promoter had a higher driven efficiency which might be regulated by transcription factor ETS-1 in bladder cancer cells, compared with wild-type hTERT promoter. Meanwhile, the artificial hTERT promoter showed a strong tumor-specific effect. The cell proliferation inhibition and apoptosis induction were observed in artificial hTERT promoter—Bax-Anti Bcl 2 combination module—transfected bladder cancer 5637 and T24 cells, but not in the module-transfected normal human fibroblasts.

Conclusions: This module offers us a useful synthetic biology platform to inhibit the malignant phenotypes of bladder cancer in a more specific and effective way.

Keywords: hTERT promoter; Bax/Bcl 2; bladder cancer

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AB142. Normal peripheral prostate stromal cells stimulate prostate cancer development: roles of c-kit signal

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Objective: To investigated the peripheral stromal cell conditioned medium (CM)-stimulated c-kit-JAK2-STAT1 pathway in prostate cancer.

Methods: CM harvested from normal prostate peripheral stromal cells was added to DU145 cells. DU145 cell viability and migration were measured by cell counting kit-8 reagent and Transwell analysis respectively. Colony and sphere formation efficiencies of DU145 cells co-cultured with CM from human prostate stromal cells were also measured. DU145 cells were stably transfected with lentivirus-mediated shRNA for c-kit silencing.

Results: C-kit expression in prostate cancer was found to be significantly higher than in benign prostatic hyperplasia and positively associated with Gleason scores. The growth, migration and capacity of clonogenic property of DU145 cells significantly increased upon exposure to peripheral stromal CM and then were inhibited after silencing the expression of c-kit. The levels of c-kit, pJAK2 and pSTAT1 were significantly induced by peripheral zone stromal CM compared with controls in serum free medium and the levels of pJAK2 and pSTAT1 decreased after c-kit silencing.

Conclusions: C-kit hyper-expression promotes the development of prostate cancer. The peripheral stromal cell CM stimulated c-kit-JAK2-STAT1 pathway in prostate cancer cell viability, migration, and capacity of clonogenic property. This may lead to a greater understanding of the role of c-kit in prostate cancer and provide a potential therapeutic target for prostate cancer.

Keywords: Prostate cancer; stromal-epithelial interaction; c-kit; signaling transduction

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AB143. Pharmacogenetic association between XRCC1 polymorphisms and improved outcomes in bladder cancer patients following intravesical instillation of epirubicin

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Objective: To investigate the association between four XRCC1 polymorphisms and patients' response to intravesical epirubicin therapy.

Methods: A total of 298 bladder cancer patients and 300 healthy controls were enrolled. Genotyping of four XRCC1 polymorphisms, including C280A, C398T, G499A and C468G, was performed using a TaqMan method.

Results: The C280A and C398T polymorphisms were significantly associated with the response to intravesical epirubicin therapy. The patients with the CT genotype at C280A had a significantly higher response rate to epirubicin treatment compared with those with the CC or AA genotypes (p=0.036). Similarly, patients with the CT genotype at C398T had a higher response rate than those with the CC or TT genotypes (p=0.013).

Conclusions: The XRCC1 polymorphisms C280A and C398T are associated with the response to intravesical epirubicin therapy in bladder cancer patients.

Keywords: Bladder cancer; intravesical epirubicin; XRCC1 polymorphisms

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**Objective:** XRCC1 is a multi-domain protein associated with bladder cancer. We investigated the relationship between the distribution of XRCC1 polymorphisms (rs915927 and rs2854501) and clinical outcomes following intravesical instillation with epirubicin (EPI) or mitomycin C (MMC).

**Methods:** A TaqMan assay was performed to determine genotypes of 240 individuals diagnosed with bladder cancer. Logistic regression was used to assess the association between polymorphisms and relapse-free survival (RFS) of patients. Quantitative real-time polymerase chain reaction was performed to determine expression of XRCC1 polymorphisms. Survival curves were generated using the Kaplan-Meier method.

**Results:** Risk of bladder cancer recurrence was significantly reduced in patients receiving EPI who had higher incidences of XRCC1 polymorphisms (P=0.009 for rs915927, P=0.001 for rs2854501). In participants administered MMC, results were not statistically significant.

**Conclusions:** Polymorphisms in XRCC1 SNP variants (rs915927 and rs2854501) were associated with improved clinical outcomes following EPI treatment.

**Keywords:** Bladder cancer; XRCC1; epirubicin; instillation; polymorphisms

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**AB144. H19-derived miR-675 contributes to bladder cancer cell proliferation by regulating p53 activation**

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**Objective:** Long noncoding RNA 19 (H19) has been shown to promote bladder cancer cell proliferation and metastasis. However, little is known about how miR-675, mature product of H19, contributes to bladder cancer cell proliferation.

**Methods:** In this study, we first evaluated the expression of miR-675 in bladder cancer tissues by quantitative real-time PCR (qRT-PCR) and defined its biological functions by flow cytometry and Western blotting.

**Results:** We found that miR-675 expression levels were remarkably increased in bladder cancer tissues as compared with adjacent noncancerous tissues or normal bladder tissue from health donors; moreover, enhanced miR-675 expression was also observed in bladder cancer cell lines. Ectopic expression of H19 significantly increased bladder cancer cell proliferation and miR-675 expression in vitro. Furthermore, overexpression of miR-675 promoted bladder cancer cell proliferation, while suppression of miR-675 induced G1 phase cell cycle arrest and promoted cell apoptosis. Western blotting analysis further identified that miR-675 inhibited p53 activation, decreased the ratio of Bax/Bcl-2 and cyclin D1 expression in bladder cancer cells; those effects may result in the abnormal proliferation of bladder cancer cells.

**Conclusions:** In conclusion, abnormal enhanced miR-675 expression increases bladder cancer growth by regulating p53 activation, and thus may be helpful in the development of effective treatment strategies for bladder cancer.

**Keywords:** Bladder cancer; miR-675; proliferation; p53

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