and immunize two rabbits, detected the valence of serofast by ELISA, and Western-Blotting detect specificity.

**Results**: SDS-PAGE confirmed the XP1-u protein was cytotectes expression, split the cytotectes and then renaturation. Then western blot detect on the antigenicity of XP1u protein with anti-His antibody and anti-XP1antibody. After immunize rabbit, the valence of serofast detected by ELISA was exceed 1:64000, Western-Blotting appeared specific XP1-u strap.

![Figure 1](image1.png)

**Fig. 1.** Western blot analysis of XP1u protein expressed in E. coli BL21. A. 1-7: anti-His antibody of jiane; B. 1-7: anti-XP1-u antibody of Santa cruz.

**Conclusions**: Succeed induce expression of human XP1-u recombint, the polyclonal antibody was made.

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**PP-025** Effect of hydrocamptothecin on proliferation and apoptosis of rat hepatic stellate cells in vitro

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**Objectives**: To investigate the effects of hydrocamptothecin (HCPT) on proliferation and apoptosis of rat hepatic stellate cells (HSC) in vitro.

**Methods**: Rat HSC line (HSC-T6) was incubated in medium with different concentrations of HCPT (0.008, 0.016, 0.031, 0.125, 0.25, 0.5, 1, 2, 4, 8, 16, 32 mg/L respectively) or without HCPT for 24, 48, 72 hours respectively, cell proliferation was assessed by MTT colorimetric assay. The rate of cell apoptosis was identified by flow cytometry (FCM). The morphological change of apoptosis was observed with transmission electron microscopy (TEM). DNA break was tested by agarose gel electrophoresis.

**Results**: HCPT significantly inhibited the proliferation of HSC-T6 in a dose-dependent and time-dependent manner. After HSC-T6 was treated with HCPT at different concentration of 0.25, 0.5, 1 mg/L for 24 h, FCM showed the cell apoptosis ratio was 13.46%±2.24%, 26.25%±5.65%, 47.05%±8.76% respectively, which is significantly higher compared with the control group (4.89%±1.80%, F=34.24, P<0.01). When treated with the concentration of 0.5 mg/L for 24 h, cell shrinkage, necleoli disappeared, chromatin condensed, arranged along side the nuclear membrane could be found by TEM, an oligonucleosomal DNA ladder of curcumin-treated cells was demonstrated by agarose gel electrophoresis.

**Conclusion**: HCPT could significantly inhibit the proliferation of HSC-T6 in a dose-dependent and time-dependent manner in vitro; HCPT could also induce apoptosis of HSC-T6 in a dose-dependent manner.

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**PP-026** Inhibitory effect of murine cytomegalovirus infection on neural stem cells’ differentiation and its mechanism

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**Background**: To investigate the influence of murine cytomegalovirus (MCMV) infection on differentiation of neural stem cells (NSCs) and its mechanism in vitro.

**Methods**: NSCs separated from fetal BALB/c mouse, were cultured and identified in vitro. The NSCs infected by MCMV were cultured in differentiation medium. The expression changes of nestin, glia fibillary acidic protein (GFAP), neuron specific enolase (NSE) (markers of NSCs and its differentiated cells) and early antigen (EA) of MCMV were studied by immunofluorescence. The ratios of NSCs and its differentiated cells were detected by flow cytometry. Real-time PCR method was employed to measure the expression levels of the keys genes Wnt3, Wnt7a, Neurogenin2, C-myc and CyclinD1 in Wnt signaling of NSCs.

**Results**: NSCs could proliferate to form neurospheres, strongly express nestin and differentiate into neurons or astrocytes in vitro. The nestin expression of the infected groups downregulated slowly and the ratios of GFAP and NSE positive cells of it were lower than that of the control (P<0.05). MCMV EA could be always detected. The mRNA levels of Wnt3, Wnt7a, Neurogenin2, C-myc and CyclinD1 of infected groups were obviously lower than that of the control (P<0.05).

**Conclusion**: MCMV could inhibit significantly the differentiation of NSCs by suppressing the differentiation genes expression.

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**PP-027** Experimental research on the effect of RNA interference specific for Smad4 gene on the activated hepatic stellate cells

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**Background**: RNA interference technology provides a powerful research tool for the study of endogenous gene function and signaling pathway. Activation of HSC is the cellular basis of hepatic fibrosis. Transforming growth factor-β (TGF-β) signaling plays an important role in the liver fibrosis. Smad4 is in a central position in the way of signal transmission, Smad4 transfer to the nucleus to regulate the transcription of target genes and gene expression induced by the lower reaches.

In this paper, designing eukaryotic vector of Smad4-specific interference specific for Smad4 gene to express Smad4-target gene in the activated hepatic stellate cells.

**Methods**: To design pSilencer3.1-H1-hygro plasmid expressing short interfering RNAs (siRNA)that target Smad4 gene region by aid of computer designing on Ambion website. The plasmid expressing small interfering RNA was transfected into the cultured cells via liposome metafectene. The Smad4 mRNA expression and protein synthesis in the HSC-T6 cell line were tested by RT-PCR and western blot technology. Collagen III was also measured in the culture media effectively.

**Results**: The plasmid expressing siRNA was successfully constructed. The Smad4 siRNA could effectively down-regulated both mRNA and protein levels of Smad4. Collagen III in the cell culture medium of HSC-T6 was reduced as well.

**Conclusion**: Smad4 targeted siRNA could effectively inhibit Smad4 expression in the HSC-T6 cell line and reduce the secretion of extracellular matrix.