March 2005 until March 2008. The samples tested for HCV-Ab by using ELISA methods and reactive samples retested in duplicate manner and repeatedly reactive samples confirmed by RIBA methods. All data analyzed in SPSS software.
We found that 1.587%, 0.862% and 0.499% respectively of blood donors were repeatedly reactive for HCV-Ab. The confirmatory result in the same period were: 0.311%, 0.314% and 0.294% respectively. The repeatedly reactive samples were significantly reduced after change the screening kit.
Our findings showed that HCV prevalence was less than some of other regions in our country and some neighbor's prevalence. Our strategy in reducing HCV prevalence such as donor selection and educational program was great effective and meet our goal in enhancing blood safety. Establishment that strategy for other centers in our province recommended.

OL-053 Screening and identification of genes trans-regulated by HCV p7 protein with microarray assay

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Introduction: Hepatitis C virus (HCV) is a major cause of chronic hepatitis, liver cirrhosis, and hepatocellular carcinoma throughout the world. Although HCV cannot be incubated efficiently *in vitro*, several of its key features have been elucidated in the past few years. HCV, cloned successfully via molecule biological technology, is an enveloped, positive single-stranded RNA (9.6-kb) virus belonging to the Flaviviridae. The HCV genome has only one ORF which is flanked by a 5 and 3 noncoding region. The ORF encodes for a large polyprotein precursor of about 3 000 amino acid residues, and this precursor protein is cleaved by the host and viral proteinases to generate at least 10 proteins in the following order: NH2-C-E1-E2-p7-NS2-NS3-NS4A-NS4B-NS5A-NS5B- COOH. HCV proteins not only function in viral replication but also affect a variety of cellular functions. HCV p7, which is located between the E2 and NS2 proteins, is a 63 residue peptide encoded by HCV genome between 2 580–2 768 nt. Although there are studies on the genomic structure, synthesis and function of p7 protein, the role of the HCV p7 protein in the virus life cycle is not known. To gain more information on the HCV p7 protein, and provide some new clues for elucidating the potential biological function of p7, we use cDNA microarray technique to screen genes regulated by p7.
Method: To investigate the biological functions of hepatitis C virus p7, and to use cDNA microarray technique to screen genes regulated by p7. The HCV p7 coding DNA fragment was amplified with polymerase chain reaction (PCR) technique by using PBRTM plasmid containing the full length of HCV genome as the template. The expressive vector of pcDNA3.1-p7 was constructed by routine molecular biological methods. The HepG2 cells were transfected by pcDNA3.1(–) and pcDNA3.1-p7, respectively using FuGENE6 transfection Reagent. The total RNA was isolated and reverse transcribed. The cDNAs were subjected for microarray screening with 1152 cDNA probes. The expressive vector has been constructed and confirmed by restriction enzyme digestion and DNA sequencing analysis. High quality mRNA and cDNA had been prepared and successful microarray screening had been conducted. From the scanning results, it was found 1 genes were up-regulated and 22 genes were down-regulated by p7 protein of HCV. HCV p7 protein is a trans-regulator. The expression of p7 protein affected the expression spectrum of HCV infected hepatocyte.
Results: See Figures 1 and 2, and table 1.

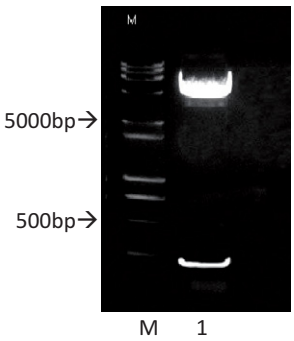

Fig. 1. Products of pcDNA3.1(–)-p7 PCR and restriction enzyme cleaved were electrophoresed in 1.0% agarose gel. Lane 1: *Eco*RI/*Bam*HI cleaved; M: DNA Marker (15,000+2,000 bp).

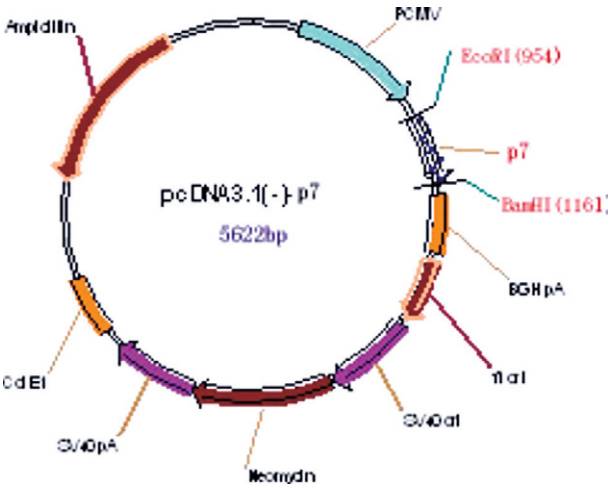

Fig. 2. Structure of expression plasmid pcDNA3.1(–)-p7.

Table 1. Trans-regulated genes by HCV p7

Accession number	Protein	Cy3/Cy5
NM_000553	Werner syndrome (WRN)	0.337
NM_002514	Nephroblastoma overexpressed gene (NOV), mRNA	0.368
AY142112	Anion exchanger SLC4A3	0.394
NM_001122	Adipose differentiation-related protein (ADFP)	0.403
AB028641	SOX11	0.406
NM_002804	Proteasome (prosome, macropain) 26S subunit, ATPase, 3 (PSMC3)	0.410
AF123320	Lymphocyte activation-associated protein	0.411
NM_015150	Raft-linking protein (RAFTLIN)	0.411
AK131542	Moderately similar to renal tumor antigen	0.405
BC052628	Armadillo repeat containing, X-linked 2	0.419
AB020706	KIAA0899 protein	0.436
NM_015270	Adenylate cyclase 6 (ADCY6)	0.436
NM_005746	Pre-B-cell colony enhancing factor 1 (PBEF1)	0.448
NM_173631	Zinc finger protein 547 (ZNF547)	0.459
BC046099	TAF15 RNA polymerase II, TATA box binding protein (TBP)-associated factor	0.473
AF370457	Ovarian epithelial carcinoma-related protein	0.476
NM_015568	PPP1R16B	0.483
NM_003419	ZNF345	0.485
NM_003260	transducin-like enhancer of split 2 (TLE2)	0.492
BC033522	p65	0.481
AK128380	FLJ46523	0.492
BX537378	C21orf4	0.469
NM_183419	RNF19	2.100

Conclusions: HCV p7 protein is a trans-regulator. The expression of p7 protein affected the expression spectrum of HCV infected hepatocyte.

OL-054 Treatment with Adacolumn in patients with hepatitis C related who have undergone kidney transplantation: Preliminary study

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Introduction: Patients who have undergone kidney transplantation (TX) and suffer from hepatic C related (HCV) cannot be treated with standard therapy (PEG-IFN combined with ribavirin) due to acute rejection risk. Furthermore, immuno-suppressive therapy facilitates progression and infection and chronic hepatopathesis. Monocytes and macrophages are known to produce extra-hepatic breeding sites and spread disease. Our aim was to lower macrophages, granulocytes, monocytes, pro-inflammatory cells and viremia levels using an extra-corporeal device:Adacolumn® (Otsuka).
Methods: The Adacolumn filter is filled with 2 mm cellulose acetate beads immersed in sterile saline solution. These carriers absorb granulocytes and monocytes/macrophages through FCR receptors. Six patients were treated in our department. All patients were affected by virale genotype 1b. Inclusion criteria: kidney transplant at under one year and HCV-RNA >800,000 copies. Patients underwent five 1-hour treatments for five consecutive days according to protocol. Kidney and hepatic parameters were evaluated as were changes in immuno-modulation (CD4, CD8) and HCV-RNA base quantities, at end of treatment and at 1 month, 2 month and 3 month follow ups.
Results: During treatment cycles and successive follow ups we observed a stabilization of kidney parameters and a non significant decrease in transaminase levels. At 3rd month follow up we observed a significant decrease in plasma HCV-RNA in 3 patients ($p < 0.01$) associated with attenuation of inflammatory phase ($p < 0.2$) and variations in immuno-modulation. Only one patient presented altered CD4+ and CD8+ where positive was observed at 3rd month. In another patient, even though immuno- modulation improved, there was no reduction in viremia.
Conclusions: The treatment was found to be safe without hemodynamic or infective complications. Considering the results this method should be used on a greater number of patients evaluating successive treatment times in case of viremia increase.