

## IN BRIEF

## ➔ BLADDER CANCER

**Two molecular subtypes identified**

Bladder cancer can be distinguished into two molecular subtypes — luminal or basal — based on their gene expression signature. The luminal subtype has an expression pattern similar to the superficial and intermediate layers of the normal urothelium, with upregulation of PPAR $\gamma$  target genes and enrichment of mutations in *FGFR3*, *ELF3*, *CDKN1A*, and *TSC1*. Basal tumours have gene expression patterns similar to the basal layer of the urothelium, with upregulation of p63 target genes and enrichment of mutations in *TP53* and *RB1*. Subtype identification can be achieved with 90% accuracy by immunohistochemically assessing GATA3 and KRT5/6 expression, which are markers for luminal and basal subtypes, respectively.

**ORIGINAL ARTICLE** Dadhani, V. *et al.* Meta-analysis of the luminal and basal subtypes of bladder cancer and the identification of signature immunohistochemical markers for clinical use. *EBioMedicine* <http://dx.doi.org/10.1016/j.ebiom.2016.08.036> (2016)

## ➔ INCONTINENCE

**Results of the RELAX study extension**

Results from the extension of the RELAX study have shown that repeat onabotulinumtoxin A treatment in women with refractory idiopathic detrusor overactivity has consistent efficacy and duration of action. Up to three injections of treatment or placebo were offered to patients over a 5-year period and no statistically significant differences for symptom outcomes or time to symptom return were observed between injections.

**ORIGINAL ARTICLE** Owen, R. K. *et al.* Comparison of the effectiveness of repeated injections of onabotulinum toxin A for refractory idiopathic detrusor overactivity: analysis of an open label extension of a randomized trial (the RELAX study). *NeuroUrol. Urodyn.* <http://dx.doi.org/10.1002/nau.23095> (2016)

## ➔ KIDNEY CANCER

**Sunitinib resistance is futile**

Inhibiting MEK can resensitize tumours to sunitinib in a patient-derived xenograft mouse model of renal cell carcinoma. Tumours in mice treated with sunitinib alone developed resistance to therapy after 30 days, whereas mice treated with either sunitinib in combination with the MEK inhibitor PD-325901 or that received PD-325901 after the onset of resistance to sunitinib continued to have a tumour response.

**ORIGINAL ARTICLE** Diaz-Montero, C. M. *et al.* MEK inhibition abrogates sunitinib resistance in a renal cell carcinoma patient-derived xenograft model. *Br. J. Cancer* <http://dx.doi.org/10.1038/bjc.2016.263> (2016)

## ➔ PROSTATE CANCER

**More cycles of docetaxel improve survival**

A *post hoc* analysis of data from the Mainsail study has shown that treatment with eight or more cycles of docetaxel is associated with improved overall survival rates in men with prostate cancer. Patients involved in the Mainsail study received either docetaxel, prednisone and lenalidomide or docetaxel, prednisone and placebo; however, men in the lenalidomide arm received fewer cycles of docetaxel owing to the toxic effects of the combination treatment. Irrespective of whether lenalidomide was part of their treatment regime, men who underwent more cycles of docetaxel had significantly superior overall survival.

**ORIGINAL ARTICLE** de Morrée, E. S. *et al.* Association of survival benefit with docetaxel in prostate cancer and total number of cycles administered a *post hoc* analysis of the Mainsail study. *JAMA Oncol.* <http://dx.doi.org/10.1001/jamaoncol.2016.3000> (2016)