Moderated Posters 5: Miscellaneous
June 25, 2013, 1400-1600

MP-05.01
The Role of FANCD2 in Renal Cell Carcinoma

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Introduction: We have previously identified using RNA micro-array a set of genes that were upregulated in aggressive renal cell carcinoma RCC cell lines. These genes included a subset belonging to the Fanconi Anemia pathway, a DNA repair pathway. Through preliminary elimination, FANCD2 was identified as the most significant of these genes and was further characterized for its role in RCC.

Material and Methods: The expression of FANCD2 RNA was assessed in praffin imbedded and fresh frozen (RCC) samples. Imune Staining of FANCD2 has been carried out for protein expression on a TMA that includes clear cell, non clear cell RCC, and normal adjacent tissue. A knock down in vitro model for FANCD2 was created using 786-0±VHL and RCC4±VHL cell lines. Western blotting for various components of the VHL pathway was carried out comparing knock down to wild type. Proliferation, branching and invasion were compared between knock down to wild type. Similarly an in vivo assay using nude mice is being carried out for tumour formation using the two cell lines.

Results: FANCD2 RNA expression is significantly increased in clear cell RCC versus normal tissue (p=0.01) but not in non-clear cell RCC versus normal (p>0.05) in paraffin imbedded samples. The same is true for fresh frozen samples (p=0.04 and p>0.05 respectively). There was no difference between FANCD2 knock down cell lines and Wild type with respect to the expression of VHL pathway components (including EGFR, TGFα and VEGF). TMA immunohistochemistry is in the prossess of being scored. FANCD2 expression does not affect proliferation rate. In contrast, FANCD2 knock down highly increases branching and invasion in VHL deficient cell lines.

Conclusion: Our results suggest that FANCD2 may play a protective role in RCC that seems independent of the VHL pathway. A VHL mutation may lead to increased FANCD2 expression to accelerate DNA repair.

MP-05.02
ERp46 Mediates Prostate Cancer Tumourigenesis in Vitro by Inhibiting Adiponectin-induced Tumour-suppressive Effects: Linking Obesity to Prostate Cancer

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Introduction: Lower levels of adiponectin, a hormone secreted from adipocytes only, are associated with an increased risk of prostate cancer (PC). Hypo-adiponectinemia is an endocrine hallmark of obesity. Adiponectin-induced tumour suppression is mediated downstream via AMPK, a key regulator of mTOR. The endoplasmic reticulum (ER) protein ERp46 has been suggested as an inhibitor of this pathway. We examined this hypothesis in vitro using PC cells.

Methods: Human 22Rv1 cells are androgen-responsive, produce PSA and express AdipoR1, but little AdipoR2. Co-immunoprecipitation with anti-AdipoR1 antibody covalently bound to magnetic Dynabeads was used, while anti-ERp46 antibody served for detection. Gain- and loss-of-function experiments were done following stable ERp46 shRNA knockdown and ERp46 overexpression, respectively.

Results: ERp46 interacted with AdipoR1, leading to a decrease in AMPK phosphorylation. Treatment with adiponectin, at 1 and 20 g/ml, significantly decreased PSA and VEGF secretion, and increased TIMP-1 secretion (ANOVA). Adiponectin, at 1 and 20 g/ml for 1 hour also activated AMPK (Thr172 phosphorylation) and led to mTOR inhibition as determined by Western blot analysis. Stable knockdown of ERp46 (9-fold) increased phosphorylation of AMPK in 22Rv1 cells compared to scrambled control, whereas stable overexpression ofERp46 (4-fold) led to a decrease in phosphorylation of AMPK. When ERp46-manipulated cells were treated with 2.5 g/mL humanrecombinant full-length adiponectin and the activation of AMPK was determined by immunoblotting, results confirmed an inhibitory role of ERp46 on AdipoR1 signaling as demonstrated by a decrease in phosphorylated AMPK.

Conclusions: ERp46 is a negative modulator of AdipoR1 activity in PC. Upon binding to AdipoR1, ERp46 inhibits the activation of AMPK and thus promotes tumourigenesis. Higher levels of adiponectin (inherently lower in obese individuals) may be needed to overcome this inhibition.

MP-05.03
Determinants of Performance on the Transfer Task of the Basic Laparoscopic Urologic Surgery (BLUS©) Curriculum Administered at Objective Structured Clinical Examinations

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Purpose: To assess determinants of performance on the Transfer Task of the Basic Laparoscopic Urologic Surgery (BLUS©) skills curriculum administered at Objective Structured Clinical Examinations (OSCEs).

Methods: After obtaining Institutional Review Board approval and informed consent, urology trainees (PGY-3 to PGY-5) from 4 different training programs (A, B, C, D) were recruited for the study. Training program A had a dedicated laparoscopic skills training program. One of the rest stations at the semi-annual urology OSCE was replaced by the Peg-transfer Task of the MISTELS. Transfer Task Times (TTTs) were compared and correlated with previous laparoscopic experience, amount of endotrainer practice and scores obtained at practice sessions and other OSCE stations.

Results: A total of 37 trainees were evaluated on 3 successive semi-annual OSCEs from May 2011 to May 2012, including 16 (43.2%) trainees from program A. Compared with trainees from programs B, C, and D, trainees from program A had significantly more training per week (0 vs. 45 min, p=0.001) and significantly lower median TTTs at OSCEs [114 (68-209) vs. 74(52- 89) sec., p=0.001] despite significantly lower number of laparoscopic cases performed within the previous 6 months [13(0-57) vs. 2(0-35), p=0.001]. For program A trainees, TTTs moderately correlated with median TTTs at practice sessions (r=0.57, p=0.001) and negatively correlated with amount of practice per week (r=-0.41, p=0.003). Thus, more training resulted in faster times at OSCEs. However, TTTs showed no correlation with the number of laparoscopic cases performed within the previous 6 months (r=0.04, p=0.76). On multivariate analysis, amount of practice per week was the only significant predictor of TTTs at OSCEs (p=0.028).

Conclusions: Performance on the transfer task of BLUS© during OSCEs significantly correlated with the amount of practice rather than the number of laparoscopic cases assisted.

MP-05.04

Detecting Tumour Hypoxia During Sunitinib Therapy in a Renal Cell Carcinoma Mouse Model Using Positron Emission Tomography

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Introduction and Objectives: Sunitinib is currently the first line therapy for metastasizing renal cell carcinoma (RCC). It has been shown to inhibit tumour angiogenesis leading to modifications of the tumour's microenvironment. Tumour hypoxia plays an important role in the metastatic potential of a solid tumour and its resistance to any chemotherapy. Therefore monitoring tumour hypoxia could potentially be used to detect and analyze therapeutic response. Positron-emission tomography (PET) was used to determine changes in tumour oxygenation during and following sunitinib therapy in a mouse RCC tumour model.

Methods: Subcutaneous Caki-1 tumours were grown for 40 days in BALB/c nu/nu mice. Mice bearing 2 subcutaneous Caki-1 tumours were sorted into 2 groups: a) receiving 40mg/kg/d sunitinib i.p. and b) vehicle control injections. PET imaging (3 h p.i.) utilizing the hypoxia radiotracer [18F]FAZA was performed after 4 days, 7 days, and 13 days of therapy, and again 12 days after 15 day therapy. In addition, biodistribution of [18F] FAZA and immunohistochemical tissue staining to detect pimonidazole HCl adducts were determined.

Results: PET experiments revealed a relatively low uptake level of [18F] FAZA into murine RCC tumours. After 7 days of sunitinib therapy (resulting in decreased tumour growth) the SUV was significantly reduced: 0.24±0.02 in control versus 0.19±0.01 in sunitinib treated mice (n=8; $p<0.05$). Allowing a 12-day drug holiday following 15 days of sunitinib therapy resulted in an increased [18F]FAZA uptake into the treated mice: 0.18±0.01 (n=5, control) versus 0.23+/-0.01 (n=6; $p<0.05$, sunitinib). Biodistribution studies with [18F]FAZA and pimonidazole staining supported the trends of uptake during and after the sunitinib therapy. In vitro cell uptake experiments showed that the [18F]FAZA uptake of 0.25±0.04 (normoxic) or of 2.46±0.42 (hypoxic; n=9) into Caki-1 cells was not changed in the presence of 3 μM sunitinib indicating no direct compound effects.

Conclusions: Uptake of [18F]FAZA tended to decrease during therapy of sunitinib, indicating a decrease in the tumour's hypoxia. However, when stopping drug therapy in tumour-bearing mice, this effect was reversed and tumour hypoxia was increased again. [18F]FAZA could potentially be used to monitor drug response of a therapy with sunitinib in RCC.

MP-05.05

Sub-acute Levels of Antibiotics Used to Treat Recurrent Urinary Tract Infections May Promote Pathogen Growth: Implications for Clinical Practice

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Introduction and Objectives: Recurrent urinary tract infections (RUTI) remain a common reason for patient visits to urology. In many cases, antibiotic prophylaxis is prescribed to prevent overgrowth of pathogens within the bladder; however, antibiotic concentrations are not constant and repeatedly fall to sub-minimal inhibitory concentrations (sub-MICs). The goal of this project was to investigate the role of antibiotics at sub-MICs on *Enterococcus* sp., an increasingly important cause of RUTI.

Methods: Seven strains of *Enterococcus faecalis* and one strain of *E. faecium* were profiled for antibiotic susceptibility using 12 antimicrobial agents commonly used in the treatment of uncomplicated and complicated urinary tract infection (UTI). Profiling was performed by standard CLSI disk-diffusion and microdilution methodologies using media of varying nutrient load to mimic variation in urine concentration.

Results: Antibiotic susceptibility remained constant for most antibiotics irrespective of the culture media. However, notable exceptions included ciprofloxacin, levofloxacin, and doxycycline, which demonstrated altered susceptibility status for 8/8, 7/8, and 7/8 *Enterococcus* strains, respectively. More importantly, there was increased growth beyond the zone of inhibition, suggesting that enterococci may utilize antibiotics to actively grow.

Conclusions: The issue of antibiotic use in urology has come under scrutiny in recent years. In fact, 25-42% of UTI cases resolve without antibiotics and only 2% develop pyelonephritis. The goal then is to achieve good symptom control, and German researchers have reported equivalent success with ibuprofen (400 mg 3 times daily) over ciprofloxacin. Our finding that enterococci not only resist antibiotics used in RUTI prophylaxis, but may use them as a substrate, could explain breakthrough infections or low-grade symptoms and signs. Overall, further re-evaluation of antibiotic prophylaxis is warranted.

MP-05.06

Combined Targeting of PI3K/AKT and AR Pathway with AZD5363 and Enzalutamide Induces Anticancer Activity in Preclinical Models of Prostate Cancer

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Introduction and Objectives: Recent advances in castrate resistant prostate cancer (CRPC) treatment using androgen receptor (AR) targeted therapies such as enzalutamide (MDV3100) have demonstrated striking improvements in survival, but resistance to therapy inevitably occurs. MDV3100 induces phospho-activation of Akt (pAkt) and inhibition of Akt results in feedback signaling leading to activation of AR, suggesting inhibiting Akt alone is not a good strategy. The objective of this study is to evaluate the effect of dual targeting Akt using AZD5363 and the AR pathway using MDV3100.

Methods: Human prostate cancer cell lines LNCaP, C4-2, 22RV1 and MDV3100 resistant cell line (MR49C) were treated with AZ5363, MDV3100 or both. Cell proliferation was assessed by crystal violet, cell cycle population was assessed by FACS analysis, and cell apoptosis was assessed by PARP cleavage and caspase-3 activity. AR transcriptional activity was assessed by probasin luciferase and AR signaling pathway was assessed by western blot and qRT-PCR. For an in vivo model, male athymic nude mice injected with LNCaP were castrated after start of tumour growth and randomly selected for vehicle, AZD5363, MDV3100 or AZD5363+MDV3100 once CRPC was identified. Tumour volume and serum PSA were monitored weekly.

Results: Combination therapy was more potent than mono-therapy. AZD5363 and MDV3100 synergize and induce cell apoptosis showing PARP cleavage and caspase 3 activity as well as increase of subG1/G0 population in all cell lines. MDV3100 abrogated AZD5363-induced up-regulation of AR transcriptional activity and up-regulation of AR dependent genes both at mRNA and protein levels. The effect of the combination in the in vivo model is currently under evaluation.

Conclusions: Combined blockade of the AKT and AR pathway with AZD5363 and MDV3100 demonstrates significant induction of apoptosis and inhibits proliferation and provides a preclinical proof of principle for using these two drugs in the clinic in CRPC.

MP-05.07

Resident Involvement in Bladder Tumour Resections is Associated with Inadequate Pathology Specimens

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Background: Transurethral resection of bladder tumours (TURBT) is a challenging procedure. Pathology specimens lacking detrusor muscle often necessitate repeat procedures, placing patients at increased perioperative risks and delaying definitive management of invasive tumours. The impact of resident learning on TURBT outcomes is unknown.

Methods: Patients who underwent TURBT from November 2011 to November 2012 at our academic centre were identified and all TURBTs performed on these patients in the last 5 years were reviewed. Resident involvement was determined by operative reports. Pathology reports were reviewed to determine tumour grade, depth of invasion, and presence of

detrusor muscle in specimens. Associations were analyzed by chi-square and T-tests.
Results: 138 patients underwent 324 TURBTs in the study period. Mean age was 73 years and 103 (75%) were male. Residents participated in 69% of procedures. Pathology was benign, Ta, Tis, T1, and T2 in 18%, 3%, 43%, 26%, and 10% respectively; 44% of tumours were high grade. The presence or absence of muscle in specimens was clearly specified in 193 cases (60%). Among these, muscle was absent in 43% of cases involving residents and 25% not involving residents (p = 0.01). Among the 119 TURBTs performed for T1 or T2 tumours, muscle was absent in 38% of cases involving residents compared to 19% not involving residents (p=0.04). Resident experience was associated with a non-significant trend towards superior specimens; muscle was absent in 50%, 48%, 47%, 35%, and 29% of TURBTs involving residents in years PGY1, 2, 3, 4, and 5 respectively (p=0.14).
Conclusions: Resident involvement in TURBTs is associated with inadequate resection specimens. Teaching residents the endoscopic skills required for optimal TURBT remains a challenge. Teaching aids, such as bench or virtual models, should be evaluated as a means of facilitating resident teaching without compromising patient care.

MP-05.08
The Role of miR-34a in the Response to Radiation Therapy in Bladder Cancer
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Introduction and Objectives: To preserve the bladder and improve outcomes in bladder cancer patients through targeting signalling pathways with radiation and understand the underlying mechanism. We investigate resistance of radiation via miR-34a expression in human urothelial carcinoma.
Methods: Human urothelial carcinoma cell lines derived from well-differentiated superficial bladder tumours as well as from high-grade invasive tumours were screened for their sensitivity to radiation. Growth inhibition was monitored by clonogenic assays. qRT-PCR analysis was used to evaluate expression of miR-34a among the human urothelial carcinoma cell lines. Methylation-specific polymerase chain reaction (MSP) was performed to determine CpG methylation status of miR-34a gene promoter among the cell lines. Association between miR-34a and sensitivity to radiation was also investigated by ectopic expression of miR-34a. Western blot was performed to evaluate protein expression.
Results: Remarkable differences in sensitivity to radiation have been shown among those cell lines in vitro. Of interest, miR-34a expression correlates with human bladder cancer cell response to radiation, with resistant cell lines showing lower expression of miR-34a and that is possibly due to CpG methylation of its promoter. Additionally, an inverse correlation was observed between miR-34a and expression of sirtuin1 (SIRT1). Ectopic expression of miR-34a in cell lines resistant to radiation showed a positive effect of miR-34a to the response therapy. Growth inhibition observed after treatment could be explained, in part, by the increased expression of the cell cycle inhibitor p21.
Conclusion: Preliminary results suggest that detection of miR-34a expression may potentially be used as a predictor of therapeutic efficacy, and its regulation represent an attractive mechanism for the management of bladder cancer therapy.

MP-05.09
Novel Oncolytic Vaccinia Virus (VAC) has Significant Activity Against Bladder Cancer
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Introduction and Objectives: Oncolytic viruses preferentially replicate in and lyse cancer cells while sparing normal cells. Virus replication requires an abundant supply of deoxyribonucleotide triphosphates (dNTPs), but because cellular enzymes that synthesize dNTPs are degraded at the end of S-phase, VAC expresses its own biosynthetic enzymes including both I4L (large, R1) and F4L (small, R2) subunits of the heterodimeric ribonucleotide reductase (RR). We have shown that deleting the F4L gene inhibits virus replication and reduces virulence in a mouse model. Most importantly for oncolytics, ΔF4L mutations render virus growth sensitive to low levels of cellular RR.
Methods: VACs are tagged with the gene encoding mCherry fluorescent protein and carrying deletions in F4L, J2R (viral thymidine kinase, TK) or both. Oncolytic activity of these VACs was evaluated in a panel of human and rodent bladder cancer or non-tumourigenic bladder cell lines *in vitro*. The orthotopic KU7-luc bladder cancer xenograft model in nude mice was used to assess the mutant VACs *in vivo*.
Results: Cytotoxicity assays showed a high degree of cell killing in infections with ΔF4L, ΔJ2R or the double mutant VACs. Highly efficient VAC replication was observed in our panel of bladder cancer cell lines and particularly in the human urothelial cell carcinoma (UCC) cell line KU7-luc as well as the rat UCC cell line AY-27. Preliminary data indicate that pre-treatment of bladder cancer cells with gemcitabine sensitized them to oncolytic VAC killing. We show that our mutant VAC selectively replicated in KU7-luc bladder cancer xenografts in nude mice. Significant tumour regression in mice treated with the mutant VAC was seen.
Conclusions: These data indicate that the VACs have retained much of their replication proficiency and cytotoxicity in bladder cancer and *in vivo* replication is selective for the cancerous cells and avoids transmission despite deletions of these essential viral genes.

MP-05.10
Inhibition of Endogenous Hydrogen Sulphide Production Reduces Hypoxia-induced Proliferation in Renal Cell Carcinoma Cell Lines
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Introduction and Objectives: Renal cell carcinoma (RCC) is the most common type of kidney cancer and is associated with a significant degree of morbidity and mortality, especially following metastasis. Although various adjuvant tyrosine kinase and mTOR pathway limiting agents have shown promising results in curbing the proliferative signalling pathways seen in RCC, they are associated with serious adverse side effects. Hydrogen sulphide (H_2S) is a newly discovered, endogenously derived small molecule responsible for many physiological and pathophysiological processes. Recent studies suggest the presence of a proliferative signalling pathway that is mediated by interplay between H_2S and T-type calcium channels activated in response to hypoxia in other cancer cell lines. The purpose of this study is to determine the role of this pathway in RCC tumour progression.
Methods: 786-O RCC cells were cultured in either normoxia (21% oxygen) or hypoxia (1% oxygen) and in the presence/absence of the long-acting H_2S-donor molecule, GYY-4137, and H_2S-producing enzyme inhibitors hydroxylamine (HA), and propargyl glycine (PAG). Cells were labeled with the proliferation marker carboxyfluorescein succinimidyl ester (CFSE) and analyzed via flow cytometry.
Results: RCC cells incubated in hypoxic conditions showed increased proliferation rates which were accentuated with the addition of exogenous H_2S. Interestingly, the inhibition of endogenous H_2S production led to a marked reduction in proliferation to levels below resting states. This decline was only observed with the use of HA but not PAG. Treatment with GYY-4137 was found to partially restore proliferation that was lost due to inhibition by HA, but did not affect the proliferation of the normoxic control.
Conclusions: These results suggest that modulating the endogenous production of H_2S using HA may represent a novel therapeutic strategy in the treatment of RCC, possibly delaying progression towards metastasis.

MP-05.11
Improving Ultrasonic-pneumatic Stone Fragmentation by Stabilization with the UroNet in a Bladder Stone Model
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Introduction and Objectives: During endoscopic cystolithotripsy, a significant amount of time can be spent isolating stone fragments. The UroNet

(US Endoscopy) is a new product, which may be used to isolate stones during endoscopic fragmentation.
Our objective was to identify the effectiveness of the UroNet (UN) during ultrasonic-pneumatic cystolithopaxy of phantom stones.
Methods: A model bladder was created using a 600cc container with a urethane cover which accommodated a 30Fr sheath. This allowed for for rotational manipulation of the nephroscope, modeling the set-up for a percutaneous cystolithopaxy. A Karl Storz 26Fr nephroscope was used with a Swiss Lithoclast to provide ultrasonic+pneumatic (Us/P) lithotripsy. A 1cm phantom stone was introduced into the bladder, and subjected to fragmentation for 60s, with or without capture and stabilization by the UN through the nephroscope. All fragments were strained and those >1 mm were recorded. Five trials with or without stabilization were performed, recording the size/number of fragments, change in dry weight, and damage to the UN. Strength of the UN was measured using a 'marble grasping force' technique, and compared to the values of 10 unused UNs.
Results: The mean time to stone capture with the UroNet through the nephroscope was 12s (range 8-45 s). Absolute weight was significantly reduced by stabilization with the UroNet, (US/P+UN 22.5+0.02% vs. US/P - UN 8.01+4.1%, ANOVA $p<0.0001$). Stabilization with the UroNet lead to a greater number of fragments (17+5 vs. 5.0+4.1 fragments/stone) and smaller fragment size (3.6+0.51 mm vs. 7.05+3.00 mm) Defects in the UN were found that ranged in size from 2-14 mm, however all retained their original strength (8.27LbF) except for one net which was rendered non-functional.
Conclusions: Stone stabilization using the UroNet for ultrasonic and pneumatic lithotripsy in a bladder stone model leads to improved stone mass reduction, fragmentation and smaller fragments.

MP-05.12
Effect of Metformin and Physical Activity on Prostate Cancer Progression in an LNCaP Xenograft Model
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Introduction: Metformin is associated with reduced cancer risk, including prostate cancer (PCa) in type II diabetic patients. Metformin exert these effects by mitigating hyperinsulinemia, which deregulates IGF-I production. Studies have found an inverse relationship between physical activity and cancer progression. Proposed mechanisms include reduction in insulin and IGF-I. Our lab reported that a high fat-high carbohydrate (HF-HC) diet increased serum IGF-I in mice and metformin significantly reduced serum IGF-1, however didn't affect tumour growth significantly. The combinatorial effects of metformin and physical activity on PCa progression have not been explored. Herein, we aim to investigate the effect of physical activity and metformin on PCa tumour growth.
Methods: Athymic nude mice (n=40) were inoculated subcutaneously with 1 million LNCaP cells. All animals were fed ad libitum with a HF-FC diet. Animals were randomized into 4 groups: control, metformin, exercise, a combination of metformin and exercise. Exercise was implemented 3 times per week using a forced exercise wheel. Metformin was administered i.p. at a dose of 50mg/kg, three times per week. Body weights, tumour volumes, and food intake were recorded tri-weekly. Blood samples were obtained by sephenous vein bleeding.
Preliminary Results: There was no significant difference in body weights of animals among the groups. Compared with control, the exercise group and combination group consumed significantly less food. Tumour volumes of animals in the exercise group and the group receiving a combination of exercise and metformin were significantly smaller compared to the controls. Further studies are underway to examine the relationship between diet, exercise and metformin administration with respect to PCa progression.
Conclusion: Ten weeks of exercise significantly slowed tumour progression. However, metformin treatment alone did not affect the tumour growth significantly in this animal model.

MP-05.13
A Novel Role for C-terminal Binding Protein (CTBP)-2 in the Pathogenesis of Male Infertility
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Introduction: The precise mechanisms underlying testicular failure in infertile men are not well understood. Spermatogenesis requires intact DNA repair systems to correct cellular insults or commit the cell to apoptosis. CTBP2, a transcriptional co-repressor, plays a role in protecting the cell from apoptosis; however, its function in male infertility has never been assessed.
Methods: Tissues were obtained from men undergoing testis biopsy for non-obstructive azoospermia (NOA; n=16) and vasectomy controls (n=5). Gene-expression microarray (Agilent Sureprint G3) screened for genetic variations. Microarray data were evaluated with heatmaps, clustering and statistical analysis. Ingenuity Pathway Analysis (IPA) software highlighted candidate genes and pathways involved. CTBP2 expression was assessed via PCR and qPCR. Immuno-staining on paraffin embedded testicular tissue was performed.
Results: IPA analysis of microarray data identified CTBP2 as a top transcription factor altered in NOA men. Genomic DNA screened with PCR directed at exons common to all isoforms identified CTBP2 in the testicular tissue of NOA and control patients. When subdivided by pathology, patients with hypo-spermatogenesis and maturation arrest exhibited a 1.3 and 1.1 fold increase respectively in CTBP2 expression. NOA men with Sertoli Cell Only (SCO) syndrome had a significantly decreased (0.7 fold) change in expression. Immunohistochemistry confirmed expression in all patients with decreased levels in those with SCO.
Conclusion: CTBP2 is present in testicular tissue and, when subdivided by histology, men with SCO syndrome exhibited the lowest expression compared to other pathologies. CTBP2 represents a novel target for investigation in men with NOA and infertility.

MP-05.14
Practice Patterns of Post-radical Prostatectomy Incontinence Surgery in Ontario
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Introduction and Objectives: Urinary incontinence is a significant complication following radical prostatectomy. Surgical management of post-prostatectomy incontinence includes artificial urinary sphincter and urethral sling insertion. We assessed practice patterns within the province of Ontario with respect to both radical prostatectomy and the resultant incontinence procedures.
Methods: We performed a population-based study of 25,346 men in Ontario, Canada who underwent radical prostatectomy between 1993 and 2006. Using hospital and cancer registry data, we identified patients who subsequently underwent an incontinence procedure. We characterized the rates of both radical prostatectomy and incontinence procedures across Ontario during the study interval. We then analyzed rates of incontinence procedures performed as compared with those expected given the radical prostatectomy case volume.
Results: 703 (2.8%) men underwent subsequent insertion of an AUS and a further 282 (1.1%) underwent a urethral sling procedure (985 total incontinence procedures, 3.9%) over the study interval. There procedures were performed at 48 different institutions; however, 56% of all procedures were performed at only 3 sites. The majority of hospitals performed significantly fewer incontinence procedures than expected give their radical prostatectomy case volume.
Conclusions: These data clearly show that a small number of academic institutions provide the majority of surgical care for men with incontinence following radical prostatectomy.