($p = 0.043$) and CXCL10 ($p = 0.028$) was noted in recurrent than in non-recurrent patients when assessed by immunohistochemistry; reverse-transcriptase PCR confirmed these results ($p < 0.05$). Inflammatory adhesion and general type (ulcer type versus elavated type) was significantly correlated with CXCR3, but the expression of CXCL10 was not correlated with tumour location, histological type, size, gender, pre-surgery CEA, and pre-surgery haemoglobin. High expression of CXCR3 was associated with a higher risk of relapse. No such relationship was noted for CXCL10 expression.

Interpretation: The CXCL10/CXCR3 axis, especially CXCR3 may be involved in inflammatory adhesion. CXCR3 and inflammatory adhesion could be used as prognostic markers and contribute to predicting clinical outcome for patients with stage II colorectal cancer.

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P0155
A COMPARATIVE STUDY OF THE OPERATIVE OUTCOMES OF CONTINUOUS VERSUS INTERRUPTED VESICOURETHRAL ANASTOMOSIS IN OPEN RADICAL RETROPUBIC PROSTATECTOMY

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Background: Vesicourethral anastomosis (VUA) is the most technically challenging part in an open radical retropubic prostatectomy (ORRP). Traditionally, it is accomplished using interrupted anastomotic sutures. The objective of this study is to describe our surgical technique of continuous VUA and compare its outcomes with that of interrupted VUA as performed by a single surgeon.

Methods: A total of 235 patients with clinically localised prostate cancer who underwent ORRP between February 2000 and June 2013 were included. They were divided into group 1 ($n = 121$), treated with interrupted VUA and group 2 ($n = 114$), treated with continuous VUA. The primary outcome measures to be evaluated included operative time, blood loss, anastomotic integrity, hospital stay, continence, potency, and occurrence of VUA stenosis. Analyses were done with Welch's t-test and Fisher's exact test. All statistical tests were done with SPSS 20.0. *P* values less than 0.05 indicate statistically significant differences.

Findings: Patients who underwent continuous VUA had significantly shorter operative (210.05 ± 1.91 versus 251.37 ± 2.74 min, $p < 0.001$) and anastomotic times (20.86 ± 0.49 versus 41.46 ± 0.58 min, $p < 0.001$); there was less estimated blood loss (510.81 ± 10.11 versus 623.89 ± 26.60 mL, $p < 0.001$), and need for transfusion (7.89% versus 27.27%, $p < 0.001$), fewer days before drain removal (3.13 ± 0.05 versus 6.15 ± 0.11, $p < 0.001$), fewer of days in hospital (3.44 ± 0.06 versus 6.36 ± 0.11, $p < 0.001$), less leakage per voiding cystourethrogram (0.88% versus 5.76%, $p = 0.035$), fewer days before urethral catheter removal (10.05 ± 0.12 versus 14.94 ± 0.2, $p < 0.001$) and fewer weeks to gain continence (7.05 ± 0.26 versus 12.46 ± 0.31, $p < 0.001$). There were two cases of VUA stenosis in each groups. There were no reported occurrences of pelvic infection, urinoma, and acute urinary retention after catheter removal.

Interpretation: Our technique of continuous VUA for ORRP provides better outcome compared to standard interrupted VUA.

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P0157
PRECLINICAL EVALUATION OF NBN-PACLITAXEL IN PANCREATIC CANCER XENOGRAFT MODELS

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Background: Despite being the standard of care, gemcitabine has limited clinical benefits in pancreatic cancer. The goal of this study was to determine whether non-biological, nanoparticle paclitaxel (NBN-Pac/IG-001) exhibits sufficiently robust preclinical activity to qualify for clinical development.

Methods: We tested NBN-paclitaxel in various pancreatic cancer cell lines and xenograft models.

Findings: The maximum-tolerated dose of NBN-paclitaxel was calculated to be 60 mg/kg versus 20 mg/kg for standard paclitaxel. NBN-paclitaxel was generally more efficacious than standard paclitaxel or gemcitabine. In vitro, enhanced activity was shown by NBN-paclitaxel as compared to standard paclitaxel or gemcitabine against four pancreatic cell lines (MIA Paca-2, Capan-1, PANC-1, and AsPC-1). This increased activity was also demonstrated in vivo in female athymic nude mice using two pancreatic cancer cell lines (MIA PaCa-2 and PANC-1) as well as in male nude BALB/c mice following the orthotopic (pancreatic) implantation of AsPC-1 pancreatic cancer cells. NBN-paclitaxel produced superior anti-tumour activity against the human pancreatic carcinoma xenograft MIA PaCa-2. The rank order (% complete regression) was as follows: NBN-paclitaxel (60, 40, 25 mg/kg), standard paclitaxel (25 mg/kg), and gemcitabine (140 mg/kg). NBN-paclitaxel produced a marked, dose-related anti-tumour effect in the PANC-1 xenograft model, with complete remission in all animals at 50 mg/kg per dose and significant partial remission at 20 mg/kg per dose. Overall, NBN-paclitaxel was shown to be more efficacious than either standard paclitaxel or gemcitabine, at equitoxic dose. NBN-paclitaxel produced dose-dependent primary tumour suppression (41%, 37% and 26%) and reduced the number of metastasised tumour nodules (22%, 19% and 13%) in the AsPC-1 metastasis model at doses of 20, 35, and 50 mg/kg, respectively. Anti-tumour activity of NBN-paclitaxel at 50 mg/kg was superior to standard paclitaxel and gemcitabine at an equitoxic dose.

Interpretation: The preclinical data suggest that NBN-paclitaxel could be superior to standard paclitaxel at an equitoxic dose. The data support further development of NBN-paclitaxel.

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P0158
RELATION BETWEEN POLYMORPHISMS AND CHEMOTHERAPY IN LUNG CANCER

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Background: Lung cancer is the leading cause of cancer-related death globally. Chemotherapy and targeted drugs increase progression-free survival, overall survival, and quality of life. Recently, we are moving towards personalised medicine based on genotypic study of enzymes involved in the metabolism, transport, and elimination of drugs routinely used in lung cancer.

Methods: After obtaining written informed consent, we analysed venous blood draws from 40 patients with NSCLC of all stages, before the start of chemotherapy. We studied polymorphisms, with real-time

PCR, in genes involved in detoxification (*GSTP1*), DNA repair (*XRCC1*, *ERCC1*) in trans-membrane transport (*ABCB1*), and metabolism of anticancer agents (*CYP2C9*, *CYP3A4*, *CYP3A5*, *CYP2D6*, *CYP1A2*, *UGT1A1*, *TSER*).

Findings: We are prospectively analysing the correlation between polymorphisms and toxicity of chemotherapy regimens and progression-free survival, overall survival, and quality of life. We expect a greater toxicity in patients with polymorphisms that result in reduced activity of enzymes involved in drugs metabolism, a reduction in transport, and in DNA repair activity.

Interpretation: Considering that there is no standard first-line chemotherapy for NSCLC, since platinum-based regimens are equally effective, the toxicity of each regimens affects choice of treatment. Therefore, the objective of this study is to identify factors that can direct choice of chemotherapy regimen based on the patient's genotype.

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P0159
BIDENS PILOSA EXTRACT INDUCES APOPTOSIS THROUGH UP-REGULATION OF DEATH RECEPTOR 5 IN HUMAN COLON CARCINOMA CELL LINES

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Background: Bidens pilosa Linn. var. radiata is a tropical weed widely present in tropical and subtropical regions, including Miyako Island, Okinawa, Japan. The whole plant or its leaves and stem are used in various folk medicines and as a popular ingredient in herbal tea for its anti-inflammatory, anti-septic, liver-protective, blood-pressure lowering, and hypoglycaemic effects. There are no reports of the effects of *B. pilosa* on human colorectal cancer cells.

Methods: We investigated the effects of *B pilosa* on the human carcinoma cell lines HCT116 and Caco-2. The former has wild-type p53, the latter has mutant p53. *B pilosa* was provided as an extract powder from Musashi Immune laboratory Co. (Miyako Island, Okinawa, Japan).

Findings: B pilosa (0.25, 0.5, and 1 mg/ml in medium) demonstrated dose-dependent growth inhibition on both cell lines, assessed by WST-8 assay (Nacalai Tesque, Kyoto, Japan). *B pilosa* induced apoptosis in both HCT116 and Caco-2 cell lines, assessed by DNA ladder. In addition, a cleaved caspase-8 was expressed in both cell lines, as assessed by western blot. In both cell lines, in spite of the p53-genetic status, *B pilosa* also increased the expression death receptor 5 (DR5), a TRAIL receptor that activates caspase-8, leading directly to the activation of caspase-3.

Interpretation: Our results suggest that *B pilosa* has the potential to prevent or treat for colon cancers through induction of apoptosis by a p53-independent pathway.

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P0160
CLINICOPATHOLOGIC CHARACTERISTICS OF MALE BREAST CANCER: A REPORT OF 21 CASES AT A RADIOTHERAPY CENTRE IN HAMADAN, IRAN

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Background: Male breast cancer accounts for less than 1% of all cancer in men and only around 1% of all diagnosed breast cancer. Despite a significant increase in the last 25 years, it still remains a rare disease.

Methods: We conducted a retrospective study from 2004–2011 with 21 male breast cancer patients. We aimed to analyse epidemiological data (age, personal and family history), tumour characteristics (size, histological type, location, TNM stage, receptors), surgery, adjuvant chemotherapy and radiation therapy, hormonal therapy and survival (relapse, follow up, death) of men who were referred to our centre with breast cancer.

Findings: The median age was 49.2 ± 14.2 years (range 30–83 years). A family history of breast cancer was noted in four cases. The main clinical complaint was a retroareolar mass in 18 (85.7%) patients. Histologically, 18 (85.7%) were invasive ductal carcinoma, one (4.7%) had ductal carcinoma in situ, and two (9.4%) had mixed histology including invasive medullary and ductal carcinoma. Hormonal therapy was delivered to 16 (76.1%) cases due to ER or PR positivity. During median follow up of 30 months (3–84 months), distant metastases were evident in four (19%) cases. During the follow-up period, only one patient died due to metastatic disease. The mean time to recurrence was 30 months.

Interpretation: The occurrence of male breast cancer is very low compared with breast cancer in women, explaining why very few investigations have been conducted in Iran. Limited coverage in the literature make gender-specific findings difficult so future research of this entity involving multi-institutional cooperation and longer follow-up is essential to provide new insights about the biological and clinical factors of this rare cancer.

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P0162
CYP1B1 POLYMORPHISMS AND HEPATOCELLULAR CANCER RISK IN A CHINESE POPULATION

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Background: CYP1B1 is a P450 enzyme which is involved in the activation of pro-carcinogens to carcinogens as well as oestrogen metabolism. We hypothesised that genetic variants in *CYP1B1* may modify individual susceptibility to hepatocellular carcinoma (HCC).

Methods: To test this hypothesis, we evaluated the associations of three *CYP1B1* single nucleotide polymorphisms (SNPs) and HCC risk in a case-control study of 468 HCC cases and 515 cancer-free controls in a Chinese population. The matrix-assisted laser desorption ionization time-of-flight mass spectrometry method and direct DNA sequencing were done to detect these polymorphisms.

Findings: In overall analysis, the three SNPs rs10012, rs1056836, and rs1800440 were not significantly associated with HCC risk. However, we found that variant genotypes containing the G allele of