demonstrated that the vaginal microbiota of reproductive-age women in the US cluster into five groups of bacterial communities; four dominated by *Lactobacillus spp*. (*L iners, L crispatus, L gasseri, L jensenii*), and one lacking significant numbers of lactobacilli and characterised by higher proportions of strictly anaerobic organisms (termed group IV). We sought to compare the vaginal microbiota of *T vaginalis*-positive and *T vaginalis*-negative women using cultivation-independent methods. To our knowledge, this is the first analysis of the relationship between *T vaginalis* and vaginal bacterial communities characterised by molecular methodologies.

Methods Self-collected vaginal swabs were obtained cross-sectionally from 396 asymptomatic US women equally representing four ethnic/racial groups. Screening for the presence of *T vaginalis* was performed using PCR targeting the 18S rRNA and ß-tubulin genes. Vaginal bacterial composition was characterised by pyrosequencing of barcoded 16S rRNA genes. The relationship between vaginal microbiota and *T vaginalis* was evaluated by Fisher's exact testing and logistic regression.

Results Of the 11 *T vaginalis*-positive cases, 8 (72%) were classified to the low-Lactobacillus group IV, 2 (18%) and 1 (9%) with communities dominated by *L iners* and *L crispatus*, respectively (p value: 0.056). Group IV was associated with an eightfold increased odds of detecting *T vaginalis* compared to women with communities dominated by *L crispatus* (OR: 8.26, 95% CI: 1.07% to 372.65%, p value: 0.04). Other than the major bacteria dominating each cluster, none of the other observed taxa showed significant association with *T vaginalis*-positivity although this may reflect few observed cases rather than lack of a true association. Ten (91%) of the *T vaginalis* cases self-reported Black ethnicity and 1 (9%) reported Asian, (p value: 0.00002).

Conclusion Molecular analyses revealed that vaginal microbiota with low proportions of lactobacilli were significantly associated with presence of *T vaginalis*. Longitudinal studies are needed to identify the causal nature of the relationship between vaginal bacterial communities and STI risk.

O1-S05.06 ASSOCIATION BETWEEN PREVALENT *BACTERIAL VAGINOSIS* (BV) AND HIV INFECTION AMONG FEMALE SEX WORKERS AT TWO AFRICAN AND TWO INDIAN SITES

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Background BV is the most common female genital infection, particularly in developing countries and reaches its highest prevalence among female sex workers (FSWs). Over the past decade, evidence has accumulated as to its role in HIV acquisition. If BV actually plays such a role, even a modest relative risk would yield an important attributable risk for HIV, particularly in developing countries where FSWs play a pivotal role in the dynamics of HIV epidemics. Yet, data on this association among FSWs are still scanty. We analysed data from high risk women screened prior to participation in a microbicide trial to estimate BV and HIV respective prevalence and to study their association, in the presence or not of other STIs.

Methods Data from Kampala, Cotonou, Chennai and Mudhol/Jhamkandi sites were analysed. Socio-demographic, behavioural and medical data were collected through individual interviews. Blood was taken for HIV and syphilis antibody testing. Genital samples were collected for BV diagnosis using Nugent scoring system, of gonorrhoea and chlamydias by SDA and of trichomoniasis (TV) and candidiasis on wet mount. Binomial log regression was used to estimate HIV prevalence ratio (PR) in relation to BV. Fitting of multivariable models was done with backward selection using the approach of proportional change of the PR. Product terms from BV and each of other STIs were included in the final model. Significance level was set at 5% for testing associations and 15% for interactions.

Abstract O1-S05.06 Table 1 Final multivariate model *describing association between *Bacterial vaginosis* and HIV infection with effect modification by Trichomoniasis, among 1367 FSWs recruited at 2 African and 2 Indian sites

Variables	HIV prevalence by exposure level: n_i/N_i (%)	Adjusted PR§	95% CI	p value
Bacterial vaginosis:				
Positive	192/651 (29.49)	1.25	1.05 to 1.48	0.01
Negative	177/716 (24.72)	1.00	–	–
Site:				
Mudhol/Jhamkandi	23/49 (46.94)	1.06	0.71 to 1.58	0.78
Cotonou	123/447 (27.52)	0.63	0.50 to 0.79	<0.0001
Chennai	56/355 (15.77)	0.50	0.33 to 0.76	0.001
Kampala (Ref.)	167/516 (32.36)	1.00	–	–
Past history of STI:				
Yes	198/615 (32.20)	1.43	1.21 to 1.70	<0.0001
No	171/752 (22.74)	1.00	–	–
Exerting an occupation besides commercial sex work:				
Yes	243/906 (26.82)	0.82	0.65 to 1.03	0.09
No	126/461 (27.33)	1.00	–	–
Current contraceptive method †:				
Hormonal	65/221 (29.41)	0.76	0.43 to 1.34	0.34
IUD	3/11 (27.27)	0.99	0.35 to 2.82	0.98
Female sterilisation	43/294 (14.63)	0.54	0.31 to 0.94	0.03
Condom	248/809 (30.66)	0.89	0.53 to 1.50	0.67
None (Ref.)	10/31 (32.26)	1.00	–	–
Oral sex in the past 30 days:				
Yes	5/30 (16.67)	0.58	0.35 to 0.95	0.03
No	364/1337 (27.23)	1.00	–	–
Age (years):				
15–19 (Ref.)	10/66 (15.15)	1.00	–	–
20–24	100/380 (26.32)	1.85	1.03 to 3.34	0.04
25–29	109/357 (30.53)	2.23	1.24 to 4.00	0.01
30–34	68/221 (30.77)	2.70	1.50 to 4.88	0.001
35–60	82/343 (23.91)	2.25	1.23 to 4.11	0.01
Education duration (years)				
0–6	240/782 (30.69)	1.00	–	–
7–12	124/548 (22.63)	0.76	0.63 to 0.92	0.005
13–17	5/37 (13.51)	0.50	0.22 to 1.13	0.10
Gonorrhoea‡:				
Positive	52/111 (46.85)	1.52	1.26 to 1.83	<0.0001
Negative	316/1253 (25.22)	1.00	–	–
Candidiasis:				
Positive	118/421 (28.03)	1.20	0.99 to 1.45	0.06
Negative	251/946 (26.53)	1.00	–	–
Syphilis‡:				
Positive	29/82 (35.37)	1.26	0.96 to 1.67	0.09
Negative	339/1282 (26.44)	1.00	–	–
Trichomoniasis‡:				
Positive	29/82 (35.37)	1.20	0.82 to 1.74	0.34
Negative	339/1282 (26.44)	1.00	–	–
BV_TV¶	–	0.61	0.32 to 1.13	0.12
		1.00	–	–

*This model includes, besides the site, all other covariables with p value <0.10 (though removal of neither resulted in a substantial change in the PR estimate during the confounders selection process), as well as TV and the product term between BV and TV.
†= 1 missing value.
‡= 3 missing values.
§PR=prevalence ratio.
¶BV_TV=product term between Bacterial Vaginosis (BV) and Trichomoniasis (TV).

Results Out of 1491 FSWs, BV data were available for 1367 among whom, BV and HIV prevalences were 47.6% (95% CI=45.0% to 50.3%) and 27.0% (95% CI=24.6% to 29.3%) respectively. In multivariable analysis (Abstract O1-S05.06 table 1), adjusting for site, age, years of education, occupation, current contraceptive method, oral sex, past history of STI, gonorrhoea, candidiasis and syphilis, BV was significantly associated to HIV (adjusted PR=1.20, 95% CI=1.01% to 1.42%, p=0.03). In addition, the PR was negatively modified by TV, whose prevalence was 6.7%: PR was 1.25 (1.05 to 1.48) and 0.76 (0.41 to 1.38) in the absence and the presence of TV respectively (p for interaction =0.12).
Conclusions Though its cross-sectional design precludes all directional interpretation of the findings, this study confirms the relationship between BV and HIV among FSWs and warrants prospective studies in this population. The negative modifying effect of TV on this association's measure needs further investigation.

Epidemiology oral session 6: Planning of HIV preventive interventions

O1-S06.01 IMPACT OF TARGETED INTERVENTIONS IN HIV EPIDEMICS AS PREDICTED BY MATHEMATICAL MODELS: A SYSTEMATIC REVIEW

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Background Mathematical modelling of sexually transmitted infections suggests that targeting intervention (TI) to high-risk heterosexual risk groups (HRG) who have disproportionately high exposure and potential for transmission within populations can be very effective. We reviewed HIV transmission modelling studies to better understand the potential impact of TIs or the contribution of HRG to overall HIV transmission across geographical regions and epidemic phases.
Methods We systematically searched PubMed with relevant key words to identify publications that used dynamical models of heterosexual HIV transmission, and then searched papers to identify studies that incorporated heterogeneity in risk, and provided estimates of the population attributable fraction of HIV infections due to HRGs (PAF), or fraction of infections prevented (PF) or change in prevalence due to TIs.
Results Of 917 titles, 283 were excluded on abstract review. Of 634 papers searched, 96 modelled heterogeneity, of which 26 were included. Six studies used non-regionalised models, 9 studied generalised epidemics (GE) in sub-Saharan Africa, nine studied concentrated epidemics (CE) in Asia, West Africa, Japan, and Europe, and two studied both epidemic types. The PAF of HRGs ranged from 13% to 17% in mature GEs with an HIV prevalence of 16%–22% across three studies. Five models explored TIs in GEs and predicted a PF of 12%–73% and a 0%–27% reduction in prevalence with >50% coverage of commercial partnerships. Ten studies modelled TIs in CEs, with overall HIV prevalence at the mature phase between 0.7% and 3.5%, and suggested that TIs could reduce prevalence by 14%–30%, with PFs of 25%–48% if >75% coverage of commercial partnerships. With <50% coverage of commercial partnerships, 1 study demonstrated a 14% reduction in prevalence at 10 years, and two studies predicted a PF between 13% and 20%. The PF of TIs implemented early in a CE with high coverage ranged between 27% and 97%. Two studies predicted that additional TIs (pre-exposure prophylaxis) associated with high levels of risk compensation in mature epidemic settings could reverse positive gains already made by increased condom use see Abstract O1-S06.01 table 1.
Conclusion Modelling studies suggest that TIs have the potential to reduce HIV in the overall population in generalised and concentrated epidemics. The relative impact of TIs depends on coverage, epidemic phase, differential risk between HRGs and remainder of the population, and the time-scale of outcome measurement.

Abstract O1-S06.01 Table 1 Summary of published modelling results on targeted intervention among heterosexual higher-risk groups

	Population attributable fraction, % (years)	Prevented fraction %, (years post-intervention)	Change in prevalence %, (years post-intervention)
Generalised epidemics			
Early	–	12 (4)	0 (1)
Mature	13 (4), 8–17 (20)	73 (1), 35 (10)	4–27 (10)
Concentrated epidemics			
Early	–	70 (1), 27(4), 85–97(10)	41–58 (10), 58–89 (30)
Mature	40 (1)	25–30 (1), 10–48 (10), 40 (11)	30 (5), 14 (10)

O1-S06.02 IMPACT OF PILL SHARING ON DRUG-RESISTANCE AND POPULATION-LEVEL EFFECTIVENESS OF A WIDE-SCALE ORAL PREP INTERVENTION IN RESOURCE-CONSTRAINED SETTINGS

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Background In 2010 two randomised trials suggested that pre-exposure prophylaxis (PrEP) products based on tenofovir, an antiretroviral drug either administered daily orally (oral PrEP) or applied topically (vaginal microbicides), significantly reduced HIV acquisition among adherent users. Behavioural studies also suggest that some PrEP users are compelled to share product with sex partners, family, or friends. Pill sharing (PS) decreases the adherence levels of the intended PrEP users and creates an uncontrolled environment for the development of drug-resistance. However PS effects on the expected population-level impact of PrEP interventions have never been assessed. Thus, we aim to evaluate the potential impact of PS on the PrEP effectiveness to prevent HIV transmission and the spread of drug-resistance in heterosexual populations in resource-constrained settings.
Methods A transmission dynamic model was used to assess the population-level impact of oral PrEP in a variety of intervention scenarios and high HIV prevalence settings. The cumulative fractions of new HIV infections prevented (CPF) and transmitted drug-resistance (TDR) are evaluated over fixed time periods under various epidemiological conditions. The influence of different factors (eg, acquisition rate, PrEP coverage, rates of resistance development) on CPF and TDR is studied through simulations, using parameter sets sampled from ranges representative of countries in Sub-Saharan Africa.
Results Without PS, a 70% effective oral PrEP intervention used by 60% of the population prevents about 52% (95% CI 49.6% to 53.8%) of all new HIV infections over 10 years (10 years CPF) if adherence is 100%. CPF increases by 7% in populations with 10% PS, assuming no efficacy reduction for those who share PrEP and reduces by 2% if the efficacy reduction for sharers (prescribers or untracked users) is 50%. However, the fraction of transmitted drug-resistance (TDR) increases 2- to 6-fold in all scenarios investigated. It depends on the success in preventing PrEP usage by HIV infected individuals.
Conclusions PS may increase the PrEP coverage level achieved in the population but it also affects the PrEP efficacy for the users who do not follow the prescribed dosing. It creates a pool of untracked users who do not receive counselling, remain hidden and unreached by the effort to avoid the PrEP usage by HIV infected individuals. This increases substantially the potential risk of drug-resistance development.