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IN BRIEF

THYROID

Epigenetics underlie silencing of NIS in thyroid cancer

Zhang *et al.* report a novel epigenetic mechanism for BRAF V600E-promoted silencing of the sodium/iodide cotransporter NIS in thyroid cancer cells that involves histone deacetylation at transcriptionally active areas upstream of the translation start of *SLC5A5*, the gene that encodes NIS. Using chromatin immunoprecipitation, the researchers analysed histone acetylation on lysine residues within the *SLC5A5* promoter under the influence of mutant BRAF. The regions affected by BRAF V600E-mediated deacetylation contain important transcription factor binding sites.

Original article Zhang, Z. *et al.* Histone deacetylation of NIS promoter underlies BRAF V600E-promoted NIS silencing in thyroid cancer. *Endocr. Relat. Cancer* doi:10.1530/ERC-13-0399

DIABETES

XBP1s regulates adiponectin multimerization in mice

Overexpression of XBP1s, the spliced form of X-box-binding protein 1, a pivotal component of the endoplasmic reticulum stress response, in mice improves systemic glucose homeostasis, a study published in *Diabetes* shows. XBP1s acts by promoting the multimerization of adiponectin in adipocytes rather than by activating adiponectin transcription. The researchers propose that this action is mediated through direct regulation of the expression of several endoplasmic reticulum chaperones involved in adiponectin maturation.

Original article Sha, H. *et al.* Adipocyte XBP1s promotes adiponectin multimerization and systemic glucose homeostasis. *Diabetes* doi:10.2337/db13-1067

CANCER

Acquired endocrine resistance in breast cancer

Acquired mutations can induce resistance to hormonal therapy in patients with breast cancer, suggest findings by Merenbakh-Lamin and colleagues. The investigators analysed samples obtained from 13 patients with metastatic breast cancer for mutations in cancer-related genes and identified a mutation in ERα in liver metastases of five patients who developed resistance to hormonal therapy. The primary tumours had not previously harboured this mutation, which structural modelling showed leads to a conformational change in the ligand-binding domain that ultimately alters binding of tamoxifen.

Original article Merenbakh-Lamin, K. *et al.* D538G mutation in estrogen receptor-α: a novel mechanism for acquired endocrine resistance in breast cancer. *Cancer Res.* doi:10.1158/0008-5472.CAN-13-1197

PANCREAS

Acinar-to-β-cell reprogramming without genetic manipulation

Transient administration of epidermal growth factor and ciliary neurotrophic factor to adult mice with chronic hyperglycaemia efficiently stimulates the conversion of terminally differentiated acinar cells to β-cell-like cells, show Baeyens and co-workers. The newly generated functional cell mass was sufficient to achieve and maintain normoglycaemia for at least 248 days. The regenerative process depended on activation of *Neurog3* and signalling through Stat3.

Original article Baeyens, L. *et al.* Transient cytokine treatment induces acinar cell reprogramming and regenerates functional β cell mass in diabetic mice. *Nat. Biotechnol.* doi:10.1038/nbt.2747