

S04.3 WHAT HAS EBOLA VIRUS TO DO WITH SEXUALLY TRANSMITTED INFECTIONS?

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Ebola virus could play both direct and indirect roles in the epidemiology and control of sexually transmitted infections (STI). The world's largest outbreak of Ebola virus had caused more than 23,000 cases and 11,000 deaths in Guinea, Liberia and Sierra Leone by the end of July 2015. Control efforts have largely interrupted transmission, but new endogenous cases have emerged in Liberia, which had been declared Ebola-free. And new cases continue to be reported in Guinea and Sierra Leone. First, the direct role of sexual transmission in sustaining Ebola virus transmission is intriguing but unknown. Sexual transmission of Ebola virus from a male survivor to a female partner in Liberia is strongly supported by epidemiological and genome sequencing data, but cannot be proven. Studies from earlier Ebola virus disease outbreaks have documented persistent viral shedding in semen and vaginal secretions. Ongoing research is examining the shedding, persistence and clearance of Ebola virus in genital fluids in more detail. Second, the Ebola virus disease outbreak has devastated the economy, diverted resources from already weak public health systems and disrupted social structures. Indirectly, the social and economic consequences of the epidemic are likely to have favoured the spread of known STI and HIV in the most heavily affected countries. This presentation will review the strengths and weaknesses of the evidence about Ebola virus as an STI and about the public health impact of the outbreak on STI and HIV control.

S04.4 TRACHOMA STRAINS IN INDIGENOUS AUSTRALIAN POPULATIONS, VARIANTS OF UROGENITAL CHLAMYDIA TRACHOMATIS

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Chlamydia trachomatis causes both urogenital tract (UGT) and ocular infections. One class of ocular infection is trachoma. Trachoma has a distinctive pathology, encompassing repeated rounds of conjunctivitis, which can lead to chronic inflammation, corneal scarring and blindness. It has long been that while strains that cause UGT infections are essentially all able to cause conjunctivitis, the strains that cause trachoma are rarely associated with UGT infection. Previous genetic studies have shown that the trachoma strains form a single evolutionary lineage within the *C. trachomatis* species.

This presentation will encompass recent advances in the understanding of the evolutionary structure and dynamics of *C. trachomatis*. Much of the work described was directed towards understanding better how service providers should respond to instances in which UGT specimens from young children are found to be *C. trachomatis* positive. While this is regarded as indicative of sexual abuse, a conceivable mechanism by which this can arise is autoinoculation from an ocular *C. trachomatis* infection. This is particularly relevant to remote northern and central Australia, where trachoma still exists, and where the extent of child sexual abuse is contentious and politically loaded. Understanding the strains associated with trachoma in Australia

could potentially underpin procedures for determining whether or not *C. trachomatis* in a paediatric UGT specimen is derived from an ocular infection.

Genome sequences of historic trachoma associated *C. trachomatis* isolates from Northern Australia will be presented. The results do not support the established model of a single trachoma lineage. While the isolates possessed alleles of the *ompA* gene previously associated with trachoma strains, these appeared to be recombined into multiple non-trachoma-associated lineages. Furthermore, there was evidence for acquisition of *pmpEFGH* alleles similar to those found in trachoma lineages. It was concluded that *ompA* and *pmpEFGH* may be functionally associated with trachoma pathology.

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S05 - STIs in vulnerable and high risk populations in the USA

S05.1 ADOLESCENT CHLAMYDIA RATES ADJUSTED FOR SEXUAL EXPERIENCE AND ACTIVITY

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Rates of reported cases of chlamydia, a sexually transmitted infection, are high among adolescent women in the United States, in particular non-Hispanic black teens. Fewer than half of female teens have ever had sex and are not at risk. We adjusted national chlamydia case rates for sexual behaviour and examined differences in adjusted rates by race. We used chlamydia case data reported to CDC, census population counts, and data from the 2002, 2006–2010, and 2011–2013 National Survey of Family Growth to calculate the adjusted rates for those who have had sex (sexually experienced) and rates for those who had sex in the past year (sexually active) across race groups for female teenagers. Overall, the rates of chlamydia are higher when adjusted for sexual behaviour. The disparity in female rates for blacks relative to whites is reduced when adjusted for sexual behaviour. For example, in 2006–2008, the black to white chlamydia rate ratio decreased from 6.7 to 3.0 after adjusting for sexual experience. From 2002–2013, decreases in female black/white disparities were more pronounced after adjusting for behaviour: 6.4 to 5.3 (crude) vs. 5.1 to 3.5 (adjusted for sexual experience) and 5.9 to 3.5 (adjusted for sexual activity). From 2002–2013, the female Hispanic to white rate ratio also decreased, but was less affected by adjustment for sexual activity: 2.1 to 1.4 (crude) vs. 2.5 to 1.5 (adjusted for sexual experience) vs. 2.7 to 1.7 (adjusted for sexual activity). The impact of adjustments can be attributed to: decrease in proportion of sexually experienced/active blacks; increase in proportion of sexually experienced/active Hispanics; and stable proportion in whites. Adjustments for sexual behaviour attenuate the chlamydia rate disparity between blacks and whites, and not adjusting for behaviour underestimates the reduction in disparities over time. Black teens remain disproportionately burdened by chlamydial infections.