can be indistinguishable, and correct identification of the etiology is essential for patient management. PCR is the gold standard for identification of herpes viruses, and PCR can also be used for direct detection of TP from genital lesions. Given the recent global resurgence of syphilis, early diagnosis using PCR is an important tool to supplement serology-based diagnosis of syphilis.

Methods The PlexPCR® VHS assay (SpeeDx) has been developed to detect and differentiate HSV-1, HSV-2, VZV and TP 211 samples (157 positive and 54 negative) were collected from Melbourne Sexual Health Centre (Victoria, Australia) from January-April 2018. Samples consisted of genital, anal/rectal, oral and non-genital swabs. The performance of the assay was evaluated at the Victorian Infectious Diseases Reference Laboratory (Victoria, Australia) and compared to reference results from in-house qPCR tests (HSV-1/HSV-2/VZV/CMV multiplex and TP singleplex). TP detection was also compared to serology results.

Results The sensitivity/specificity of each target compared to in-house qPCR was 100%/99.4% for HSV-1, 96.0%/98.8% for HSV-2, 100%/100% for VZV and 100.0%/100.0% for TP. Analysis of TP PCR results compared to serology are still pending.

Conclusion Molecular diagnosis of genital lesions using PlexPCR VHS allows rapid identification of pathogens with high sensitivity and specificity, enabling appropriate patient management.

Disclosure No significant relationships.

Background Chlamydia trachomatis is an obligate intracellular bacterium and is the most common notifiable infection in the United States. It spends its entire developmental cycle in a membrane bound cytosolic vacuole termed the inclusion, which protects it from otherwise deleterious host innate immune responses. Interferon gamma (IFNγ) plays a critical role in the clearance of Chlamydia in vitro and in vivo, at least in part by inducing cell-autonomous immunity in infected epithelial cells. Chlamydia muridarum, a rodent pathogen with high genomic synteny to C. trachomatis, is completely susceptible to human cell-autonomous immune responses in vitro. In contrast, C. trachomatis is highly resistant to these IFNγ-induced responses. In published coinfection experiments, infections cohabited by these species are resistant to recognition by cell-autonomous immunity, suggesting that C. trachomatis has evolved active mechanisms to evade recognition by host cytosolic immune surveillance. These mechanisms are completely unknown.

Methods To identify chlamydial genes that may be involved, we have taken advantage of a previously generated library of interspecies chimeras, each of which has a genome that is predominately C. trachomatis serovar L2 with discrete regions of C. muridarum genes recombined in (range = 12–131 recombined genes in each individual chimera). We have used these chimeras in an initial screen looking for ubiquitin recruitment to inclusion membranes—an established marker of cell-autonomous immunity recognition.

Results We have identified four chimeras that are ubiquitinated following IFNγ stimulation. These four have zones of recombination overlapping with one another, providing us with 11 candidate genes.

Conclusion This outcome highlights the utility of our chimera library, especially when used to identify genetic factors underlying phenotypes for which C. trachomatis and C. muridarum are disparate. Future characterization of the candidate genes in this screen will identify chlamydial virulence factors that aid in immune evasion of IFNγ-induced host responses, and may inform design of future vaccines.

Disclosure No significant relationships.