A (mtTFA) and indirect role via up-regulation of the p53R2 expression. Recently more than 6 kinds of p53 isoform have been reported and they played the different role in DNA repair pathways, cell-cycle checkpoints and cell apoptosis. p53 and its different p53 isoforms play what kind of role in mitochondrial toxicity induced by NRTIs is unclear. In this study, we identified the role of wild type p53 and its isoforms (N40P53 and N133P53) in mitochondrial toxicity induced by AZT and oxidative stress.

In this study we treated the A549 p53+/−, H1299 N40 P53+/+, H1299 N133P53+/+ and H1299 P53−/− cell lines with 5 to 200 μM AZT for 12 hours for studying cell death and the expression of p53R2, p21 and bax via p53R2 (Bax, P21) pGL3-Luciferase reporter gene assay, real-time PCR and Western Blotting; treated cells with 30uM AZT for 5 weeks, 10 weeks and 1pM H2O2 for 1 hour/week to study the mtDNA mutation, mtDNA deletion and mitochondrial toxicity. The cells were treated with p53R2 interference RNA to study the p53R2 how regulated the DNA Pol γ capability.

The result showed that A549 p53+/− cell death was more sensitivity to high concentration of AZT. The increased cell death was detected from 120 uM of AZT and more that 80% cell were into apoptosis in 2000 μM AZT. But other three cell lines have strong tolerance to AZT toxicity. They are only less than 10% cell death in 2000 μM AZT. The results from report gene assay and real-time PCR assay suggested that wild type p53, N40 P53, N133P53 played different role in p53R2, P21 and Bax promoters. All of wild type and isoform P53 up-regulated the p53R2 expression, but isoform P53 played a domain negative regulation in Bax expression. The p21 expression was co-stimulated by wilde-type P53 and N40, N133P53. D-loop of mtDNA mutation and mtDNA quality assay showed that A549 p53+/− cell has lower mtDNA mutation rate and almost no mtDNA loss following 5 weeks AZT stress. N40 P53, N133P53 cells have lower mtDNA mutation too, but mtDNA loss is significant higher than that in A549 p53+/− cells. H1299 P53 null cells has higher mtDNA mutation rate and mtDNA loss than that in wild-type P53 and N40 P53, N133P53 isoform cells. Conclusion: Both wild type and N40 P53, N133P53 isoform p53 play protect role in AZT induced mitochondrial toxicity. The P53R2 would be involved in the central molecular pathway of p53 reduced mtDNA mutation and mtDNA loss induced by AZT.

**PP-147** Liver injury in HIV-1-infected patients receiving non-nucleosides reverse transcriptase inhibitors-based antiretroviral therapy

Zaicun Li*, Lili Dai, Yanqing Gao, Haiying Li, Xiaojie Huang, Caiping Guo, Tong Zhang, Hao Wu. Clinical center for STDs and AIDS at Beijing Youan Hospital, Capital Medical University

**Objectives:** To study the features of liver injury in HIV-1-infected patients receiving non-nucleosides reverse transcriptase inhibitors-based antiretroviral therapy (ART).

**Methods:** 75 patients receiving non-nucleosides reverse transcriptase inhibitors-based ART were retrospectively studied. The patients were divided into 2 groups: liver injury group (n=45) and non-liver injury group (n=30). The features of liver injury were analysed. The prevalence of HBV and/or HCV, hepatotoxic drugs use and NVP or EFV use were compared between two groups.

**Results:** 45 (60%) patients, 31 (68.9%) males and 14 (31.1%) females, aged 12–52 years, averaged 39±5 years, experienced at least one episode of liver injury. 40 (53.3%) co-infected with HBV and/or HCV, 42 (56%) patients had concomitant use of antituberculosis drugs, 46 (61.3%) and 29 (38.7%) received regimen containing NVP or EFV, respectively. Grade 1 liver injury were observed in 26 (34.7%) patients, grade 2 in 16 (21.3%), grade 3 in 2 (2.7%) and grade 4 in 1 (1.3%), respectively. 3 (4%) patients discontinued ART due to liver injury. There were 29 (64.4%) patients coinfected with HBV and/or HCV, 32 (71.1%) received regimens containing NVP, and 30 (66.7%) had concomitant use of antituberculosis drugs in liver injury group, respectively, significantly higher than those in non-liver injury group [11 (36.7%), 14 (46.7%) and 12 (40%), respectively. P=0.018, 0.033, 0.023, respectively].

**Conclusions:** Liver injury in HIV-1-infected patients receiving non-nucleosides reverse transcriptase inhibitors-based antiretroviral therapy was mild to moderate, those who coinfected with HBV and/or HCV, had concomitant use of antibaculosis drugs or cotrimoxazole and received a regimen containing NVP were prone to liver injury.

**PP-146** Incidence and characterization of acute HIV-1 infection among high-risk self-identified men who have sex with men in Beijing, China

Xiaojie Huang*, Haiying Li, Zhiying Liu, Caiping Guo, Yanqing Gao, Zaicun Li, Yan Fu, Tong Zhang, Dexi Chen, Xiaoning Xu, Hao Wu. Beijing YouAn Hospital, Capital Medical University

**Objective:** To investigate the HIV incidence and the baseline demographic data, clinical characteristics of acute HIV infections among self-identified men who have sex with men (MSM) population in Beijing.

**Methods:** From May 2007 to February 2009, a hospital-based cohort of HIV-uninfected MSM was established for a natural history study of HIV-1 infection. The cohort was followed every two months to determine HIV incidence by pooling nucleic acid testing (NAT) and blood specimens, cell apoptosis and cell death.

**Results:** After screening 2861 individuals, a hospital-based cohort of 1529 uninfected high-risk MSM was established. During 1147.575 person-years of follow-up, 8424 samples were screened for acute HIV infection by pooling nucleic acid testing (NAT) of third-generation enzyme immunoassay (EIA)-negative samples. The prevalence of chronic infection was 9.29% posing a challenge in HIV-negative MSM. 63 infections occurred yielding seroincidence rate of 5.49 per 100 person-years. 15 acute HIV-infected subjects were identified by pooled NAT. Median HIV RNA was 5.34 copies per milliliter (log10 viral load) (VL) =220,000.

**Conclusions:** HIV prevalence in MSM population is significantly greater than that in the general population, which reflects the current epidemic in Beijing and justifies prevention programs aimed at this group. This high-risk population may be suitable for future studies on acute HIV infection, HIV treatment, vaccine, and prevention of onward transmission strategies.

**PP-148** Simultaneous disseminated mycobacterium avium complex (MAC) and pulmonary cryptococcosis in an AIDS patient: presentation of immune reconstitution inflammatory syndrome (IRIS) requiring corticosteroid therapy

Azadeh Nasseh*, Baya Omidnia, Ladan Ahmadi. Department of Medicine; Lenox Hill Hospital; New York, NY, USA

**Introduction:** Immune reconstitution syndrome, caused by restoration of the capacity to mount an inflammatory response, can lead to morbidity and mortality in HIV patients.

**Case description:** A 38 y/o presented with fever and weight loss. HIV test was positive and blood culture grew Cryptococcus Neoformans. CD4 was 45 cells/ml and viral load (VL) 800,000 copies/ml. Chest CT revealed hilar lymphadenopathy and RLL lesion. Few weeks into treatment with Amphotericin B and subsequently Fluconazole, combination antiretroviral therapy (CART) was added to PCP and MAC prophylaxis. After 4 weeks, he presented with fever and cervical lymphadenopathy. CD4 was 322, VL 80,000. Bronchial washing showed Cryptococcus and MAC. Amphotericin B, Rifabutin, Ethambutol and Azithromycin were started. One month later fever and lymphadenopathy continued. Biopsy of a node showed necrotizing granuloma due to MAC. IRIS with Disseminated MAC was diagnosed and considering progressive...