expression cancers compared with mock counterparts (survival, P = 0.034). In animal models, HK2-overexpression cancers also induced morphologic change and CD133 activity.
Conclusion: High HK2 expression in bladder cancers induced over-secretion of lactate, which was associated with metastatic behaviors through the cancer stem cell formation, EMT promotion and nuclear translocation of phosphorylated NF-κB and Twist1. HK2 may be a novel oncoprotein and play as target for bladder cancer therapy.

PD11-6:
INHIBITION OF AUTOPHAGY ENHANCES EVEROLIMUS (RAD001)-INDUCED CELL DEATH IN HUMAN BLADDER CANCER CELLS

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Purpose: Mammalian target of rapamycin, mTOR, a downstream protein kinase of phosphoinositide 3-kinase (PI3K)/AKT signaling pathway, has been recognized to play a central role in controlling cancer cell growth. The PI3K/AKT/mTOR pathway promotes tumor growth and survival while suppressing autophagy, a catabolic process in cells to sustain energy homeostasis by collecting and recycling cellular components under stress condition. Conversely, inhibitors of the mTOR pathway such as Everolimus (RAD001), induce autophagy that promote tumor survival and thus, these agents potentially limit their own efficacy. We hypothesized that inhibition of autophagy in combination with mTOR inhibition would improve the cytotoxicity of mTOR inhibitors in bladder cancer.
Materials and Methods: The cytotoxicity of RT4 (grade I), 5637 (grade II), HT1376 (grade III) and T24 (grade III) human bladder cancer cells treated with RAD001 alone or combined with autophagy inhibitors (3-methyladenine (3-MA), bafilomycin A1 (BafA1), chloroquine (CQ) or hydroxychloroquine (HCQ)) was accessed by WST-8 cell viability kit. The autophagy status in cells was performed by the detection of microtubule-associated light chain 3 form II (LC3-II) using immunofluorescent staining and Western blot. Acidic organelles (AVOs) formation in treated cells was determined by acridine orange (AO) vital staining. Inhibition of mTOR pathway by RAD001 was monitored by home-made QPCR gene array and the detection of phospho-mTOR by Western blot. Induced apotposis was determined by measurement of caspase 3/7 activity and DNA fragmentation in cells after treatment.
Results: Advanced bladder cancer cells (5637, HT1376 and T24) were more resistant to RAD001 than RT4. Autophagy flux detected by the expression of LC3-II showed RAD001 induced autophagy. AVOs formation was detected in cells treated with RAD001 and inhibited by the addition of 3-MA or Baf A1. Co-treatment of RAD001 with autophagy inhibitors further reduced cell viability and induces apoptosis in bladder cancer cells.
Conclusion: Our data suggest that coordinate inhibition of the mTOR and autophagy pathway promotes apoptosis, and could be a new therapeutic paradigm for the treatment of bladder cancer.

Podium-12
LUTS

PD12-1:
THE ROLE OF HELICOBACTER PYLORI ANTIGEN IN CHRONIC PROSTATITIS

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Purpose: Epidemiological evidences had shown that patients with Helicobacter pylori (H.p.) infection had more urological prostatitic diseases. And chronic prostatitis patients had significantly higher positive anti-H.p. rate. Therefore, we investigated the possible role of H.p. in chronic prostatitis.
Materials and Methods: We set up chronic prostatitis model by introducing H.p. into male rats. Physiological bladder changes along with pain sensitivity in scrotum and tail base were evaluated with cystometrogram and Von Frey filament correspondently. Local prostatic inflammation was checked with Western blot and immunohistochemical stain. Systemic inflammation was evaluated by checking cytokines in spleen.
Results: Hypersensitivity in rat scrotum confirmed the development of chronic prostatitis. Tail base sensitivity showed less significant correspondence. There was significant difference caspase 1 expression in H.p. antigen stimulated prostate. Matured caspase 1 ratio also increased. However, local IL-1b did not seem to have significance. Systemic inflammation was confirmed by significantly increased TNF-alpha in spleen protein extraction.
Conclusion: H.p. antigen may induce local and systemic inflammation. Local effect in prostate induces chronic prostatitis-like response. Further investigation on the systemic effect of H.p. antigen may be necessary to confirm its immunological role on chronic prostatitis.

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PD12-2:
CELLULAR AUTOPHAGY OF HUMAN STEM CELLS IN THE PROCESS OF DIFFERENTIATION

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Purpose: The study is conducted to understand the cellular autophagy of human adipose derived stem cells during the process of smooth muscle cell (SMC) differentiation.
Materials and Methods: Human adipose derived stem cell (hADSC) were induced differentiation into SMC by the use of low-serum level smooth muscle induction medium (SMIM) during SMC differentiation. Real-time PCR analysis were used to check the mRNA expression of smooth muscle marker genes such as α-smooth muscle actin (SMA), SM22α, Calponin, Caldesmon and smooth muscle myosin heavy chain (MHC). We used western blot assay and immumofluorescence staining were also usedImmunofluorescence staining of the cellular actin cytoskeleton to testify the change at protein level.
Results: There was increased expression of smooth muscle marker genes such as SMA and smooth muscle MHC from hADSCs which were exposed to SMIM for 6 weeks. Increased cellular complexity and granularity in induced hADSC, suggesting the intracellular organelles might be increased during the process of SMC differentiation. The lysosome content is significantly increased but mitochondria and endoplasmic reticulum are not. The increased protein content of the lysosomal-associated membrane protein 1 (LAMP-1) confirmed the increase in lysosome content during SMC differentiation. On the other hand, conversion from LC3-I to LC3-II was increased during SMC differentiation and significant increase was observed at the 3w-differentiation.
Conclusion: These results suggest that autophagy appears to be up-regulated in the early stage of SMC differentiation. Autophagy might play an important role in SMC differentiation of ADSC which may be potential biomaterial for the treatment of urinary incontinence and for bladder reconstitution.

PD12-3:
RESULTS OF THE SURGICAL TREATMENTS OF ULCER TYPE INTERSTITIAL CYSTITIS

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Purpose: The pathophysiology of ulcer type interstitial cystitis (IC) is still unclear. Various medical and surgical therapies have been used without a

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