**FCGR SNPs Affect Response to High-Dose IL2 in mRCC**

Fc-gamma receptor (FCGR) genotypes have been shown to influence patient outcome following treatment with mAb-based immunotherapeutics in different cancer settings. Erbe and colleagues show that those FCGR SNP genotypes (FCGR2A, FCGR3A, and FCGR2C) related to greater FCGR function, associate with better response following treatment with IL2 (i.e., not an mAb-based immunotherapeutic) for metastatic renal cell carcinoma patients. This work raises important questions regarding endogenous antibody responses potentially involved, either in ADCC or in antibody facilitated antigen presentation, in a variety of therapies where a role for endogenous antibody influence was not previously considered.

*See article by Erbe et al., p. 2159*

**MYD88 L265P and non-L265P DLBCL Genomic Profiles**

MYD88 mutations, notably the L265P variant, are a distinguishing feature of ABC subtype DLBCL. To explore the genomic profiles of MYD88 mutant DLBCL, Dubois and colleagues studied 361 DLBCL cases submitted to next generation sequencing, gene expression profiling, and clinical and prognostic analyses. Distinct genomic profiles for MYD88 L265P and non-L265P mutant DLBCL were highlighted; clustering analysis also segregated subgroups per associated alterations among patients with the same MYD88 mutation. Importantly, the survival of MYD88 L265P mutant ABC DLBCL was significantly improved by associated CD79B mutations. This study emphasizes the importance of genomic profiling to stratify patients for targeted therapy.

*See article by Dubois et al., p. 2232*

**Multiplex Genome Editing to Generate Universal CAR T Cells**

Ren and colleagues established a method of high-efficient multiplex CRISPR/Cas9 editing to create triple-disrupted CAR T cells by electroporating the CAR T cells with RNA of CAS9 and gRNAs. The study shows the simultaneous and highly efficient disruption of the TCR and HLA expression so that universal CAR T cells can be produced. Checkpoint-resistant universal CAR T cells via additional disruption of PD1 showed improved antitumor efficacy in preclinical mouse models. The study shows highly efficient triple ablation (80%) in primary human T cells and that the efficiency of multiplex genetic editing has been sufficient to support future clinical trials.

*See article by Ren et al., p. 2255*

**Rab37/TSP1 Inhibits Angiogenesis and Metastasis**

Cancer cells shape the microenvironment to promote malignancy by secreted factors. Tzeng and colleagues show that Rab37 small GTPase mediates the cross-talk between cancer cells and endothelial cells via exocytosis of antiangiogenic thrombospondin-1 (TSP1) for suppressing neoangiogenesis in esophageal squamous cell carcinoma (ESCC). Rab37-mediated exocytosis of TSP1 from cancer cells inhibited the activation of FAK/paxillin/ERK migration signaling in both cancer cells and their surrounding endothelial cells. Dysfunction of Rab37/TSP1 axis impaired the suppressive effects on tumor neovasculature and correlated with poor prognosis. The findings indicate a potential therapeutic value of targeting this pathway to treat ESCC.

*See article by Tzeng et al., p. 2335*
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