METHODS: We conducted a retrospective cohort study using two pregnancy cohorts at Grady Memorial Hospital (GMH): all pregnant women with deliveries from July 1, 2016 to June 30, 2018 and all deliveries by pregnant WLHIV at GMH from January 1, 2011 to June 30, 2018. The primary outcome was any HDP, including gestational hypertension (gHTN) and preeclampsia; both gHTN and preeclampsia were also examined independently. The primary exposure variables were HIV status, cART regimen [Integrase Strand Transfer Inhibitor (INSTI)-containing, Protease Inhibitor (PI)-containing (no INSTI), or non-nucleoside reverse transcriptase inhibitor (NNRTI)-containing (no INSTI or PI)], and timing of cART initiation (before or during pregnancy). We generated adjusted odds ratios (ORadj) using multivariable general estimated equation models to evaluate the association of the exposures with HDP.

RESULTS: Among 85 deliveries by 80 WLHIV and 3,556 deliveries by 3,465 women without HIV, pregnant WLHIV had no significant differences in odds of any HDP, gestational hypertension, or preeclampsia compared to HIV-negative women. Among 327 deliveries by 265 pregnant WLHIV, taking INSTI-containing regimens increased the odds of having any HDP (ORadj 4.03, 95% CI 1.98-8.19) and gHTN (ORadj 4.02, 95% CI 1.70-9.53) compared to PI-containing regimens. Timing of cART initiation was not associated with HDP.

CONCLUSION: INSTI-containing regimens were associated with increased odds of developing HDP, and specifically gHTN, compared to PI-containing regimens. Although cART initiation timing was not significantly associated with occurrence of HDP, larger studies are needed to confirm these findings.

LEARNING OBJECTIVES: Identify whether HIV seropositivity, cART regimen, or timing of cART initiation is associated with increased risk of HDP in a cohort of women at Grady Memorial Hospital.

23 Characterizing condomless sex among new contraceptive implant users who are living with or at risk for HIV in Kenya

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OBJECTIVES: Since condoms are the only method that prevents both pregnancy and HIV/STI transmission, dual method use of condoms and long-acting reversible contraceptive methods would ideally be used in high HIV prevalence settings. Therefore, our objective was to assess whether self-reported condomless sex differs between HIV-positive and HIV-negative women who recently initiated a contraceptive implant in a high HIV prevalence setting in Kenya.

METHODS: We used data from an ongoing, prospective pharmacokinetic study of HIV-positive and HIV-negative women ages 18-45 in Kisumu, Kenya, who recently initiated a levonorgestrel or etonogestrel implant. HIV-positive women were using efavirenz- or dolutegravir-based antiretroviral therapy (ART) and were virally suppressed at enrollment. At each study visit, women were asked how often they used a male or female condom during sex since the last visit. We used Poisson regression with generalized estimating equations to assess if risk of reporting condomless sex in the 12-week period following implant initiation differed by women's HIV status. In separate models, we tested for differences in trends in reported condomless sex over time using an interaction term for HIV status and study week. Multivariable models adjusted for condomless sex reported at baseline and number of prior pregnancies.

RESULTS: At the time of analysis, 91 HIV-positive and 49 HIV-negative women were enrolled and had reached week 12 of the study. Recent condomless sex was reported by 13% of HIV-positive women and 45% of HIV-negative women at baseline and 42% of HIV-positive women and 94% of HIV-negative women at week 12. Over the first 12 weeks of implant use, the risk of reporting condomless sex was 45% lower among HIV-positive women than HIV-negative women in the adjusted model (RR: 0.55, 95% CI: 0.45, 0.68). We found no evidence that changes in condomless sex reported over time differed between HIV groups (interaction p=0.47).

CONCLUSION: Report of condomless sex increased after implant insertion in both groups. However, in the first 12 weeks of implant use, HIV-positive women were significantly less likely to report condomless sex than HIV-negative women. This may be partly explained by counseling received by HIV-positive women about condom use for dual pregnancy prevention given potential drug interactions between implants and some ART.

LEARNING OBJECTIVES: Learners will be able to describe dual method use by HIV-positive and HIV-negative women newly initiating contraceptive implants in Kenya.

24 Hematopoietic type I interferon signaling controls Zika virus viremia after intravaginal exposure

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OBJECTIVES: We use a mouse model of sexually-transmitted Zika virus (ZIKV) to investigate the mechanisms of systemic and uterine spread after intravaginal exposure to ZIKV.

METHODS: Mice were treated with Depo-provera to synchronize their estrous cycles. Mice were infected intravaginally with Cambodian ZIKV by pipetting 10ul of the virus into the vagina. ZIKV infection was monitored by collecting vaginal washes, blood, and tissues for histology. ZIKV infection was detected by using ZIKV-immune rat serum to stain tissues, and RT-qPCR was used to quantify ZIKV in the blood and vaginal washes. Mice lacking the innate antiviral type I interferon signaling (IFN) are highly susceptible to ZIKV infection. Previous studies have shown that mice lacking different components of the IFN pathway including the anti-viral receptors (toll-like receptor 7 [TLR7] and mitochondrial antiviral signaling protein [MAVS]), the transcription factors upstream of IFN (Interferon regulatory factor 3 [IRF3] and IRF7), and the IFN receptor (IFNAR) were infected. Bone marrow chimeras lacking IFNAR in the stroma or hematopoetic compartment were also infected.

RESULTS: ZIKV infects vaginal epithelial cells in all mice tested, including those with intact IFN signaling which are highly resistant to the virus. It spreads to vaginal submucosal cells in mice lacking IFNAR in the hematopoetic compartment. This spread to the submucosal cells in the vagina correlates with viremia and infection of the uterine submucosa.

CONCLUSION: While ZIKV is primarily a mosquito-borne virus, it can also be transmitted by sexual exposure. We investigate the mechanisms by which interferon signaling restricts ZIKV infection after vaginal exposure. We show that interferon responsiveness of the hematopoietic compartment is essential for blocking ZIKV spread to the vaginal submucosa, uterine submucosa, and blood. These studies provide insight into how sexual exposure to ZIKV leads to systemic infection.

LEARNING OBJECTIVES: Learners will be able to identify the role of interferon signaling in blocking systemic ZIKV spread after vaginal exposure.