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IN BRIEF

GENETICS

Epigenetic modifications to cytokine promoters in NSCLC

A study has demonstrated for the first time that the levels of DNA methylation of the promoter regions of the genes encoding IL-1ß, IL-6 and IL-8 are increased in human non-small-cell lung cancer (NSCLC) cells. High DNA methylation levels of these gene promoters were associated with reduced mRNA levels in lung cancer cells, whereas the mRNA levels of these genes were higher in normal bronchial epithelial cells or adjacent nontumourous tissues. This inverse relationship was strongest for IL-1ß, in which hypermethylation was associated with silencing of *IL1B* in cancer cells.

Original article Tekpli, X. *et al.* DNA methylation at promoter regions of interleukin 1B, interleukin 6, and interleukin 8 in non-small cell lung cancer. *Cancer Immunol. Immunother.* doi:10.1007/s00262-012-1340-3

GENETICS

SNP genotyping reveals correlation with glioma development

Seven low-frequency single-nucleotide polymorphisms (SNPs) at 8q24.21, identified using techniques such as long-range PCR, have been associated with the risk of developing various gliomas. In the 1,657 case and 1,301 control samples examined, the SNP rs55705857 was significantly correlated with the development of oligodendroglial tumours and gliomas with *IDH1* or *IDH2* mutations. Astrocytomas without *IDH* mutations were not significantly associated with rs55705857, which suggests that the risk locus in 8q24 interacts with *IDH* mutations in the development and progression of gliomas.

Original article Jenkins, R. B. *et al.* A low-frequency variant at 8q24.21 is strongly associated with risk of oligodendrogial tumors and astrocytomas with *IDH1* or *IDH2* mutation. *Nat. Genet.* doi:10.1038/ng.2388

GENETICS

Possible driver mutations in small-cell lung cancer identified

Integrative genome analysis of the exome (27 tumours, 2 cell lines), genome (2 tumours) and transcriptome (15 tumours) of small-cell lung cancer (SCLC) has revealed a high rate of protein-changing mutations. Included amongst these mutations was inactivation of *TP53* and *RB1*. Additionally, mutations in *CREBBP*, *EP300* and *MLL*—which encode histone modifiers—were detected, as were mutations of *PTEN*, *SLIT2* and *EPHA7*. These mutations might drive SCLC development and progression, representing possible targets for therapy for this aggressive cancer type.

Original article Peifer, M. *et al.* Integrative genome analyses identify key somatic driver mutations of small-cell lung cancer. *Nat. Genet.* doi:10.1038/ng.2396

GENETICS

SOX2 is amplified in small-cell lung cancer

A study of more than 50 tumour samples and cell lines of small-cell lung cancer (SCLC) has shown that *SOX2* is amplified in approximately 27% of cancers. Several fusion transcripts were also identified, including *RLF–MYCL1*, although its role in SCLC is unclear. Cell proliferation was suppressed *in vitro* by silencing *SOX2* (using short hairpin RNAs) and *MYCL1*, which implicates these genes in driving SCLC and suggests a plausible therapeutic strategy.

Original article Rudin, C. M. *et al.* Comprehensive genomic analysis identifies *SOX2* as a frequently amplified gene in small-cell lung cancer. *Nat. Genet.* doi:10.1038/ng.2405