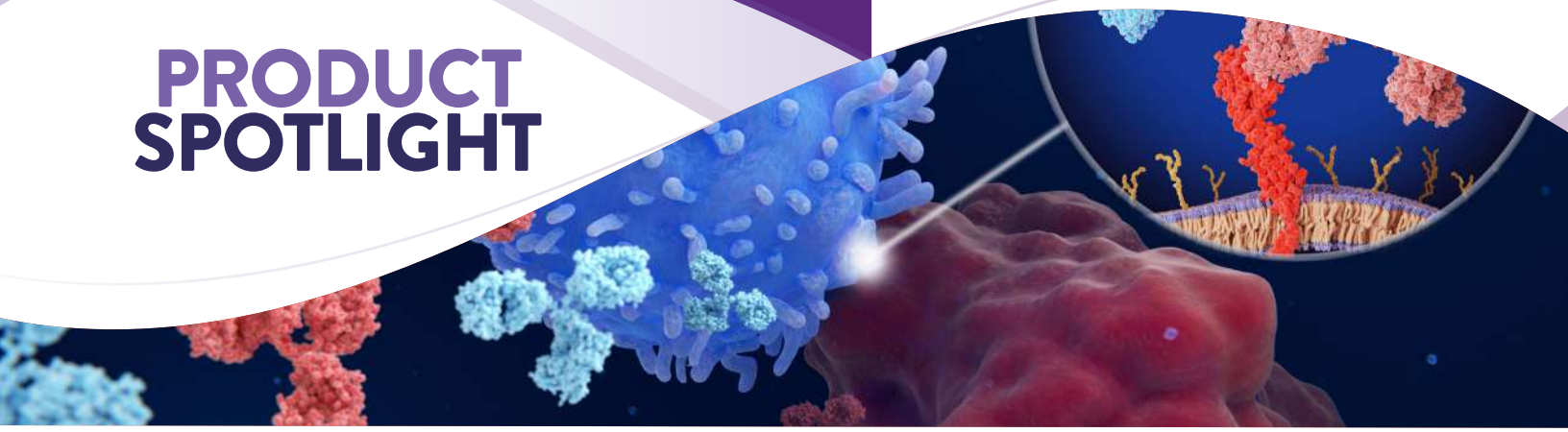
ATCC

PRODUCT SPOTLIGHT



CHECKPOINT LUCIFERASE REPORTER CELLS

Immune checkpoint inhibitors have shown recent success in the treatment of lung, liver, breast, renal, and skin cancers; however, the built-in complexity of the immunological models and the variable drug responses among different cancer types are currently the most conspicuous challenges in this area of immuno-oncology. To facilitate large scale drug discovery of this growing class of immunomodulator, ATCC created tumor and immune cell lines with high endogenous expression of checkpoint inhibitory and co-stimulatory expression levels. For easy tracking of candidate blocker efficacy, the reporter cell lines contain gamma interferon activation site (GAS)-response element or nuclear factor of activated T cells (NFAT)-response element upstream of the luciferase gene. This portfolio includes a wide range of clinically relevant targets, including PDL1/2, B7-H3, PD1, and CTLA-4. These novel cell lines can be incorporated into simple blocking assays or be integrated into sophisticated co-culture cell-based drug screening assays.

Table 1: ATCC Checkpoint Luciferase Reporter Cells

Designation	ATCC® No.	Disease	Biomarker	Tissue of origin	Status
HCC827-GAS-Luc2	CRL-2868-GAS-LUC2™	Adenocarcinoma	PD-L1	Lung	Available
MG-63-GAS-Luc2	CRL-1427-GAS-LUC2™	Osteosarcoma	CD-155	Bone	Available
NCI-H1650-GAS-Luc2	CRL-5883-GAS-LUC2™	Adenocarcinoma	B7-H3	Lung	Available
SUP-T1 [VB]-NFAT-Luc2	CRL-1942-NFAT-LUC2™	Lymphoblastic Lymphoma	PD-1	Pleural effusion	Coming soon

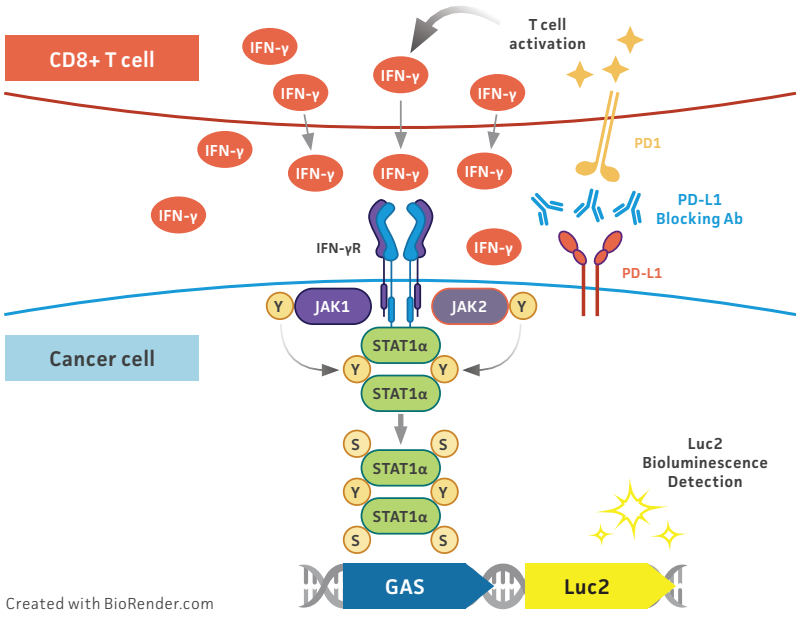


Figure 1: Mechanism of action. Luciferase signal generated by HCC827-GAS-Luc2 cells upon T cell activation through PD-L1 blockade.

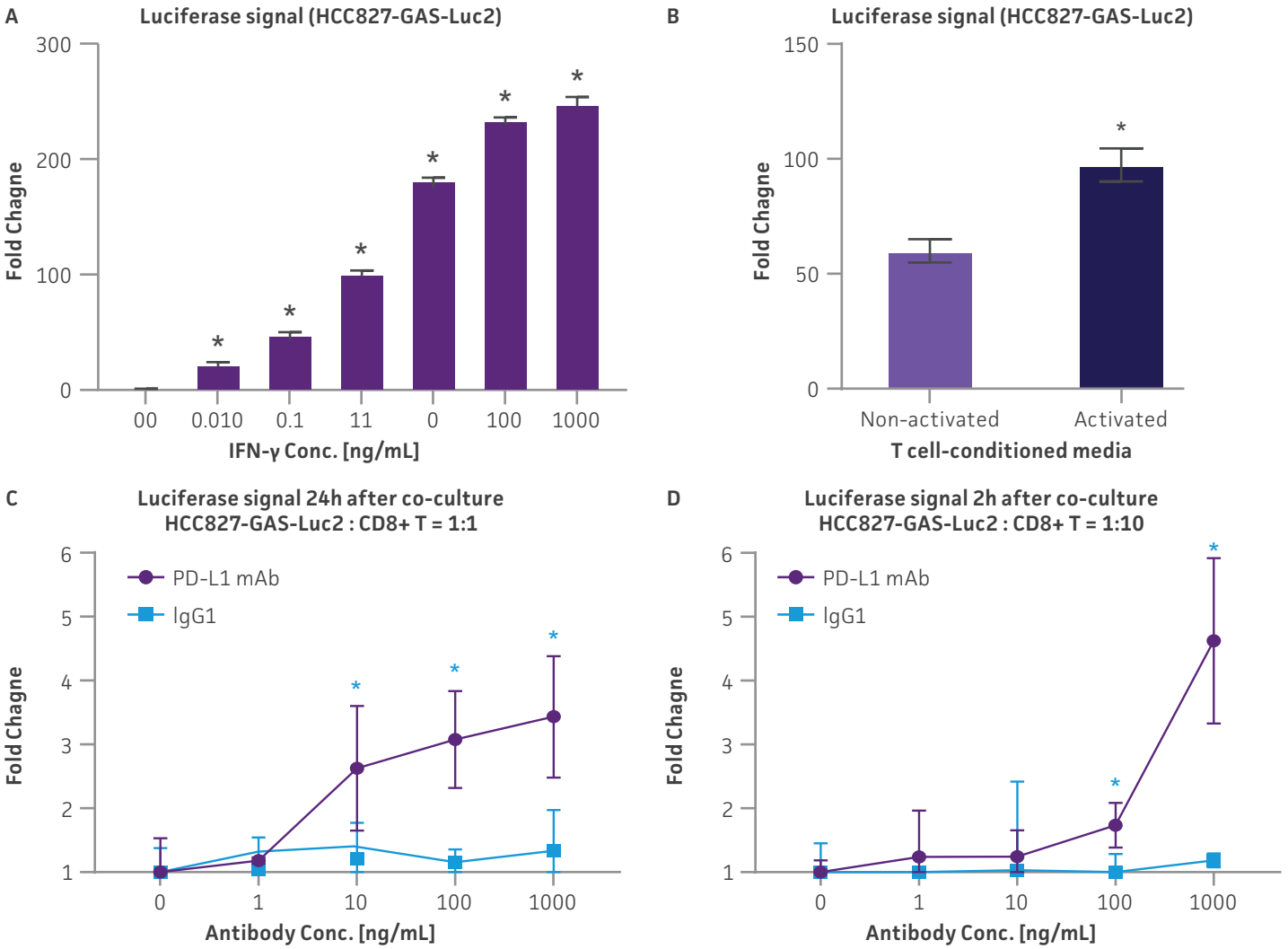


Figure 2: Evaluation of HCC827-GAS-Luc2 cell line. Luciferase expression from HCC827-GAS-Luc2 cells upon signaling activation by (A) IFN-γ stimulation (0.01 – 1,000 ng/ mL), (B) conditioned-media stimulation from checkpoint matched non-activated and activated primary CD8+ T cells, and (C, D) co-culture with primary human CD8+ T cells in the presence of PD-L1 blocking antibody or isotype control IgG1 (1-1,000 ng/mL). N=3 in all experiments. *, P < 0.05.

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