



Even with progress, there are **opportunities for advancement** in GC/GEJC/EAC^{1,2}

GC/GEJC/EAC

~40%

diagnosed as
metastatic³⁻⁶

Patients with metastatic
GC/GEJC/EAC

~6%

survive 5 years
after diagnosis^{5,6}

Although better outcomes
may be associated with
HER2-targeted therapies,⁷

~80%

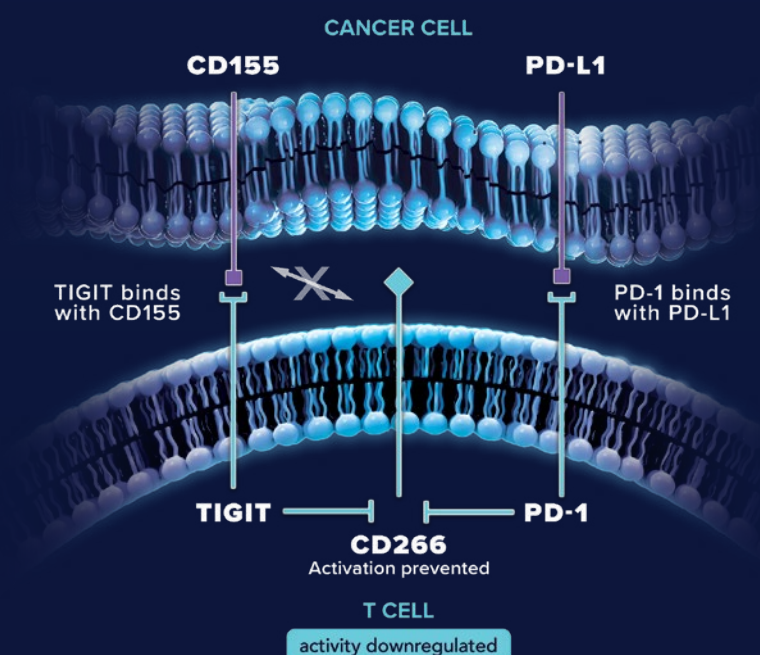
of gastroesophageal
adenocarcinoma is
HER2-negative⁸



Currently available anti-PD-1 + chemotherapy regimens result in median OS of ~13-15 months and median PFS of ~7-8 months⁹⁻¹¹

Illuminating potential for HER2-negative metastatic GC/GEJC/EAC through 2 pathways^{1,9,12,13}

TIGIT and PD-1 are among the immune checkpoints often distinctly co-expressed on activated immune cells. While PD-1 pathway activation and its associated downstream effects on immune response have been well studied in GC/GEJC/EAC, there may be opportunity for further impact. Preclinical data indicate that combined inhibition of the TIGIT and PD-1 pathways may improve T cell proliferation and may increase cytokine secretion to enhance T cell activity, potentially addressing the body's downregulated antitumor response.



Exploring the TIGIT and PD-1 pathways together may offer a novel path forward for new possibilities in HER2-negative metastatic GC/GEJC/EAC

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EAC=esophageal adenocarcinoma; GC=gastric cancer; GEJC=gastroesophageal junction cancer; HER2=human epidermal growth factor receptor 2; OS=overall survival; PD-1=programmed cell death protein 1; PD-L1=programmed cell death ligand 1; PFS=progression-free survival; TIGIT=T cell immunoglobulin and ITIM domain.

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