

Abstract Supplement

Oral abstracts of the 10th IAS Conference on HIV Science
21-24 July 2019, Mexico City, Mexico



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frequencies relative to their baseline (median 44.9% to 57.9%, Wilcoxon $p = 0.012$). Th17 cell frequencies 1-month post-initiation were also significantly higher compared to women randomized to Cu-IUD ($p < 0.001$) and LNG-Implant ($p = 0.038$). Activated CD38 + Th17 cells significantly increased between pre- and post-contraceptive initiation among women in the DMPA-IM arm ($p = 0.040$) but not in the other arms. There were no changes in cervicovaginal cytokine concentrations after contraceptive initiation in any arm. Vaginal bacterial diversity increased significantly among women assigned to Cu-IUD (mean Shannon index 1.22 versus 1.54, paired t-test $p = 0.025$) between baseline and post-contraceptive initiation but not for women assigned to LNG-implant or DMPA-IM. Beta diversity significantly differed between arms post-contraceptive initiation (PERMANOVA $p = 0.021$), with women assigned to Cu-IUD transitioning to more diverse bacterial communities.

Conclusions: These are the first data comparing markers of HIV-1 risk among women randomized to effective contraceptives. Our observation that DMPA-IM elicits increases in the frequency and activation status of Th17 cells, critical target cells for HIV, provides a plausible mechanism by which DMPA-IM may influence HIV transmission.

TUPDD0206LB

Quantifying the contribution of different aged men and women to onwards transmission of HIV-1 in generalised epidemics in sub-Saharan Africa: A modelling and phylogenetics approach from the HPTN071 (PopART) trial

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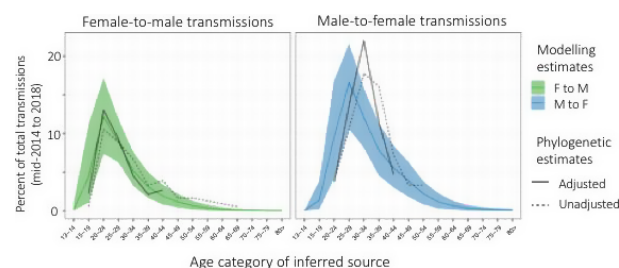
Background: Understanding the spread of HIV during HIV prevention trials has high potential to inform future intervention programming. Here, we provide a first characterisation of the residual age- and

gender-specific transmission dynamics during the HPTN 071 (PopART) trial using orthogonal methods, and investigate the potential impact of suppressing transmissions from inferred source populations.

Methods: First, epidemic predictions were made using an individual-based model (IBM) calibrated on trial data. Model parameters of the sexual network were derived from surveys on sexual behaviour. Second, model predictions were tested against phylogenetic estimates obtained with *phyloscanner* from viral short-read next-generation sequencing (NGS) data from three trial communities in Zambia.

Results: Phylogenetic analysis identified 180 probable transmission pairs and the direction of transmission between them. 62% of these transmissions were from men to women, the same as was predicted by the IBM. The phylogenetic analysis predicted men to be 5.4 years than women in male-to-female transmission (IBM predicted 5.5 years), and 3.9 years older in female-to-male transmissions (the IBM predicted 2.9 years). The age distribution of transmitters agreed with modelling predictions, more closely after adjusting for sampling bias (Figure 1). According to both the model and phylogenetics analysis, onwards transmissions peaked in 25 to 29 year old (y.o.) men and 20 to 24 y.o. women. Modelling the prevention of all transmissions from 25 to 29 y.o. men and 20 to 24 y.o. women reduced cumulative incidence over the trial period (mid-2014 to 2018) by 20% and 19% respectively.

Conclusions: Our results validate predictions of a mathematical model using phylogenetic data. These results support observations that there is a significant contribution of young people to HIV transmission in sub-Saharan Africa, especially 25 to 29 y.o. men. These results highlight that if universal testing-and-treatment (UTT) does not reach young people, and 25 to 29 y.o. men in particular, then the effect of UTT on reducing incidence may be limited.



Abstract TUPDD0206LB-Figure 1.