gene which caused amino acid to change from Thr to Met at position aa118 and researched the effects of this mutation on blood HBsAg screening test.
Methods: HBsAg Gene, which was amplified by PCR, was cloned and sequenced, then mutational amino acids were recovered the wild type at aa117 or/and aa118 position. By insertion of HBsAg Genes, expressing vectors were constructed and transient expressions were committed in 293T cells. Suspensions of cell culture were collected and tested by the panel which contained ten kinds of HBsAg testing kits provided by companies.
Result: The profile of results tested by Panel were similar between express suspension of mutation HBsAg gene and donation serum and most of kits but two failed. But the profile was significant difference compared with normal quality control serum. When mutation was recovered at 117 position, the profile of results did not change. But mutation was recovered at 118 position, the profile of results changed, and all kits can effetely test this HBsAg.
Conclusion: Mutation of HBsAg at 118 position which was changed from T to M leaded to failure tests by most of kits.

PP-100 Induction of effective anti-HBV specific T cellular responses with dendritic cells that modified by a recombinant adenovirus vector expressing HBsAg and CTLA-4 ScFv

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Objectives: To investigate the mechanism and evaluate the therapeutic potential of Ad-S-ScFv transduced DCs in HBV transgenic mice.
Methods: The ScFv of CTLA-4 was ligated and the recombinant Ad-S and Ad-S-ScFv were constructed. DCs were transfected with rAd (Ad-S-ScFv, Ad-S, etc), and HBV-Tg mice were randomly assigned to receive different rAd-transduced DCs twice at 3-week intervals. The HBV specific IFN-γ^+ $CD8^+$ T cells proportion, the HBsAg-specific T cellular proliferation and cytotoxic activity of splenocytes were measured. The serum HBV markers and ALT levels, the histology and the expression of HBcAg, HBsAg in tissue samples of liver were also assessed. The expression and phosphorlation level for the key components of the intracellular signal pathway of MAPK and PI3K/Akt were detected.
Results: Compared with either DC/Ad-S or DNA immunization, DC/Ad-S-ScFv can induce much stronger type I immune responses and HBV-specific CTLs, and more significantly reduce the titer of serum HBsAg and HBV DNA, and also reduced the expression of HBcAg and HBsAg in the liver of HBV-Tg mice. The phosphorlation expression of Erk1,2 in the liver tissue of DC/Ad-S-ScFv immunized mice were significant increased, but not for the the PI3K/Akt signal protein.
Conclusion: Ad-S-ScFv transduced DCs may be a promising candidate for a CTL-based vaccine for chronic HBV infection, and the combination with other antiviral strategy could probably acquire the complete clearance of HBV DNA.

PP-101 Pattern of hepatitis B 'e' antigen (HBeAg) in hepatitis B surface antigen (HBsAg) confirmed positive cases

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Objective: The aim of this study was to explore the pattern of HBeAg status in HBsAg true positive cases attending National Reference Laboratory of Nepal.
Materials and Methods: Secondary data were collected by record review. Serum specimens confirmed for HBsAg positive cases (n=512), by neutralization method using bioKit, Spain, were subjected to semi-automatic enzyme immunoassay (EIA) GB, Taiwan for the detection of HBeAg during the period of November 2006 to February 2009. Qualitative results were calculated by cut-off formula provided in the kit insert and the produced data were analyzed.
Results: Of the true HBsAg positive (n=512) cases, 76.17% (n=390) were males and 23.83% (n=122) were females. Data showed that 29.49% (n=151) cases were found to be positive for HBeAg, among which 31.28% (n=122) were males and 23.77% (n=29) were females. Age wise distribution of HBeAg seropositivity showed 71.52% (n=108) cases were in the age group 21—50 years, the most active and productive age group in Nepal.
Conclusion: Such higher prevalence of hepatitis B virus infection in its actively multiplying phase poses a problem of greater transmission rate in the communities. As males are more mobile than females in Nepal our communities are exposed to more risk of getting hepatitis B virus infection.
Similarly, HBeAg positive mothers may give birth to babies with HBsAg positive status who ultimately become chronic carrier of hepatitis B virus.

PP-102 The study of the $CD4^+CD25^+$treg induced by human plasmacytoid dendritic cells from chronic hepatitis B *in vitro*

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Background: This study was undertaken to investigate whether PDCs are involved in the generation of a higher proportion of $CD4^+CD25^+$ Treg cells in chronic HBV infection compared with healthy people.
Methods: The amount, phenotype, and function of Treg in $CD4^+$ T cells primed by PDC from 46 chronic HBV patients, 25 healthy controls, and 10 individuals with a resolved HBV infection were studied by the ways of flow cytometry, RT-PCR, ELISA and proliferation assay.
Results: $CD4^+$T cells primed by PDC from chronic HBV patient were more effective than $CD4^+$ T cells primed by PDC from healthy controls and resolved HBV patients in suppressing the HBcAg-specific proliferation and the interferon production. The IL-10 and TGF-β1 could be also detectable in the supernatants of PDC-primed $CD4^+$ T cells. A higher percentage of Treg, defined as CD4, CD25, CD45RO, and CTLA-4-positive cells, was detected within the population of $CD4^+$ T cells primed by PDC from chronic HBV patients compared with healthy controls and individuals with a resolved HBV infection. Accordingly, $CD25^+$Treg from PDC-primed $CD4^+$ T cells displayed a high Fox P3 messenger RNA level. Depleting $CD4^+CD25^+$ Treg from $CD4^+$ T cells primed by PDC from chronic HBV patients, healthy volunteers and resolved HBV patients made PDC-primed $CD4^+$ T to lose the capability in suppressing HBV-specific T-cell.
Conclusion: PDCs from the patients with chronic HBV infection induce the generation of a higher proportion of $CD4^+CD25^+$ Treg compared with the healthy peoples.

PP-103 Abnormal expression of TGF-β1 and IGF-II associated with HBV replication in human hepatoma tissues

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Background: The abnormality of many growth factor expressions involved in the formation and development of HCC, and asscosiated with the alteration of gene methylation status. However, their molecular mechanism and expression rule remains not too clear. In the present study, we investigated the relationship