the HPV 16 E6 gene variations and amino acid changes in cervical intraepithelial lesions. HPV 16 positive formalin-fixed, paraffin-embedded tissue (FFPE) that consisted of 30 cases of squamous cell carcinoma (SCC), 30 cases of high-grade squamous intraepithelial lesion (HSIL) and 10 cases of low-grade squamous intraepithelial lesion (LSIL) were used. Three gene specific primers were used to amplify E6 gene by PCR and PCR products were directly sequenced. The results showed that nucleotide (nt) variation at position 178, from T to G (T178G) was most commonly found in SCC (66.7%) then HSIL (63.3%) and only 40% in LSIL. In contrast, European (E) prototype was found in 10%, 16.7% and 40% in SCC, HSIL and LSIL respectively. The other minor amino acid changes were detected including E6 L83V, E6 R10I, E6 R10T, E6 R10K, E6 L12V, E6 Q14D, E6 Q14H, E6 E18K, E6 K34R, E6 H78Y, E6 R141K, E6 R147K. These nt variations were classified as European variant (E350G) found in HSIL and LSIL(10% and 20% respectively), American Asian (AA) variant, African 2 (Af-2) variant and Javanese135C variant found in 13.3% VS 6.7%, 6.7% VS 3.3% and 3.3% VS 0% of SCC VS HSIL, respectively. This study concluded that HPV 16 Asian(As) variants may be an oncogenic risk of cervical cancer progression in Thai women. To further study, analysis of its oncogenic potential should be more investigated for its clinical significance and interpretation of clinical outcome.

**8010** POSTER

**P53 codon 72 polymorphism does influence cervical cancer development in a German population**

A. Soliman[1], E.I. Braicu[2], J. Sehouli[2]. [1]*Bethesda Evang. Krankenhaus Mönchengladbach Akademisches Lehrkrankenhaus der RWTH, Gynecological department, Mönchengladbach, Germany;* [2]*Charite Medical University Campus Virchow Clinic, Gynecological department, Berlin, Germany*

The aim of our study was to assess the impact of p53 codon 72 polymorphism in HPV related cervical lesions.
**Methods:** In this study, 117 healthy pregnant women previously tested for PAP smears and 111 HPV-positive patients with different cervical lesions were enrolled. The study group included 47 patients with benign lesions, 17 CIN I and CIN II diagnosed patients, and 47 patients with CIN III and invasive cervical carcinoma. For the detection of p53 codon 72 polymorphism, PCR amplification was carried out. The PCR products of p53 and HPV were all sequenced. We analyzed HPV- DNA using following primers: HPV 1/2 and GP 5/6.
**Results:** When the samples were analyzed using the primer pair HPV 1/2, 36 samples out off 105 were negative, same samples being positive when the PCR was performed with GP5/6 primer pair. Our study showed that 34.29% of the HPV PCR were false negative or undetected by PCR that uses only HPV 1/2. Sixty eight per cent of homozygous Arg in the high-risk HPV-group developed CIN III and invasive carcinoma compared to only 52.3% of the heterozygous and 42.9% homozygous Pro. Sequencing detects *high-risk HPV* ignored by ELISA which distorts results needed for accurate conclusion (8.2 % were added to *high-risk HPV-group*).
**Conclusions:** Our data suggest that women homozygous for Arginine are more susceptible for developing HPV 16/18-related high-risk cervical lesions. Using primer pair HPV GP5/6 increases sensitivity in HPV-PCR undetected when using HPV 1/2 primer pair.

**8011** POSTER

**The synergistic effects of nedaplatin and cispaltin on the proliferation and apoptosis of human ovarian carcinoma Skov-3 and cervical carcinoma hela cell line**

L. Li[1], T. Tan[1], S. Su[1]. [1]*Southeast University, Clinical Cancer Center, Nanjing, China*

**Background:** To study the synergistic effects of nedaplatin(NDP) and cispaltin(DDP) on the human ovarian carcinoma Skov-3 and cervical carcinoma Hela cell line.
**Materials and Methods:** The inhibition effects were evaluated by MTT assay. Cell apoptosis was detected by flow cytometry. The changes of Ki-67, Bax and Bcl-2 in mRNA and protein level were quantified by RT-PCR and Western blot.
**Results:** The growth inhibition of Skov-3 was dose-dependent after exposure to the NDP or DDP alone. The interaction of the two drugs was synergistic at higher concent rations according to the Median-effect principle. The inhibition rate of NDP, DDP and combinative treatment group was 39.04±1.26%, 45.04±1.45%, 56.21±1.44% (Skov-3) and 44.76±2.11%, 46.90±0.99%, 56.63±1.83% (Hela) respectively and the cells apoptotic rate was tended to increase. Compared with the NDP or DDP alone treatment group, the combinative treatment group's Ki-67 and bcl-2 mRNA (protein) expression were decreased but the expression of Bax mRNA (protein) were increased.
**Conclusions:** Compared to the effects of NDP or DDP alone at high concentrations, combination of NDP and DDP at low concentrations proves to be much more effective in the inhibition of the proliferation and the induction of the apoptosis of Skov-3 and Hela cell line.

**8012** POSTER

**Combined effects of cyclooxygenase-1 and cyclooxygenase-2 selective inhibitors on the growth of ovarian carcinoma in vivo**

W. Li[1], Z.L. Ji[1], J. Wang[1], H.R. Jiang[1]. [1]*Nanjing Medical University of Hangzhou Hospital, Department of Gynecology, Hangzhou, China*

**Background:** Nonsteroidal anti-inflammatory drugs (NSAIDs) are known to be potent inhibitors of the cyclooxygenases. The present study was designed to investigate the combined effects of cyclooxygenase (COX)-1 and cyclooxygenase (COX)-2 selective inhibitors on the growth of carcinoma in in SKOV-3 ovarian carcinoma xenograft-bearing mice.
**Material and Methods:** Human ovarian SKOV-3 carcinoma cells xenograft-bearing mice were treated with SC-560, a COX-1-selective inhibitor, 6 mg/kg alone and celecoxib, a COX-2-selective inhibitor, 50 mg/kg alone i.g. daily for 21 days. The expression of COX-2 and COX-1 at protein and mRNA levels in the control groups was detected by immunohistochemistry and reverse-transcription polymerase chain reaction (RT-PCR). Angiogenesis of both COX inhibitors was measured by Wester blotting. In addition, prostaglandin $E_2$ ($PGE_2$) levels was determined by ELISA.
**Results:** In combination therapy with SC-560 and celecoxib, tumor volumes was significantly reduced compared with that of control group ($P < 0.05$). In treatment groups, both COX inhibitors significantly reduced intratumor $PGE_2$ levels (all $p < 0.01$). SC-560, administered in combination with celecoxib inhibited the COX associated up-regulation of VEGF. COX-1 and COX-2, mRNA, and protein levels are elevated in tumor tissues.
**Conclusions:** These studies demonstrate synergism between two COX inhibitors and that COX-1 and COX-2 may to some extent contribute to tumor formation independently and inhibitor combination treatment thus has particular potential for chemoprevention of ovarian cancer.

**8013** POSTER

**PDCD6 and ovarian cancer metastasis, findings of a proteomic study**

D. Su[1], L. Ying[1], Y. Gao[1], L. Gu[1], W. Sun[2], H. Yu[3], D. Katsaros[4], S. Xu[1]. [1]*Zhejiang Cancer Hospital, Research Institute, Hangzhou, China;* [2]*Zhejiang Cancer Hospital, Department of Pathology, Hangzhou, China;* [3]*Yale University School of Medicine, Departments of Epidemiology and Public Health, New Haven, USA;* [4]*University of Turin, Department of Obstetrics and Gynecology, Turin, Italy*

**Background:** To identify proteins involved in ovarian cancer metastasis and to evaluate the clinical significance of our finding, we analyzed the proteomics of ovarian cancer cells and verified the results in tumor samples.
**Materials and Methods:** A comparative proteomic analysis involving two-dimensional gel electrophoresis and mass spectrometry identification was performed to identify proteome alterations between HO-8910, a human ovarian cancer cell line, and its highly metastatic subline HO-8910PM. Differences in protein expression between the two cell lines were further validated with western blot. Immunohistochemistry and RT-PCR were also performed to confirm the in vitro findings in tumor tissues and to analyze their associations with clinical and pathological features of ovarian cancer.
**Results:** Twenty-one spots with significant difference in expression (two-fold increase or decrease) were detected, and of them, 17 proteins were successfully identified and characterized. Programmed Cell Death 6 (PDCD6) was one of the proteins whose overexpressoin in HO-8910PM as compared to HO-8910 was further confirmed by western blot analysis. Compared to primary ovarian cancer and normal ovarian tissues, cancer cells metastatic to lymph nodes had significantly increased expression of PDCD6 (100% vs. 72.5% vs. 40%, $P = 0.01$). PDCD6 was mainly located in the cytosol of normal ovarian cells or ovarian cancer cells, whereas in metastatic cells, PDCD6 was mostly translocated to the nucleus. Furthermore, *PDCD6* mRNA expression was significantly correlated with clinical stage, tumor grade and histology. Patients with stage III or IV disease had higher *PDCD6* mRNA expression than patients with stage I or II disease, $p = 0.005$; *PDCD6* mRNA was highly expressed in patients with grade 3 compared to those with grade 1 or 2, $p = 0.016$; serous ovarian cancer expressed more *PDCD6* than non-serous ovarian cancer, $p = 0.014$.
**Conclusions:** The study demonstrated that PDCD6 overexpression was related to metastatic ovarian cancer cells and aggressive ovarian tumors. As such, PDCD6 may be a useful marker for predicting tumor metastasis and/or a therapeutic target for ovarian cancer treatment.