Background *Trichomonas vaginalis* (TV) infection is a prevalent parasitic STD that may increase the risk of acquiring other STI including HIV. Trichomoniasis has been increasingly reported in older female populations. Detection methods include the InPouch culture system and GenProbe's APTIMA *Trichomonas vaginalis* nucleic acid amplification test (NAAT). Both systems detect TV (viable or genetic material, respectively) from vaginal swabs. In addition to comparing TV detection technologies, this study attempted to look at infection status in younger versus older macaque populations.
Methods 24 sexually mature female pigtailed macaques were challenged with TV (ATCC 50148, human isolate), and followed weekly for five weeks, before metronidazole treatment. The reproductive age span for pigtailed macaques is approximately 4 to 18 years of age. Twelve animals aged 4–7 and twelve animals older than 13 years were enrolled. Paired vaginal swabs were collected weekly from each animal for culture and NAAT detection assays.
Results Each TV-challenged macaque developed trichomoniasis. Of 199 matched samples (culture and NAAT), 13 had discrepant results. Six of these were likely due to false culture results. Four samples represented the transition time from positive to negative status in three animals. It is plausible that organisms detected by NAAT were no longer viable (thus culture-negative). There is no obvious explanation for the three remaining discrepant results.

Trichomoniasis infection was self-limited (resolved prior to metronidazole treatment) in eight animals: two older and six younger macaques. Two of these younger macaques experienced intermittent discrepant results after testing negative by both methods for two consecutive weeks.
Conclusions NAAT detection appears to be more sensitive and less prone to erroneous results in this laboratory's experience. There may be a trend for younger animals to self-clear TV infection faster than older animals, which might explain the increased TV infection rates noted in older women.

P1.044 A CROSS-SECTIONAL STUDY COMPARING SERUM VITAMIN D LEVELS IN HUMAN IMMUNODEFICIENCY VIRUS-INFECTED AND -UNINFECTED INDIVIDUALS

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Background Since the evolution of human immunodeficiency virus (HIV) infection, every effort has been made to increase the survival and quality of life of the infected patients. In this regard, metabolic/nutritional derangements are among the most important problems. So, we planned this study to evaluate the serum level of vitamin D as an essential micronutrient with known immunologic roles in HIV-infected patients and compare it with that of HIV-uninfected individuals, and to identify risk factors for possible hypovitaminosis D in the former group.
Methods This was a cross sectional study on 35 HIV-infected patients (cases) and 35 HIV-uninfected individuals (controls). All the participants were > = 18 years-old. Patients with conditions, like tuberculosis, or using drugs, with known effects on serum vitamin D level were excluded. The control group was matched for age, sex, nutritional habits and occupation (exposure to sunlight). HIV infection was confirmed in the cases with 2 positive HIV ELISAs and then a positive HIV Western Blot test. Serum level of vitamin D was measured by ELISA method. Chi-square and independent T tests were used for analysis of data.
Results Of the HIV-infected patients, 9 people (25.7%) were receiving antiretroviral therapy. Twenty-three HIV-infected patient (65.7%) had hypovitaminosis D (vitamin D < 30 ng/mL), comparing with only one person (2.8%) in controls (p value < 0.05). The mean level of vitamin D in the serum was significantly lower in HIV-infected patients (25.78 ng/mL) compared with HIV- uninfected individuals (41.4 ng/mL) (p value < 0.008). There was no association between hypovitaminosis D and sex, age, body mass index, CD4+ cell count, haemoglobin level and antiretroviral therapy in HIV-infected patients.
Conclusion This study shows the possible association of HIV-infection with vitamin D deficiency, and thus the evaluation of this group of patients for hypovitaminosis D seems reasonable.

P1.045 ZINC IONOPHORES INHIBIT HERPES SIMPLEX VIRUS TYPE 1 AND 2 REPLICATION THROUGH DYSREGULATION OF UBIQUITIN-PROTEASOME PATHWAY

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Background Herpes simplex virus types 1 and 2 (HSV-1 and HSV-2) are among the most prevalent human pathogens in both industrialised and developing regions. In the current study, we reported that two zinc ionephores, pyrrolidine dithiocarbamate (PDTC) and pyrithione (PT) could inhibit herpes simplex virus type 1 and type 2 (HSV-1 and HSV-2) replication.
Methods Viral replication was evaluated via detecting HSV late gene product (Glycoprotein, gD) using In-cell Western. NF-κB, MAPK activation, viral immediate-early genes expression and profile of cellular ubiquitin-conjugates were determined by Western blot. Protein localization was investigated via immunofluorescence staining. Viral protein expression on mRNA level was quantified by real-time-PCR. 26S Proteasome activation was determined using fluorogenic substrate, Suc-LLVY-AMC.
Results PDTC and PT inhibited HSV-1 and HSV-2 gD expression and the production of viral progeny, which was dependent on zinc ion. Further studies showed that these two compounds suppressed the HSV immediate-early gene, the infected cell polypeptide 4 (ICP4) expression, but had less effect on ICP0. HSV infection could interact with cellular ubiquitin-proteasome system (UPS) and cause loss of high molecular weight ubiquitin-conjugates. It was found that PDTC and PT could interfere UPS, leading to the inhibition of HSV-2-induced IκB-α degradation to prevent NF-κB activation and enhanced PML stability in nucleus. However, PDTC and PT did not show direct inhibition of 26S proteasome activity. Instead, these two zinc ionphores induced import of extracellular zinc ions into cells, which facilitated dysregulation of proteasome function and accumulation of intracellular ubiquitin-conjugates. Other evidence was that the inhibitors of ubiquitin activating enzyme E1 and deubiquitinase also inhibited HSV replication, implying that UPS was required for effective replication of HSV-1 and HSV-2
Conclusion Homostasis of ubiquitin cycle and UPS were critical for HSV gene expression and replication and that the ubiquitin-proteasome pathway is a potential drug target for HSV infection.

P.02 - Clinical Sciences Track

P2.001 PERFORMANCE OF THE HOLOGIC GEN-PROBE APTIMA ASSAYS AND PANTHER™ INSTRUMENTATION FOR THE CONFIRMATION OF *NEISSERIA GONORRHOEAE* IN GENITAL AND NON-GENITAL SAMPLES

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