DETECTION OF GENITAL MYCOPLASMAS IN WOMEN VISITING THE INFERTILITY CLINIC OF AN ACADEMIC HOSPITAL, PRETORIA, SOUTH AFRICA

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Introduction Sexually transmitted infections (STIs) continue to be a significant public health problem with a high burden in women of reproductive age. Rates of Chlamydia trachomatis, Neisseria gonorrhoeae and Trichomonas vaginalis are frequently tested for and rates of infection are generally high in African settings, but the prevalence of other genital STIs is largely unknown. The aim of this study was to determine the prevalence of genital mycoplasmas (Mycoplasma genitalium, M. hominis, Ureaplasma parvum and U. urealyticum) in women visiting the infertility clinic of a tertiary academic hospital in South Africa.

Methods In this pilot evaluation self-collected vaginal swabs were obtained from 51 women visiting the infertility clinic. The genomic DNA was extracted from the swabs using the ZR Fungal/Bacterial DNA Miniprep (Thermo Scientific, USA) and analysed using the Anyplex II STI-7 (Seegene, Korea) real-time PCR assay for the simultaneous detection and identification of seven STIs including the four mycoplasma species.

Results The real-time PCR assay detected the following genital mycoplasmas and co-infections in the 51 women: U. parvum (55% (28/51)), M. hominis (20% (10/51)) and U. urealyticum (16% (8/51)); none of the specimens tested positive for M. genitalium. Among the nine patients where mixed infections were observed, M. hominis and Ureaplasm spp. were frequently detected together (67% /6/9). In addition to the mycoplasmas, one woman tested positive for C. trachomatis; N. gonorrhoeae and T. vaginalis were not detected.

Conclusion This pilot study demonstrated an unexpectedly high rate of genital mycoplasma infections among women visiting an infertility clinic. The burden of genital mycoplasma infection is largely unknown and warrants further investigation, in particular with regards to the prevalence and clinical significance in different population groups.

Support: Anyplex II STI-7 kits provided by Seegene, Korea
LACTIC ACID EXERTS ANTI-GENOMIC CHARACTERISATION OF URETHRITIS - USA

**Methods**

A 3D model of A2EN cervical epithelial cells was used to determine the impact of lactic acid on cell proliferation. We tested if low proliferation affects infection. A2EN cells were exposed to lactic acid, proliferation chemical inhibitors or LCM followed by infection with *C. trachomatis* L2. Proliferation and infectivity were evaluated by microscopy.

**Results**

At pH 4, d-lactate and LCMs from high d-lactate producing vaginal *Lactobacillus* spp. afforded maximal protection compared to l-lactate. Interestingly, high infectivity was observed with HCl at pH 4, indicating that pH alone is not responsible for this protection. Exposure to d-lactate or LCMs reduced cell proliferation. Chemical cell proliferation inhibitors dramatically reduced *C. trachomatis* infectivity.

**Conclusion**

These results suggest a differential role for vaginal *Lactobacillus* spp. in protecting against *C. trachomatis* infections and potentially other STIs. This protection is driven by the production of d-lactate, which acts on epithelial cells by inhibiting cell proliferation, which appears to be required for infection.

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**P1.58 LACTIC ACID EXERTS ANTI-CHLAMYDIA TRACHOMATIS ACTIVITY ON THE EPITHELIUM BY REDUCING HOST CELL PROLIFERATION**

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**Introduction**

Epidemiological studies have demonstrated that the vaginal microbiota can significantly impact the risk of acquiring sexually transmitted infections. The human vagina often contains *Lactobacillus* spp., which produce lactic acid and create an acidic environment (pH 3.5–4) thought to reduce vaginal STIs. Unlike high d-lactate producers, *Lactobacillus* spp. that produce low amounts or no d-lactate, while achieving low pH do not reduce *Chlamydia trachomatis* infectivity. Further, exposure to culture supernatants from d-lactate producing *Lactobacillus* spp. reduces epithelial cell proliferation. We tested if low proliferation affects infection.

**Methods**

A 3D model of A2EN cervical epithelial cells was exposed to lactic acid (D, L or D/L) at concentrations that produce pH 7, 5.5 and 4 or to several *Lactobacillus* spp. conditioned media (LCM) and infected with *C. trachomatis* serovar L2. Lysates from these A2EN cells were used to infect HeLa cells, and IFUs counted to determine infectivity. 2D A2EN cells were exposed to lactic acid, proliferation chemical inhibitors or LCM followed by infection with *C. trachomatis* L2. Proliferation and infectivity were evaluated by microscopy.

**Results**

At pH 4, d-lactate and LCMs from high d-lactate producing vaginal *Lactobacillus* spp. afforded maximal protection compared to l-lactate. Interestingly, high infectivity was observed with HCl at pH 4, indicating that pH alone is not responsible for this protection. Exposure to d-lactate or LCMs reduced cell proliferation. Chemical cell proliferation inhibitors dramatically reduced *C. trachomatis* infectivity.

**Conclusion**

These results suggest a differential role for vaginal *Lactobacillus* spp. in protecting against *C. trachomatis* infections and potentially other STIs. This protection is driven by the production of d-lactate, which acts on epithelial cells by inhibiting cell proliferation, which appears to be required for infection.