Introduction

Urine is the specimen of choice for CT and NG molecular testing from males and many of the next generation testing platforms are not FDA approved for use with urethral swabs. Urethral swabs remain valuable in research settings that may require that the specimen is collected in a manner preserving organism viability. The goal of this study was to assess the Abbott m2000 Realtime (m2000), and Roche COBAS 4800 (c4800) systems for the identification of CT and NG in urethral swabs collected in chlamydia transport medium (CTM).

Methods

Archived male urethral swabs collected in CTM from STD clinic attendees were tested. These specimens were originally tested for CT and NG on a platform FDA-approved for urethral swabs collected in CTM (Roche COBAS Amplicor, (cAMP)). Two hundred μl of CTM was added into each manufacturer’s collection device. This was subsequently tested on the m2000 and c4800 systems according to the manufacturer’s instructions. CT and NG results obtained from each platform were compared to the original cAMP results to determine sensitivity and specificity. Agreement between platforms was measured by calculating the kappa coefficient.

Results

One hundred urethral swab specimens were available for testing (28 CT and 25 NG positives). When compared to results obtained with the cAMP, the sensitivity and specificity of the m2000 for the detection of CT was 100% and 98.6%, respectively; the sensitivity and specificity on the c4800 was 96.4% and 98.6%. For NG, the sensitivity and specificity of the m2000 assay was 100% and 98.7%; the c4800 assay was 100% sensitive and specific. Agreement was excellent for both platforms when compared to cAMP with kappa scores of >0.95 for CT and >0.97 for NG.

Conclusion

The m2000 and c4800 platforms have similar performance characteristics to cAMP for the detection of CT and NG using urethral swabs in CTM. This study will provide investigators with additional options when designing protocols that require the preservation and ability to recover viable organisms from men.

HPV IS ASSOCIATED WITH AN ALTERED METABOLOMIC PROFILE IN THE VAGINAL TRACT

Introduction

Recent studies have reported associations between bacterial vaginosis (BV) and cervicovaginal HPV. To obtain further insight into this relationship, we examined the vaginal microbiota and metabolome of women who were HPV+ and HPV-.

Methods

Thirty-nine women self-collected mid-vaginal swabs that were profiled for bacterial composition by 16s rRNA gene amplicon sequencing, metabolites by both GC/MS and LC/MS-based metabolomics and 37 types of HPV DNA with the Roche HPV Linear Array genotyping test. Data were analysed by multiple linear regression controlling for confounding factors, as well as principal components analysis, partial least squares discriminatory analysis and linear discriminant analyses. All reported results have an adjusted p-value (q-value) <0.05.

Results

Vaginal microbiota were clustered into community state types CST-I (L. crispatus-dominated), CST-III (L. iners-dominated) and CST-IV (low-Lactobacillus/BV-associated taxa). Overall, HPV+ women had higher polyamine and phospholipid concentrations than HPV- women in a model which controlled for CST and smoking status. Significant differences in metabolomic profiles of HPV+ and HPV- women were also evident in each stratum of CST. Among women who were CST-III, HPV+ women had higher concentrations of biogenic amines and glycogen-related metabolites compared to HPV- women. Within CST-IV, HPV+ women had lower concentrations of glutathione, glycogen, and phospholipid-related metabolites than HPV- women. Women with high-risk HPV strains had lower concentrations of amino acids, lipids and peptides compared to women who were hrHPV-.

Conclusion

Detection of HPV was associated with altered vaginal concentrations of biogenic amines, glutathione and lipid-related metabolites. Reduced glutathione and oxidised glutathione have known associations with HPV and may be representative of increased oxidative stress and total glutathione depletion. Elucidating a causal relationship between microbial metabolites associated with increased oxidative stress and HPV infection warrants further investigation.
for the first immunisation Freund’s complete adjuvant was used while for the second to fifth immunisation we used Freund’s incomplete adjuvant, in the sixth week only the antigen was used. At week 0, 3 and 6, rabbits were bled for the evaluation of the immune response by the indirect ELISA. The final bleeding was performed and the serum obtained was stored at −20°C until use. For the purification of the antibodies we use a protein A purification kit and the integrity of the immunoglobulins was verified by electrophoresis, the titration of the obtained polyclonal antibody was performed by indirect ELISA. We evaluated the antibody by Western blot, Dot blot and inhibition of hemagglutination using as antigen the ATCC 14018 of G. vaginalis. We showed that the antibody recognises the VLY of ATCC 14018 of G. vaginalis. To corroborate that the antibody inhibited erythrocyte lysis, hemagglutination inhibition assays were performed and we showed that the use of this antibody decreased the cell lysis in around 80%.

**Conclusion** We produce a polyclonal antibody against the VLY of G. vaginalis capable of inhibiting the erythrocyte lysis. This antibody will be useful in investigating the role of VLY in the pathogenesis of G. vaginalis during the development of BV.

**Research** Here we demonstrate that Bb-Tp0751 adheres to HUVECs under stationary conditions. The laminin receptor (LamR) was identified as an endothelial receptor for Tp0751. LamR is a brain endothelial receptor for other neurotropic invasive pathogens, including Neisseria meningitidis. Current investigations will validate the Tp0751-LamR interaction and characterise the functional outcomes of Tp0751 adhesion to endothelial cells.

**Conclusion** These investigations reveal the mechanics of T. pallium attachment to endothelial cells, the fundamental step in the process of T. pallidum vascular dissemination. A complete understanding of this process will provide opportunities to prevent T. pallidum attachment to the host vasculature to facilitate syphilis vaccine development.

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**P1.29 ATTACHMENT OF THE SYPHILIS SPIROCHETE, Treponema pallidum, TO THE VASCULAR ENDOTHELIUM**

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**Introduction:** Treponema pallidum is the causative agent of venereal syphilis, a human-specific sexually transmitted infection characterised by multi-stage disease and diverse clinical manifestations. T. pallidum undergoes rapid hematogenous dissemination, accessing distant organ sites and penetrating tissue, placental, and blood-brain barriers. Tp0751 is an adhesion that interacts with the host vasculature and mediates bacterial adherence to endothelial cells under shear flow conditions. This study explores Tp0751-mediated adhesion to the vascular endothelium.

**Methods** Tp0751, expressed in a non-infectious model spirochete [Borrelia burgdorferi (Bb-Tp0751)], was assessed for a gain-of-function adhesion phenotype using attachment assays. Interaction specificity was probed with competitive inhibition studies using synthetic peptides of Tp0751 host-binding regions. Affinity chromatography coupled with mass spectrometry was used to identify endothelial receptors for Tp0751. Membrane receptors isolated from human umbilical vein endothelial cells (HUVECs) were incubated with Tp0751-affinity columns and interacting proteins were identified with mass spectrometry.

**Results** Here we demonstrate that Bb-Tp0751 adheres to HUVECs under stationary conditions. The laminin receptor (LamR) was identified as an endothelial receptor for Tp0751. LamR is a brain endothelial receptor for other neurotropic invasive pathogens, including Neisseria meningitidis. Current investigations will validate the Tp0751-LamR interaction and characterise the functional outcomes of Tp0751 adhesion to endothelial cells.

**Conclusion** These investigations reveal the mechanics of T. pallium attachment to endothelial cells, the fundamental step in the process of T. pallidum vascular dissemination. A complete understanding of this process will provide opportunities to prevent T. pallidum attachment to the host vasculature to facilitate syphilis vaccine development.

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**P1.30 IN VITRO ACTIVITY OF GEPOTIDACIN AND OTHER ANTIMICROBIALS AGAINST MYCOPLASMAS AND UREAPLASMAS**

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**Introduction** Mycoplasma and Ureaplasma spp. are important pathogens of the respiratory and urogenital tracts. Antimicrobial resistance limits treatment options. Gepotidacin (GEP), a novel triazacacenaphthylene topoisomerase II inhibitor that inhibits DNA replication by a mechanism and target distinct from fluoroquinolones was tested against 85 isolates of Mycoplasma pneumoniae (Mp), Mycoplasma hominis (Mh), Mycoplasma genitalium (Mg), Ureaplasma parvum (Up), and Ureaplasma urealyticum (Us) in comparison to azithromycin (AZI), clindamycin (CLI), tetracycline (TET), levofloxacin (LEV), and moxifloxacin (MOX). Organisms tested included strains known to be resistant to TET, LEV, and/or AZI. This work was supported by GSK and funded through OTA HHSO100201300011C with HHB/BARD.

**Methods** MICs were determined using broth microdilution in accordance with Clinical and Laboratory Standards Institute Guidelines.

**Results** GEP was active against 25 Mp, MIC range 0.032–0.125 μg/ml, including 5 that were AZI-resistant, with MIC90 (0.125 μg/ml), equivalent to MOX. GEP was active against 10 Mg. MIC90 (0.032 μg/ml) was 4-fold < MOX. GEP MICs against 25 Mh ranged from 0.5 to 2 μg/ml with MIC90=2 μg/ml, making it less active than other agents, including MOX (MIC90=0.125 μg/ml), with exceptions of 1 LEV and 2 TET-resistant organisms, for which GEP MICs were unaffected. GEP was less active against 25 Ureaplasma spp. (MIC range 1–8 μg/ml), MIC90=8 μg/ml. There was no effect on GEP MICs in 9 Ureaplasma spp., with resistance to LEV, AZI, and/or TET. GEP minimum bactericidal concentrations for 4 isolates of Mg, 4 Mh, 4 Mp, 3 Us, and 1 Up were >3 dilutions > MICs, indicating bacteriostatic effect.

**Conclusion** GEP warrants further study to treat infections due to Mycoplasma spp., particularly organisms resistant to other antimicrobials as it was active against isolates resistant to AZI, TET, LEV, and/or MOX.