger group and 42-52 years for the older group. Years lived were calculated using a life table approach with discounting applied from the year of infection.[407] Life tables were constructed using five-year age intervals for the conditional probabilities of all-cause mortality and survival from UN WPP projections.[287] Deaths were assumed to occur at the midpoint of each age interval. Age weights were not applied, in line with GBD and WHO conventions valuing health losses equally across age.[905,906]

For each interval, the undiscounted years lived (YL_u) were calculated by incorporating the probability of surviving from age at infection to the start of the age interval (p_i), and the probability of death during the interval (p_d).

$$\mathrm{YL}_u = p_i \left[2.5\, p_d + 5\,(1 - p_d)\right] \tag{E.41}$$

The years lived were then discounted (YL_d) for each interval using a 3% annual discount rate applied from infection onset to the interval midpoint:

$$YL_d = \sum_{k=0}^{n-1} \frac{1}{(1+r)^{t+k}} \tag{E.42}$$

Where r was the annual discount rate, t was the number of years from infection onset to interval start (including sequelae onset delays), n was the number of years with the sequela, and k indexed each year within the interval.

Undiscounted and discounted years lived were summed across relevant age intervals (Table E6) and used to calculate lifetime QALYs lost due to each sequela.

Table E6: Years lived with health losses due to each sequela

Sequelae	Age group	Age of infection	Duration sequela	Years lived	
				Undiscounted	Discounted
Chronic: CPP and TFI	15-24 years	20	10	9.75	7.28
Chronic: CPP and TFI	25-49 years	37	10	9.23	6.92
Maternal: Stillbirth and LBW	15-24 years	20	10	9.89	8.56
Maternal: Stillbirth and LBW	25-49 years	37	10	9.59	8.46
Stillbirth and LBW	Neonate	0	64	56.5	26.3

E.2 Supplementary results

E.2.1 Transmission model calibration

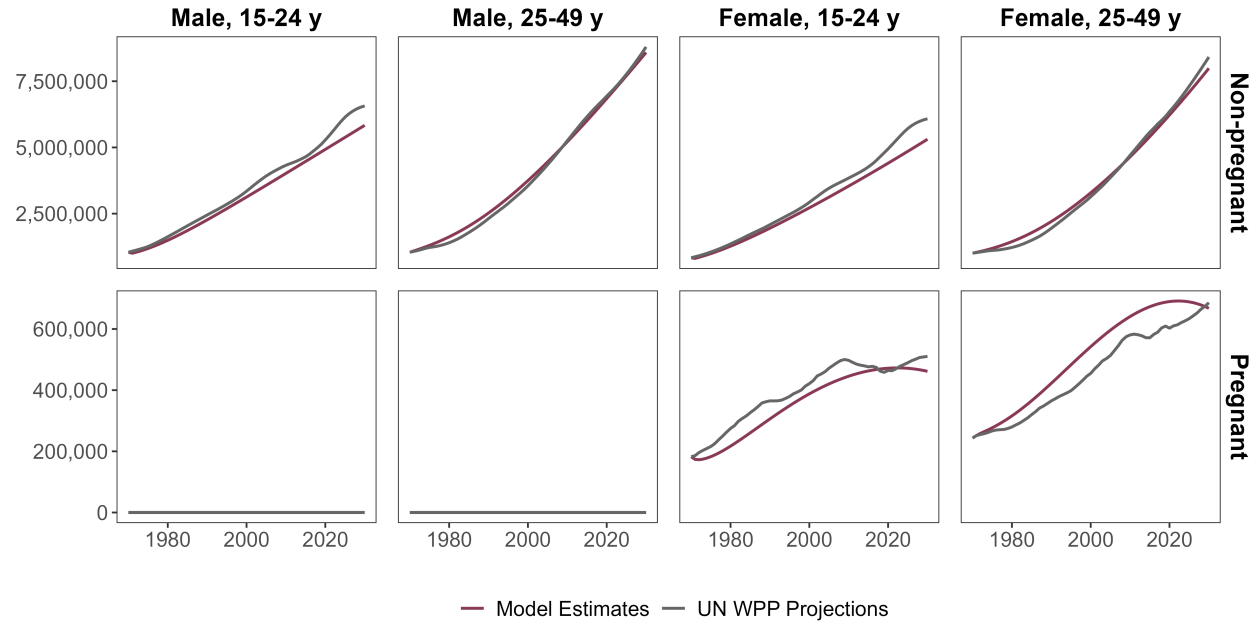


Figure E2: Comparison of model population estimates with UN WPP projections

Deviations between model estimates and UN WPP annual projections resulted from demographic parameters in the model, which were derived from cohort-based calculations and linearly interpolated between 1970 and 2030.

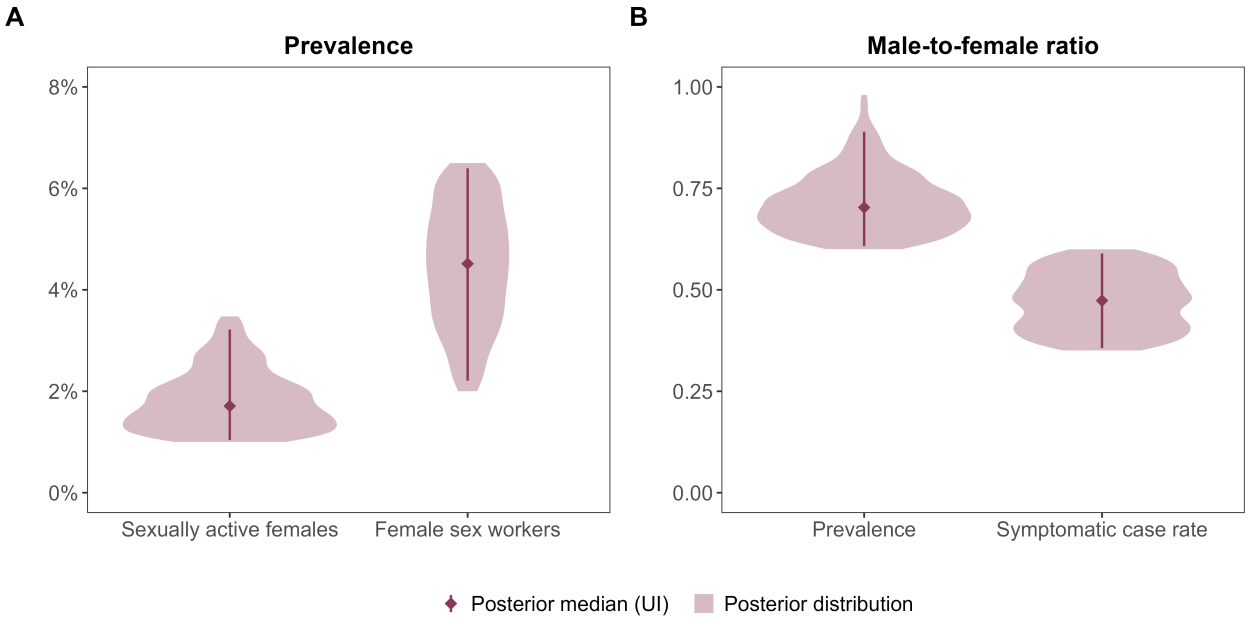


Figure E3: Distribution of baseline model outcomes after calibration

Posterior distributions of baseline model outcomes within calibration target ranges at equilibrium in 2025, for (A) gonorrhoea prevalence among sexually active females and FSW and (B) male-to-female ratios for prevalence and symptomatic case rates. Shaded areas represent the distribution of each outcome across retained posterior samples; points show the median, and vertical lines indicate the 95% uncertainty interval.

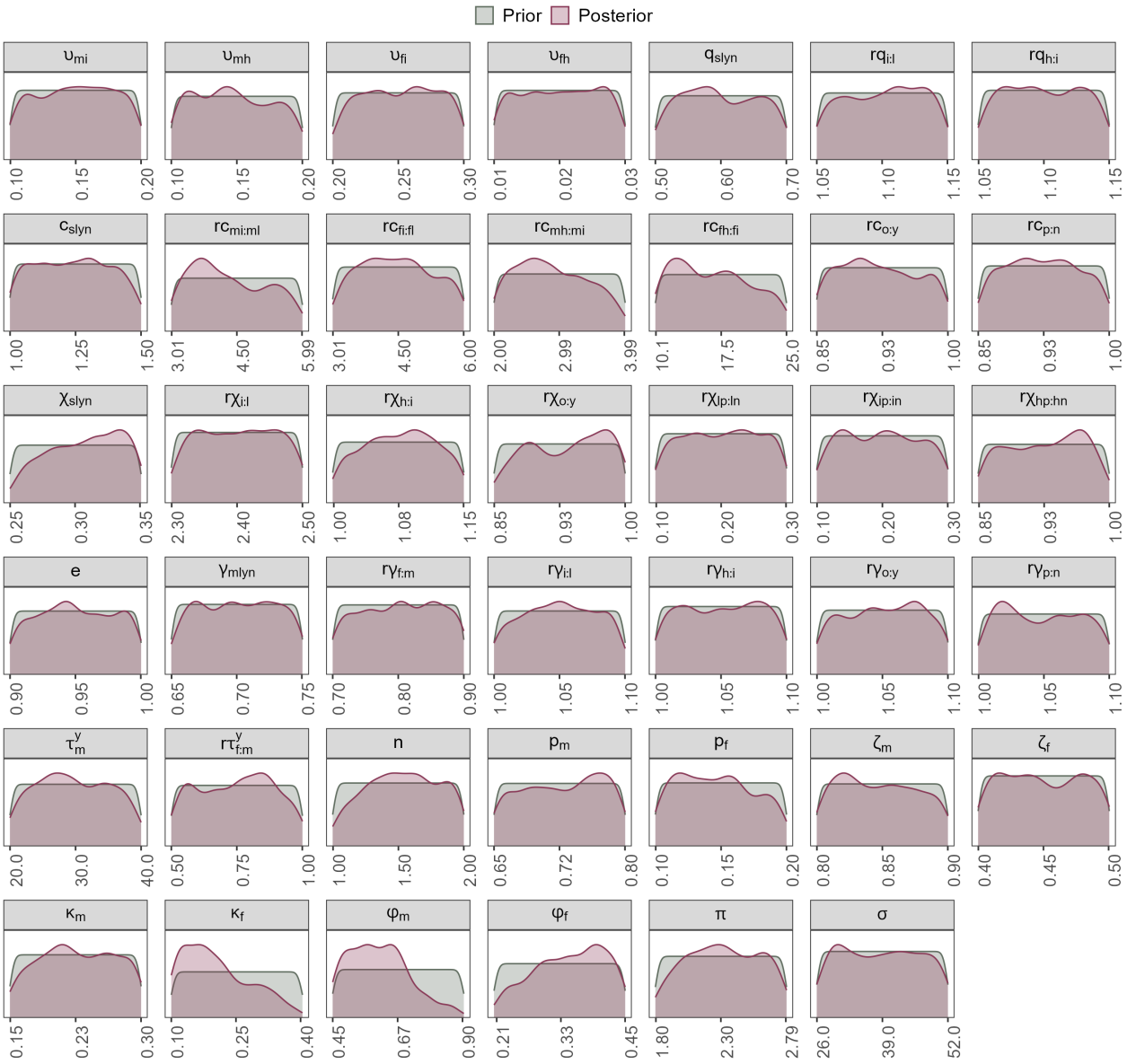


Figure E4: Prior and posterior distributions for calibrated parameters

Distributions for calibrated model parameters, stratified by prior (50 000 initial samples) and posterior (536 retained samples). Parameters are defined in Tables 6.1 and E2.

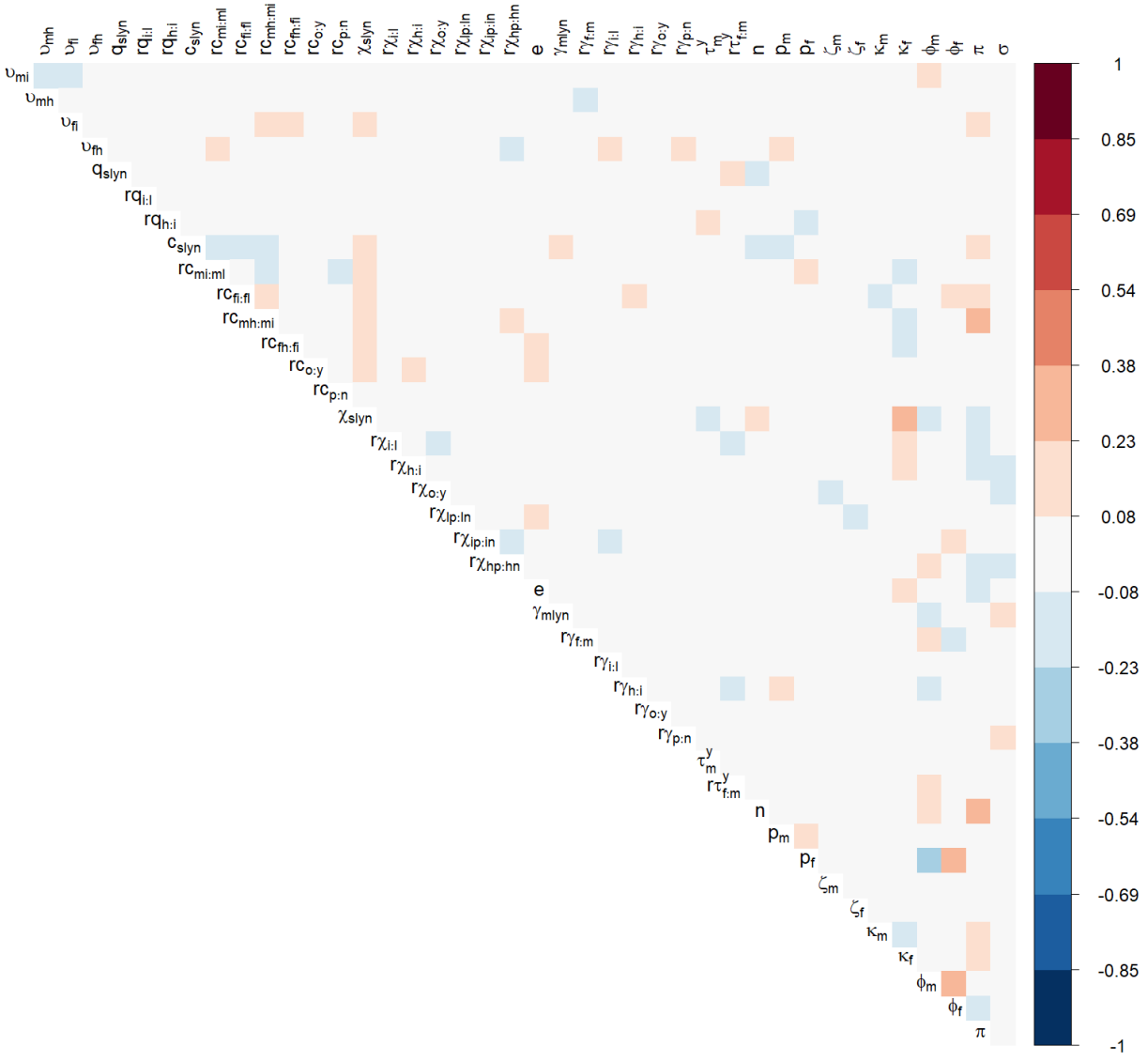


Figure E5: Rank correlations among posterior samples for calibrated model parameters

Parameters are defined in Tables 6.1 and E2.

E.2.2 Baseline outcomes

Table E7: Predicted gonorrhoea burden at baseline, 2025

Variable	CFSW	FSW	Men	Pregnant	AGYW
Population size (millions)	1.91 (1.35 - 2.58)	0.26 (0.14 - 0.39)	13.1 (Fixed)*	1.16 (1.13 - 1.19)	4.85 (4.82 - 4.88)
Prevalence (%)	3.56 (1.99 - 6.74)	4.74 (2.42 - 6.56)	1.28 (0.74 - 2.34)	1.89 (1.14 - 3.59)	1.79 (1.10 - 3.32)
Incident infections per 100 individuals, per target population	13.1 (6.88 - 24.8)	11.9 (5.67 - 18.6)	4.62 (2.70 - 8.29)	4.97 (2.79 - 9.14)	4.68 (2.76 - 8.52)
QALYs lost per 1000 individuals, per target population	0.56 (0.27 - 1.19)	22.8 (11.1 - 36.0)	0.21 (0.11 - 0.45)	80.1 (45.1 - 145)	1.98 (1.21 - 3.67)
Proportion total incident infections in target population (%)	21.4 (13.7 - 29.3)	2.48 (1.00 - 5.55)	50.0 (45.4 - 55.0)	5.37 (4.57 - 6.41)	14.5 (12.6 - 16.5)
Proportion total QALYs lost in target population (%)	0.84 (0.48 - 1.60)	4.42 (1.67 - 10.4)	2.07 (1.31 - 3.58)	80.6 (76.1 - 83.8)	5.69 (4.53 - 7.20)

Results presented as medians with uncertainty intervals and rounded to three significant figures. *Total population size by sex and age is fixed to match annual UN WPP projections.[287] Variation introduced in population size estimate for AGYW due to calibrated probability of being ever sexually active. AGYW: sexually active adolescent girls and young women; CFSW: clients of female sex workers; FSW: female sex workers.

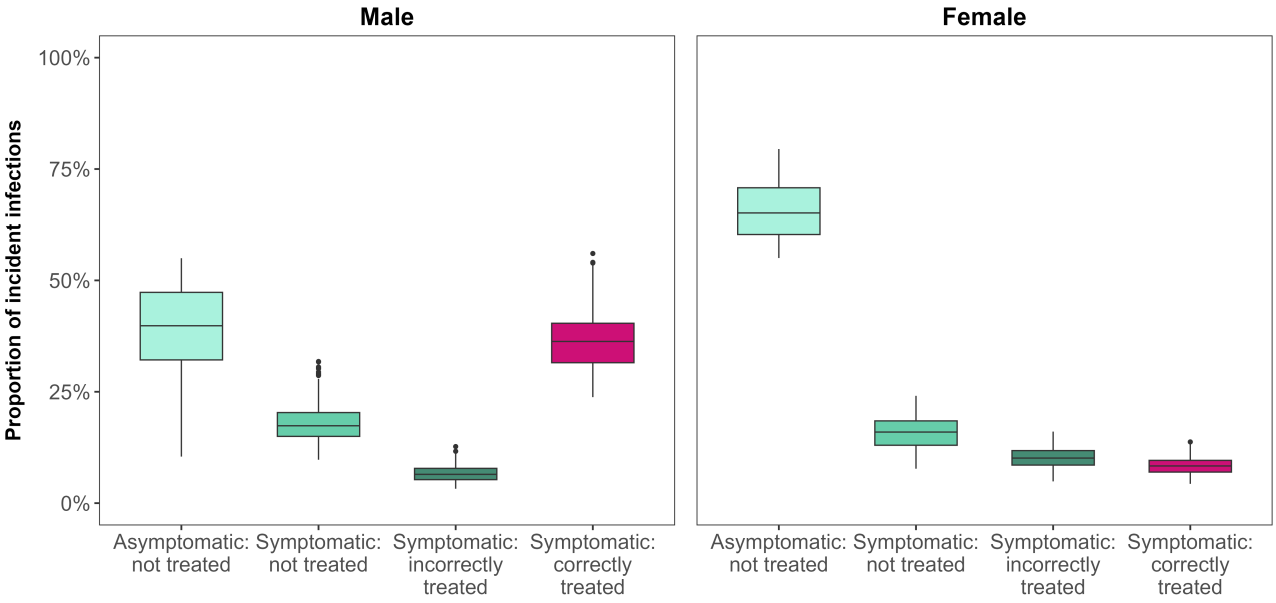


Figure E6: Distribution of incident gonorrhoea infections by symptom and treatment status at baseline, 2025

Proportion of incident gonorrhoea infections in 2025 that were asymptomatic and did not access care, symptomatic and did not access care, symptomatic and accessed care but received incorrect treatment, or symptomatic and accessed care and received correct treatment. Proportions presented as medians with 95% uncertainty intervals.

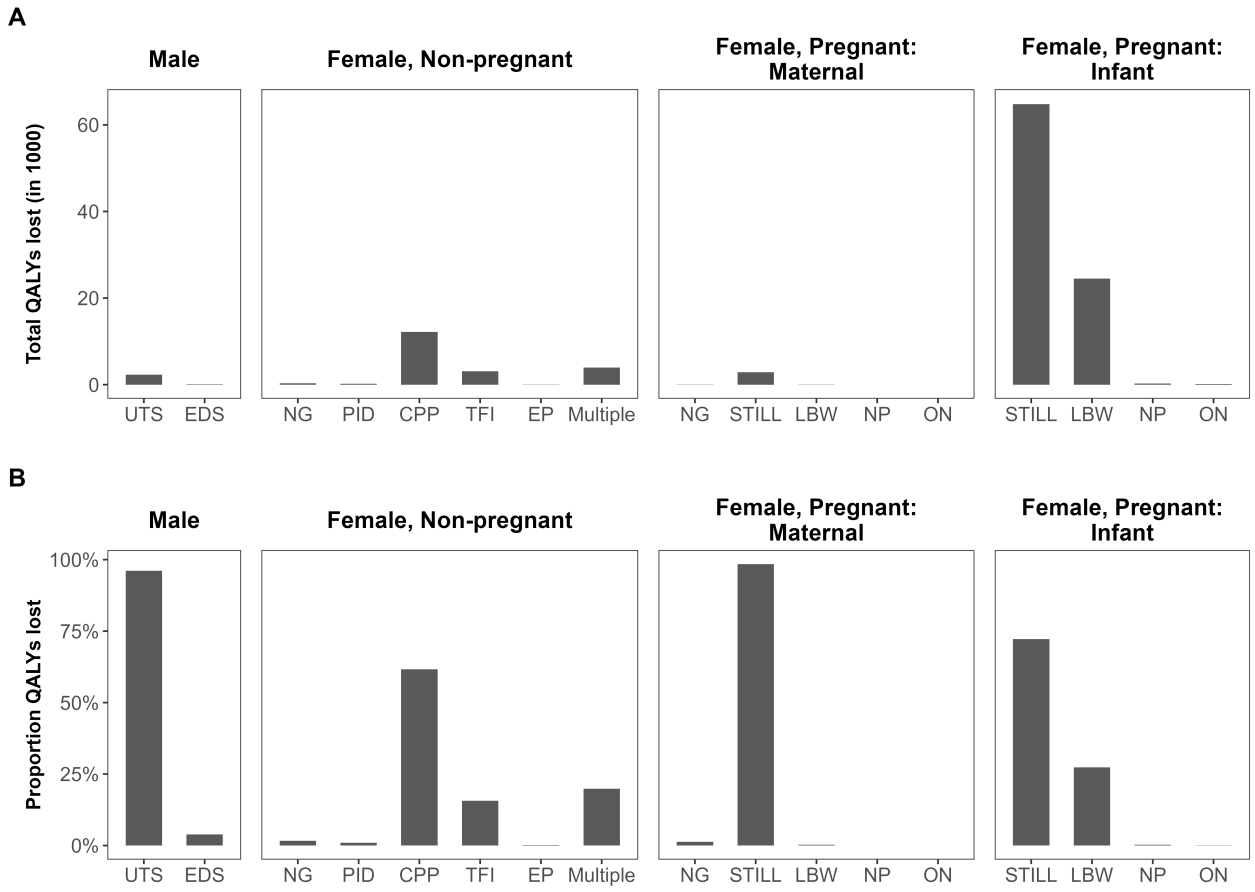


Figure E7: Decomposition of total discounted lifetime QALYs lost

Decomposition of total discounted lifetime QALYs lost per population group as (A) number of QALYs lost and (B) proportional distribution of QALYs lost among each population group. Bars represent median estimates. CPP: chronic pelvic pain, EDS: epididymitis, EP: ectopic pregnancy, LB: live birth, LBW: low birth weight, NG: *Neisseria gonorrhoeae*, NP: neonatal pneumonia, ON: ophthalmia neonatorum, PID: pelvic inflammatory disease, TFI: tubal factor infertility, UTS: urethritis, Multiple: combined sequelae of CPP, TFI, and EP.

Table E8: Number of stillbirth and LBW cases among pregnant women at baseline, 2025

Indicator	Median (95% UI)
Pregnancy with untreated gonorrhoea	
Number of births	52 330 (29 373 - 95 066)
Number of livebirths	49 870 (27 993 - 90 598)
Number of stillbirths	2 459 (1 381 - 4 468)
Number of LBW livebirths	8 777 (4 927 - 15 945)
Pregnancy with treated gonorrhoea or no gonorrhoea	
Number of births	1 489 681 (1 436 143 - 1 544 233)
Number of livebirths	1 456 089 (1 403 758 - 1 509 410)
Number of stillbirths	33 592 (32 385 - 34 822)
Number of LBW livebirths	165 819 (159 860 - 171 892)
Overall	
Number of births	1 546 822 (1 508 141 - 1 587 113)
Number of livebirths	1 510 811 (1 472 471 - 1 550 092)
Number of stillbirths	36 200 (34 940 - 37 609)
Number of LBW livebirths	175 310 (170 205 - 180 127)

QALYs estimated using the number of stillbirth and LBW cases among pregnant women with untreated gonorrhoea. Cases among pregnant women with treated gonorrhoea or no gonorrhoea were also estimated to validate that model population-level estimates aligned with data used to inform parametrisation (34 985 total stillbirths[415] and 172 310 LBW livebirths[416] in Kenya in 2015.)

E.2.3 Intervention outcomes

Table E9: Predicted population-level impact of diagnostic testing and screening strategies among each target population with limited POCT availability, 2025–2030

Variable	CFSW	FSW	Men	Pregnant	AGYW
Diagnostic testing strategy					
Coverage of POCT among eligible cases* (%)	18.4 (8.55 - 40.2)	66.8 (26.0 - 100)	7.95 (3.91 - 16.2)	33.6 (15.8 - 71.4)	13.1 (5.99 - 28.6)
Percentage of infections averted relative to baseline (%)	2.21 (0.77 - 5.31)	2.07 (0.63 - 5.59)	1.28 (0.46 - 3.03)	1.00 (0.47 - 2.12)	0.93 (0.43 - 1.99)
Percentage of QALYs gained relative to baseline (%)	2.23 (0.78 - 5.28)	2.17 (0.71 - 5.58)	1.29 (0.47 - 3.05)	3.51 (1.75 - 7.22)	0.85 (0.39 - 1.82)
Infections averted per 100 tests	99.8 (45.7 - 165)	94.3 (41.9 - 193)	58.7 (27.1 - 96.3)	44.1 (28.1 - 66.2)	40.6 (26.3 - 64.0)
QALYs gained per 100 tests	10.4 (4.83 - 17.8)	10.1 (5.33 - 20.0)	6.07 (2.90 - 10.1)	16.2 (11.2 - 23.1)	3.80 (2.54 - 5.82)
Screening strategy					
Coverage of POCT among eligible cases* (%)	0.98 (0.71 - 1.41)	5.77 (3.90 - 11.1)	0.14 (0.14 - 0.15)	0.82 (0.80 - 0.85)	0.52 (0.47 - 0.60)
Percentage of infections averted relative to baseline (%)	0.58 (0.41 - 0.82)	0.62 (0.20 - 1.52)	0.12 (0.10 - 0.13)	0.08 (0.06 - 0.10)	0.13 (0.11 - 0.16)
Percentage of QALYs gained relative to baseline (%)	0.58 (0.41 - 0.81)	0.65 (0.24 - 1.50)	0.11 (0.10 - 0.13)	0.30 (0.25 - 0.34)	0.12 (0.10 - 0.14)
Infections averted per 100 tests	25.5 (14.6 - 44.2)	26.9 (9.97 - 59.1)	5.04 (3.15 - 8.16)	3.51 (2.04 - 5.97)	5.89 (3.56 - 9.87)
QALYs gained per 100 tests	2.64 (1.44 - 4.70)	2.89 (1.20 - 6.04)	0.52 (0.31 - 0.87)	1.32 (0.80 - 2.42)	0.55 (0.34 - 0.92)

Results presented as medians with uncertainty intervals and rounded to three significant figures. Limited POCT availability was modelled as 25 000 tests annually. *Cases refer to the number of individuals accessing care for vaginal or urethral discharge (diagnostic scenario) or attending routine healthcare services (screening scenario). Individuals may be counted more than once due to repeat infections or multiple healthcare visits. AGYW: sexually active adolescent girls and young women; CFSW: clients of female sex workers; FSW: female sex workers.

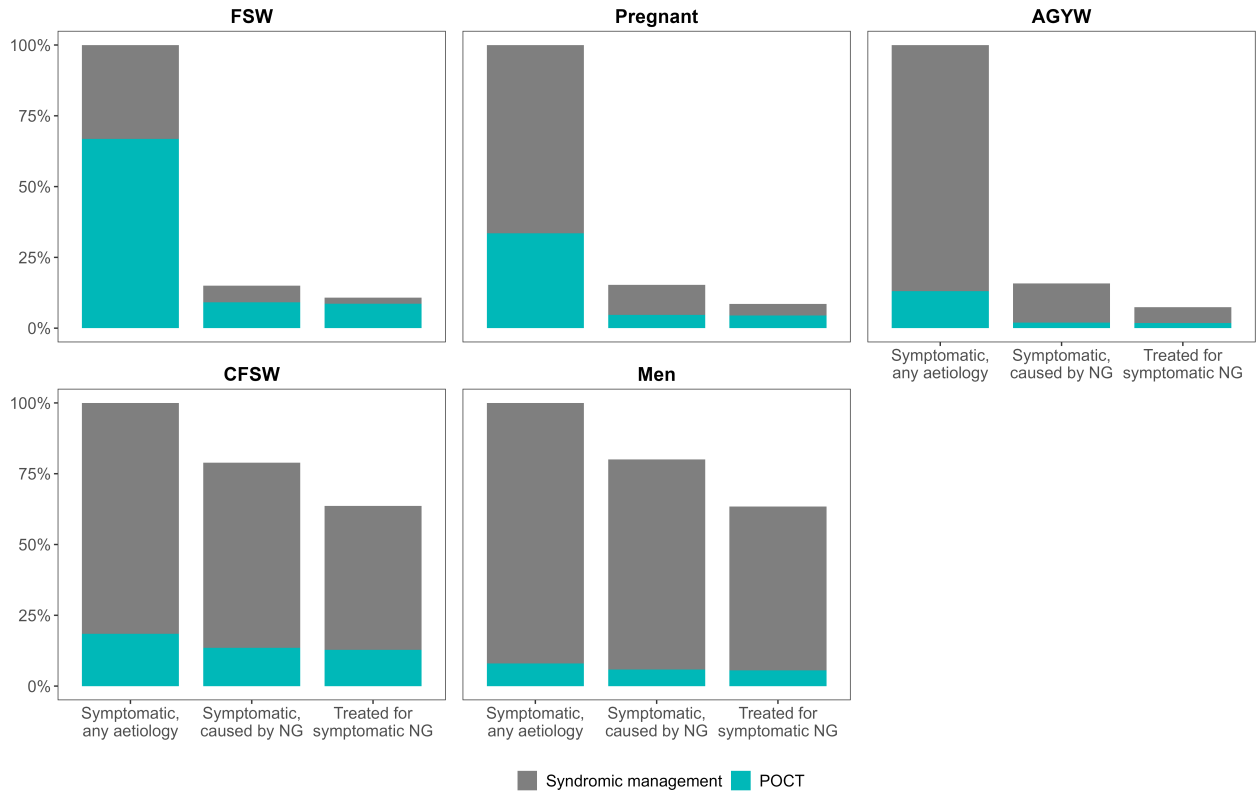


Figure E8: Cascade for diagnostic testing strategy among symptomatic cases accessing care by target population, 2025-2030

Relative proportion of symptomatic cases accessing care that are attributable to any aetiology, attributable to gonorrhoea, and correctly treated for gonorrhoea between 2025 and 2030, under limited POCT availability (25 000 annually). Bars represent the median number of cases, scaled relative to the total number of symptomatic cases accessing care for any aetiology. Colours indicate the proportion of cases receiving syndromic management or POCT.

Table E10: Predicted population-level impact of diagnostic testing and screening strategies among each target population with maximum POCT availability, 2025–2030

Variable	CFSW	FSW	Men	Pregnant	AGYW
Diagnostic testing strategy					
Average number POCT used annually (×100 000)	1.39 (0.65 - 2.97)	0.38 (0.13 - 0.99)	3.19 (1.59 - 6.46)	0.72 (0.35 - 1.54)	1.96 (0.90 - 4.28)
Percentage of infections averted relative to baseline (%)	11.6 (4.87 - 24.1)	3.07 (0.73 - 10.0)	15.3 (6.81 - 28.9)	3.05 (1.64 - 4.97)	6.93 (3.81 - 10.7)
Percentage of QALYs gained relative to baseline (%)	11.6 (4.87 - 24.1)	3.33 (0.83 - 10.1)	15.4 (6.86 - 28.9)	10.7 (6.43 - 15.8)	6.28 (3.45 - 9.76)
Infections averted per 100 tests	102.0 (46.2 - 171)	93.9 (41.9 - 191)	60.3 (27.4 - 99.8)	43.9 (28.1 - 66.0)	39.7 (25.9 - 62.1)
QALYs gained per 100 tests	10.5 (4.83 - 18.2)	10.1 (5.32 - 20.0)	6.15 (2.92 - 10.5)	16.1 (11.1 - 22.8)	3.73 (2.49 - 5.70)
Screening strategy					
Average number POCT used annually (×100 000)	25.4 (18.0 - 34.4)	4.33 (2.25 - 6.38)	175.4 (175.1 - 175.6)	30.4 (29.7 - 31.1)	47.6 (42.8 - 52.1)
Percentage of infections averted relative to baseline (%)	36.3 (27.7 - 42.2)	7.25 (2.16 - 19.5)	45.8 (40.3 - 49.1)	7.59 (6.07 - 9.53)	17.46 (14.6 - 20.5)
Percentage of QALYs gained relative to baseline (%)	36.1 (27.9 - 41.9)	7.65 (2.50 - 19.7)	45.5 (40.1 - 48.5)	28.0 (24.6 - 31.8)	15.9 (13.3 - 18.8)
Infections averted per 100 tests	15.6 (8.48 - 28.6)	19.3 (6.96 - 42.0)	2.84 (1.68 - 4.86)	2.76 (1.60 - 4.73)	4.06 (2.38 - 6.88)
QALYs gained per 100 tests	1.60 (0.85 - 2.95)	2.08 (0.86 - 4.31)	0.29 (0.17 - 0.52)	1.03 (0.62 - 1.90)	0.38 (0.23 - 0.66)

Results presented as medians with uncertainty intervals and rounded to three significant figures. Maximum POCT availability was modelled as all eligible cases tested. Cases refer to the number of individuals accessing care for vaginal or urethral discharge (diagnostic scenario) or attending routine healthcare services (screening scenario). Individuals may be counted more than once due to repeat infections or multiple healthcare visits. AGYW: sexually active adolescent girls and young women; CFSW: clients of female sex workers; FSW: female sex workers.

E.2.4 Sensitivity analyses

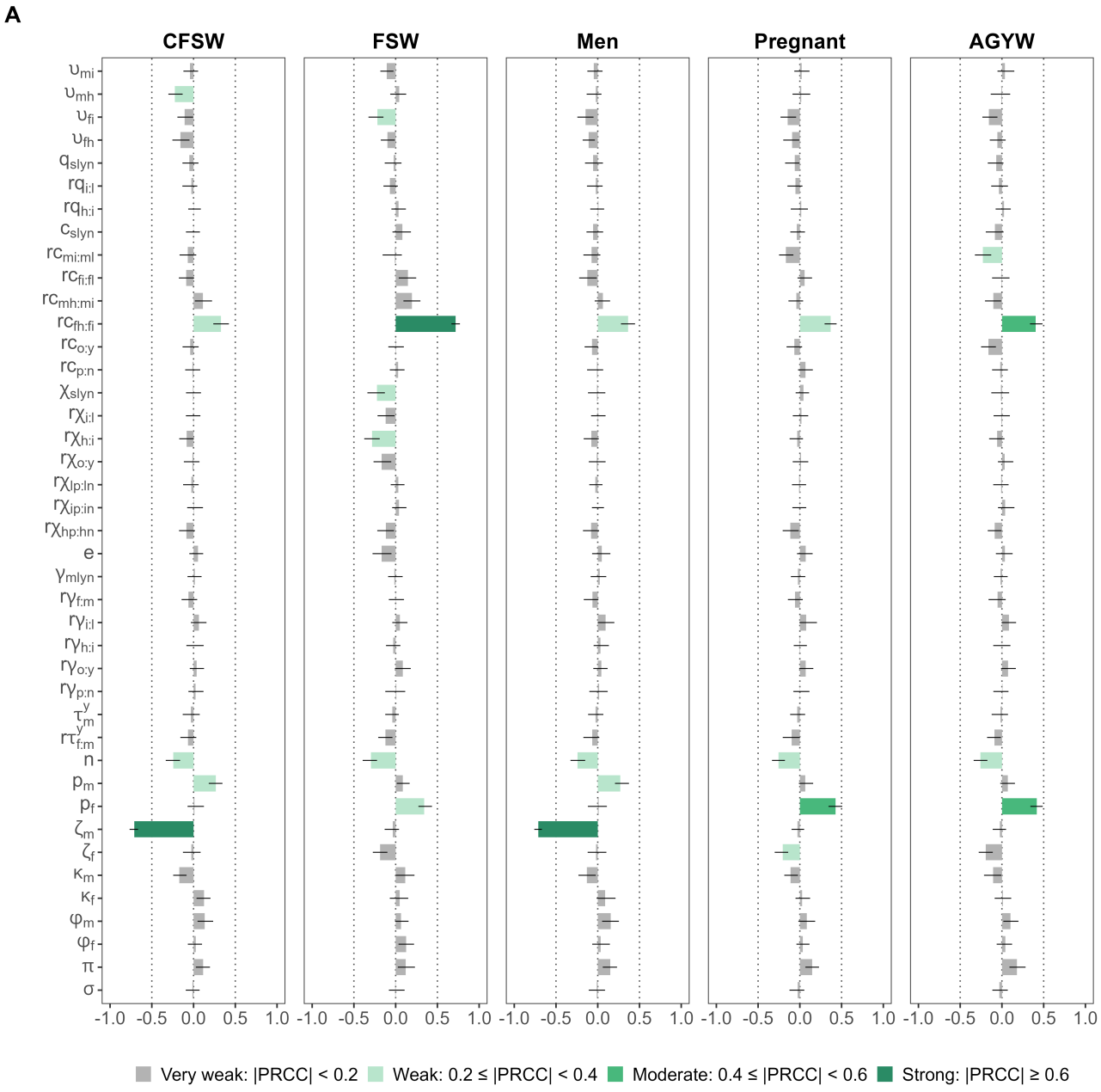


Figure E9: Correlation between parameter values and the proportion of infections averted by test strategy and target population

Partial rank correlation coefficients between posterior parameter values and the proportion of incident gonorrhoea infections averted under limited POCT availability (25 000 annually) between 2025 and 2030 for (A) diagnostic testing and (B) screening strategies across each target population. Bars and lines indicate median and 95% confidence intervals. Parameters are defined in Table 6.1.

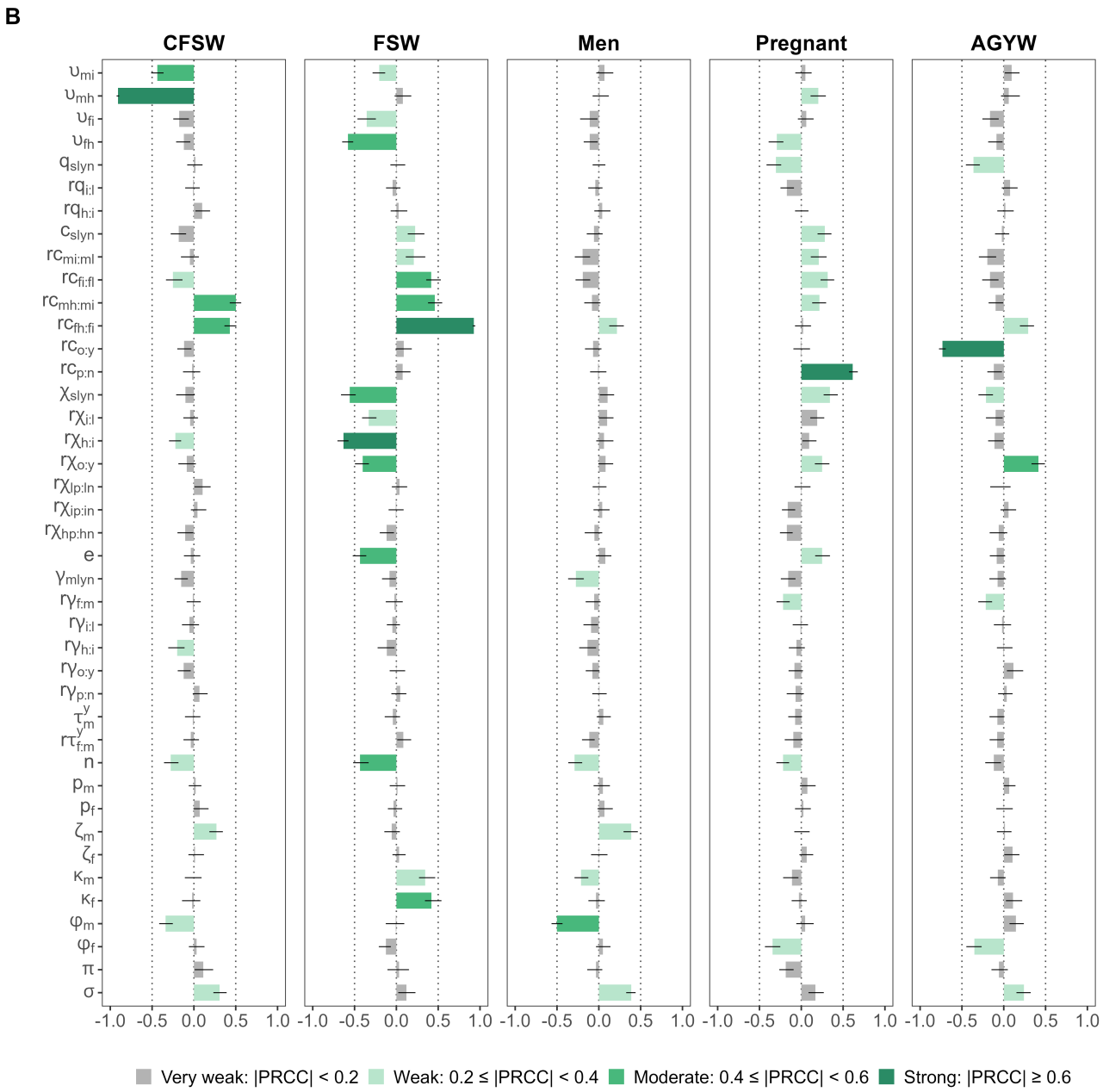


Figure E9: Correlation between parameter values and the proportion of infections averted by test strategy and target population (continued)

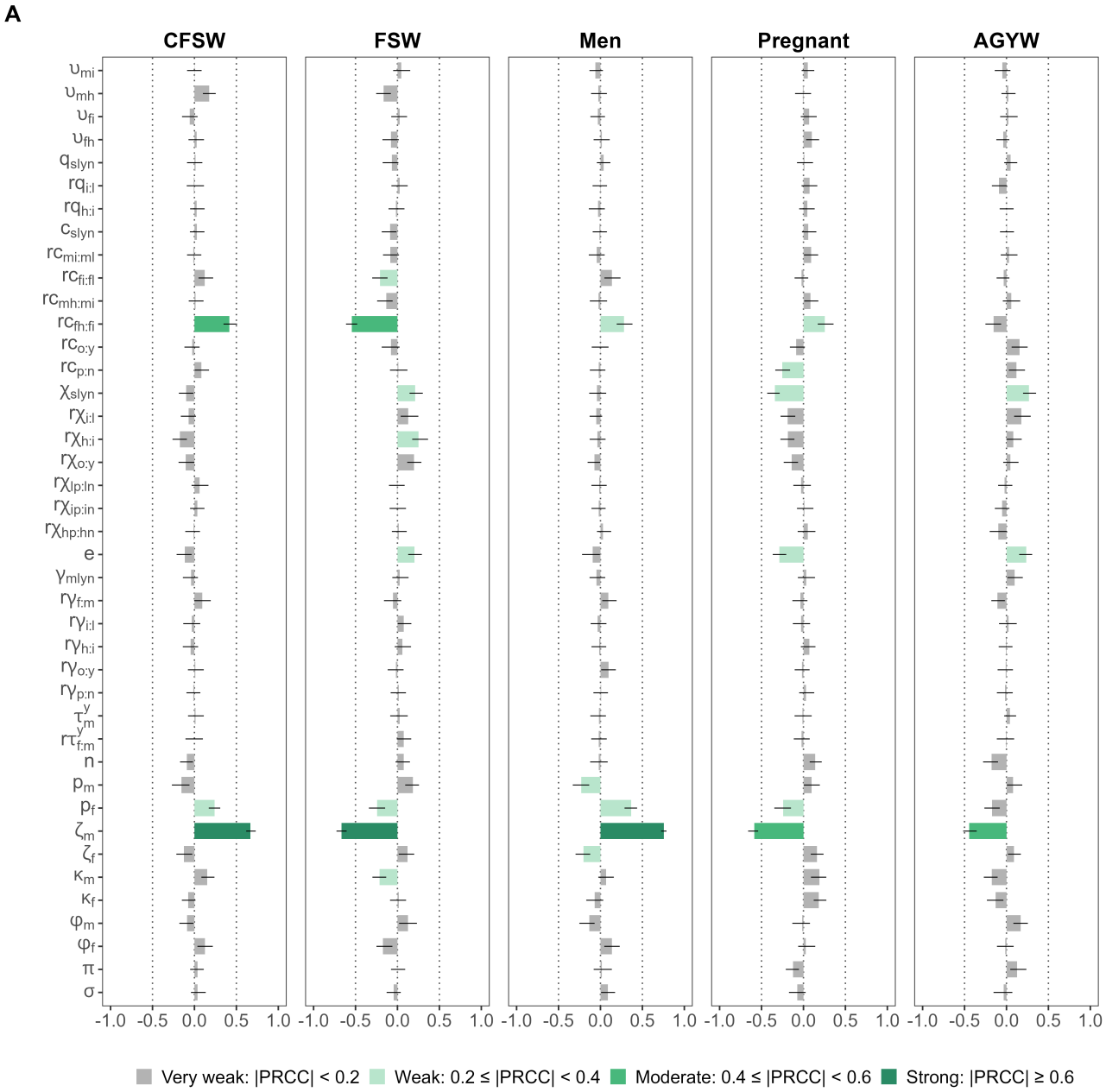


Figure E10: Correlation between parameter values and target population prioritisation order by test strategy

Partial rank correlation coefficients between posterior parameter values and the prioritisation order of each target population according to the proportion of incident gonorrhoea infections averted under limited POCT availability (25 000 annually) between 2025 and 2030 for (A) diagnostic testing and (B) screening strategies across each target population. Bars and lines indicate median and 95% confidence intervals. Parameters are defined in Tables 6.1 and E2.

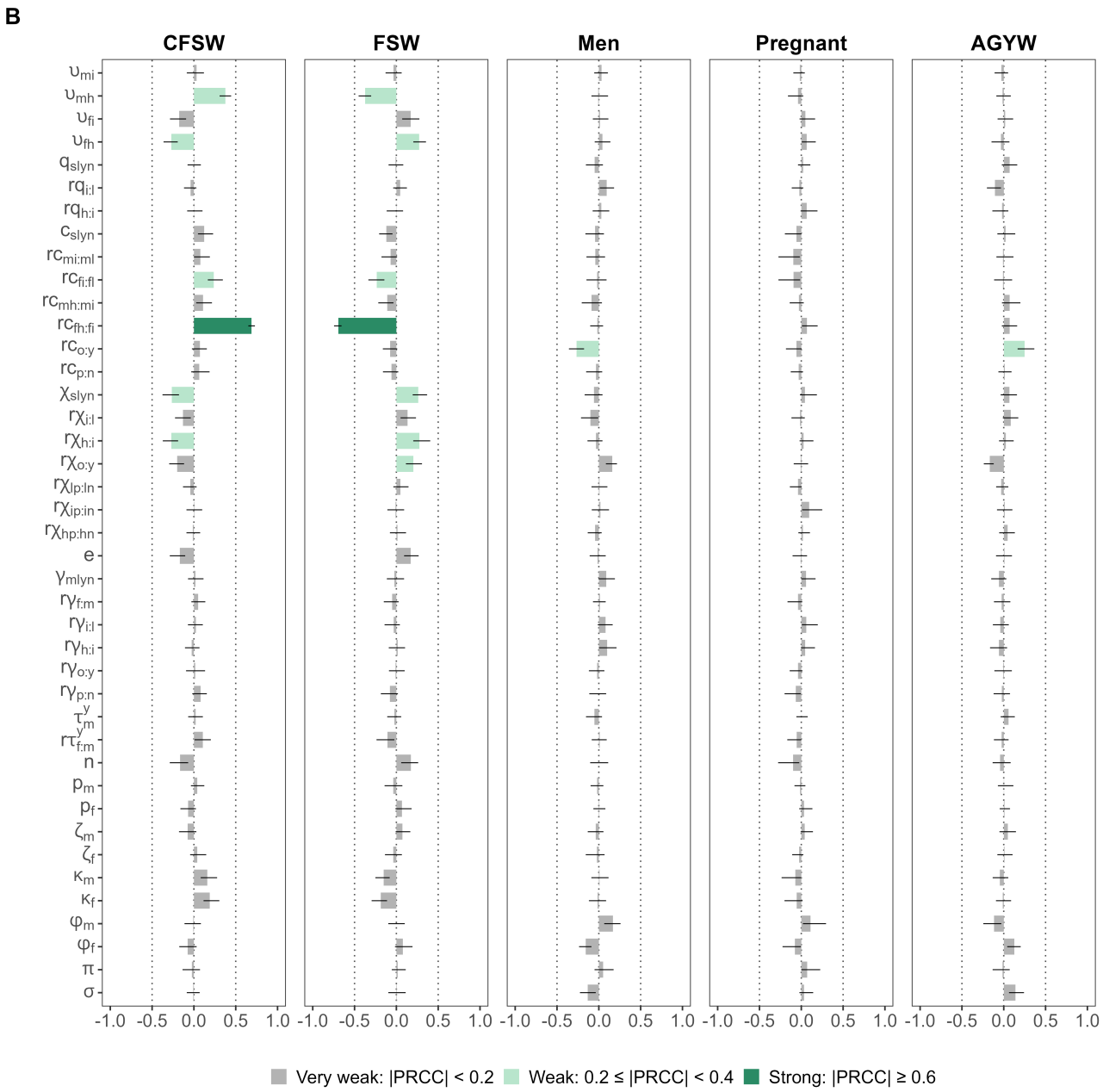


Figure E10: Correlation between parameter values and target population prioritisation order by test strategy (continued)

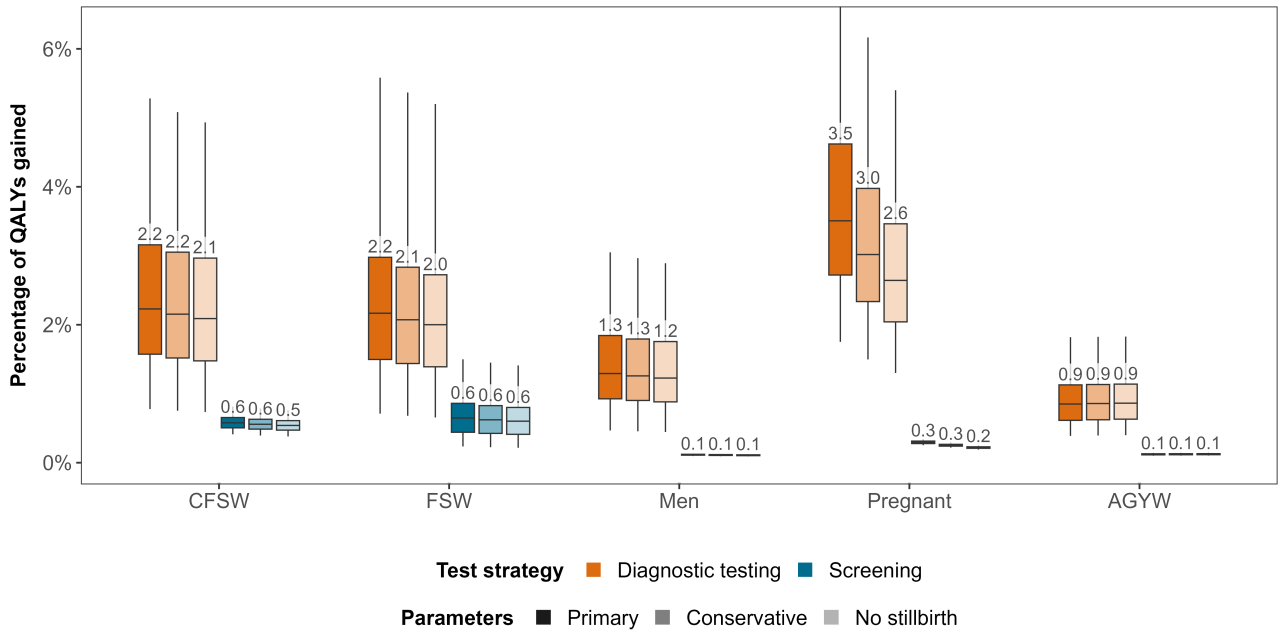


Figure E11: Influence of parameter assumptions for pregnancy-related sequelae on the predicted population-level impact of diagnostic testing and screening strategies, 2025–2030

Population-level impact of deploying gonorrhoea POCTs (25 000 annually) among each of five target populations between 2025 and 2030 through either diagnostic testing of individuals accessing care for urethral or vaginal discharge, or screening of attendees at routine healthcare services. Impact was assessed as the percentage of lifetime QALYs gained relative to the baseline scenario of syndromic management only, under three sets of parameter assumptions: (i) Primary, as used in the main analysis, (ii) Conservative, which increased the utility weight for stillbirth from 0 to 0.5, and reduced the duration of infant LBW-associated losses from lifetime to 10 years, and (iii) No stillbirth, which increased the utility weight for stillbirth from 0 to 1 to exclude stillbirth QALY losses for infants. Parameters describing maternal QALY losses for stillbirth and LBW remained unchanged. Boxplots represent the median (horizontal line), interquartile range (box), and uncertainty interval (whiskers). AGYW: sexually active adolescent girls and young women; CFSW: clients of female sex workers; FSW: female sex workers; POCT: point-of-care test; LBW: low birth weight; QALY: quality-adjusted life year.