Methods To develop a genital lesion assay, the syphilis target was added to our existing HSV-1, HSV-2 and VZV assay (also containing a DNA extraction/amplification control) in a single-well multiplex qPCR. The performance of the assay was evaluated on 90 genital specimens for which in-house PCR results for syphilis had been determined.
Results The genital lesion assay showed robust performance in multiplex with sensitive detection to 10 copies for all targets. The multiplexed assay detected 54/57 syphilis positives, corresponding to a sensitivity and specificity of 94.7% and 100.0%, respectively. The assay also detected 4 HSV-1 and 2 HSV-2 infections (2 and 1 syphilis co-infections, respectively).
Conclusion The lesion assay offers simultaneous detection and differentiation of pathogens that cause genital lesions. In response to the current emerging syphilis outbreak, this assay could provide a rapid and effective method of determining the infectious agent responsible for genital lesions, supporting earlier detection and rapid treatment to reduce morbidity or worse outcomes.

P1.34 DONOR OF MOSAIC PENA GENE OF CEFTRIAXONE RESISTANT *NEISSERIA GONORRHOEAE* FC428 AND GU140106

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Introduction Gonorrhoea is a global health concern because *N. gonorrhoeae* has now acquired resistance to antibiotics including the 3rd generation cephalosporin, ceftriaxone. Mosaic structure in penicillin-binding protein 2 gene (*penA*) is the main cause of ceftriaxone resistance which is formed by natural transformation of DNA of ceftriaxone-resistant (CRO^R) commensal *Neisseria* spp. However, none of the previous studies has fully elucidated the source of transformed DNA. In this study, we show that genetic comparison between CRO^R-*N. gonorrhoeae* strains (GU140106 and FC428) and CRO^R – *Neisseria* commensals.
Methods CRO^R-*Neisseria* commensals were isolated from pharyngeal swabs. Genome sequence was obtained by MiSeq, Illumina, and the sequence of *penA* flanking region (*mraW-NGO*1540-*penA-murE-dcaA*) was obtained using the *N. gonorrhoeae* FA1090 as a reference sequence. Alignment of the compared sequences were prepared by CLUSTALW. Species identification of *Neisseria* commensals was performed using phylogeny constructed by 53 ribosomal proteins (*rps*) sequences.
Results Based on the sequence similarity of *penA* gene, two CRO^R-*Neisseria* commensals were speculated to be the origin of each mosaic *penA* of GU140106 and FC428. Both of the candidates were *N. cinerea* according to the *rps* phylogeny. The DNA region including *penA* originated from *N. cinerea* which consist the part of mosaic *penA* in the CRO^R-*N. gonorrhoeae* was about 1.3 kb including of parts of *penA* and *murE*. Further analysis revealed that mosaic *penA* of GU140106 occurred by recombination between CRO^R-*N. cinerea* and CRO^S -*N. gonorrhoeae* which the NG-MAST type was same with GU140106. The mosaic *penA* of FC428 was also thought to emerged in the same way with GU140106. However, there was a limitation because of the lack of CRO^S-*N. gonorrhoeae* that has the same NG-MAST type with FC428.
Conclusion The two CRO^R-*N. gonorrhoeae*, GU140106 and FC428, might acquire resistance by homologous recombination with *Neisseria* commensals, and *N. cinerea* might be the donor of mosaic *penA* which causes resistance to ceftriaxone in *N. gonorrhoeae*.

P1.35 SURFACE MODIFIED SOLID LIPID NANOPARTICLES FOR THE TARGETED DELIVERY TO BRAIN: MANAGEMENT OF HIV-1 ASSOCIATED DEMENTIA

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Introduction HIV-Associated Dementia (HAD) is a significant neurological complication which occurs years after the acute viral sero-conversion reaction responsible for progressive Immuno-suppression and high viral loads. Many patients infected with HIV-1 suffer cognitive impairment ranging from mild to severe HAD. With Present available treatment system, there is no satisfactory treatment for HAD available.
Methods In this study, nifedipine loaded solid lipid nanoparticles (SLN) were developed for targeting drug into CNS, to block the apoptosis by HIV-1 virus. This would decrease the process of neurodegeneration and increase survival time of neuronal cells. Also, this targeted delivery to brain will minimise the systemic effect of nifedipine, avoiding its delivery peripherally.The uncoated SLN were prepared by Solvent Injection Method and coated with tween 80 and Lyophylized. Shape and surface morphological studies were done by Scanning and Transmission Electron Microscopy (TEM). The *in-vitro* release profile of entrapped drug was studied using dialysis membrane. *Ex-vivo* studies consisted of DNA fragmentation followed by *in-vivo* studies.
Results The SEM and TEM images show smooth and spherical surface of SLN. I*n-vitro* release profile of drug shows more than 90% of drug release in 48 hours. DNA fragmentation was determined in presence and in absence of gp120 mimicking agent which shows no DNA fragmentation thus developed carrier system works properly in releasing drug and blocking apoptosis in cortical cells. Fluorescence microscopy shows qualitative uptake and localization pattern of coated SLNs in brain.
Conclusion: *In-vitro* and *in-vivo* studies results shows more specific delivery of Nifedipine to Brain. DNA Fragmentation and Cell Viability studies shows dementia blocking activity on brain cells. Brain specific delivery of Nifedipine could reduce the dose and potential systemic side effects, thus providing site specific delivery to brain. Thus, CNS delivery of these Nifedipine loaded SLNs via Intra-venous delivery will also open new opportunities for other Anti-Retroviral drug delivery to brain.