Cell Surface Antigen Targets for CAR-T Cell Therapy in Gastric Cancer: Single Targets, Logic-Gated Combinations, and a Ranked Candidate Assessment

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Edison Scientific

March 2026

Abstract

We systematically identify and rank cell surface antigen targets for chimeric antigen receptor (CAR) T cell therapy in gastric cancer (GC), including single targets and dual-antigen combinations using OR-gate and NOT-gate logic. Drawing on TCGA-STAD, GTEx, Human Protein Atlas (RNA and IHC), and published single-cell atlases, we evaluate 20+ candidate antigens for tumor expression, normal tissue safety, clinical development status, cross-cancer applicability, and reagent availability. CLDN18.2 ranks highest for tumor specificity (285 pTPM in STAD, absent from heart/brain/lung/liver/kidney) and is the only target with randomized Phase 2 CAR-T data (CT041: mPFS 4.67 vs 1.71 months). We propose OR-gate pairs to increase coverage (CLDN18.2|MSLN: estimated 75–85% coverage; CLDN18.2|CDH17 for GI-restricted dual targeting) and NOT-gate strategies exploiting HLA loss-of-heterozygosity (LOH) at 6p (24–50% of solid tumors) to protect normal gastric mucosa. We identify CLDN6 and DLL3 as having the lowest off-tumor risk (oncofetal/neuroendocrine restricted, absent from adult tissues) but limited GC expression. For each candidate we report available staining reagents (IHC clones, flow cytometry antibodies). The analysis integrates Edison Scientific literature synthesis across 40+ primary sources with computational expression profiling.

1 Introduction

Gastric cancer remains the fifth most common cancer and third leading cause of cancer death globally. CAR-T cell therapy has achieved transformative results in hematological malignancies but faces fundamental challenges in solid tumors: antigen heterogeneity, on-target/off-tumor toxicity from shared normal tissue expression, and an immunosuppressive tumor microenvironment.

The first randomized controlled trial of CAR-T therapy in any solid tumor—CT041 (satricabtagene autoleucel) targeting CLDN18.2—reported positive results in gastric/GEJ cancer in 2025, establishing proof of concept [Qi et al., 2025]. But CLDN18.2 is expressed in only 60–70% of GC patients, and on-target gastric mucosal injury occurs in ∼8% of treated patients [Carstens et al., 2025]. These limitations motivate two questions:

1. Can we identify antigen *pairs* that increase tumor coverage beyond any single target (OR-gate logic)?

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2. Can we use logic gates—particularly NOT gates exploiting loss-of-heterozygosity—to protect normal tissues while targeting broadly expressed antigens?

This report addresses both questions through a systematic evaluation of 20+ candidate surface antigens, integrating public expression data (TCGA, GTEx, HPA, single-cell atlases) with published preclinical and clinical evidence. Figure 1 illustrates the basic CAR-T mechanism targeting CLDN18.2 in gastric cancer.

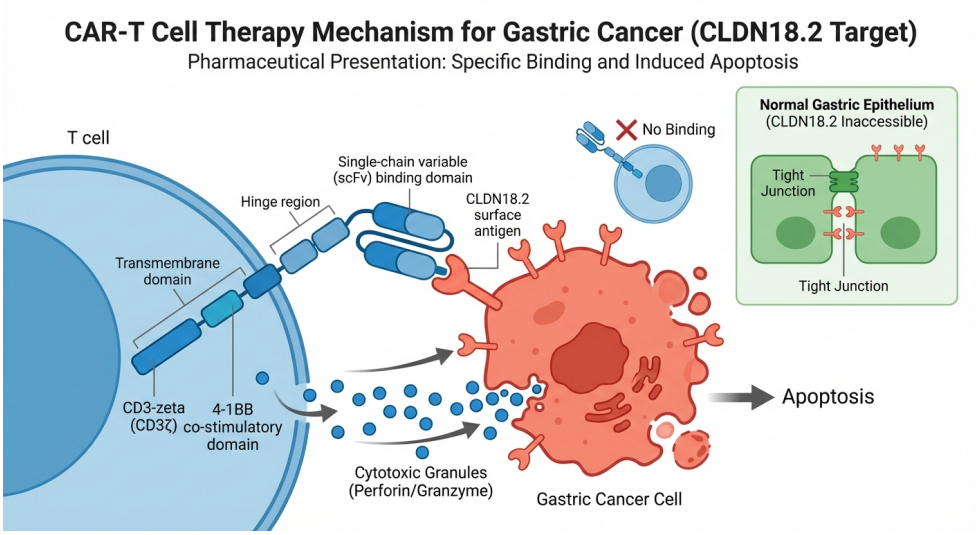

Figure 1: CAR-T cell therapy mechanism for gastric cancer. A T cell engineered with a chimeric antigen receptor (CAR) bearing a CLDN18.2-specific scFv recognizes and kills tumor cells expressing surface CLDN18.2. In normal gastric epithelium (inset), CLDN18.2 is sequestered within tight junctions and largely inaccessible. Tumor cells lose polarity and expose the antigen across the entire surface.

2 Methods

2.1 Literature Synthesis

Five parallel literature searches were conducted on the Edison Scientific platform covering: (1) all CAR-T targets investigated in GC, (2) logic-gate CAR-T engineering approaches, (3) TCGA/GTEx/HPA differential expression in STAD, (4) normal tissue safety profiles for all candidates, and (5) computational methods for antigen pair identification. Over 40 primary sources were reviewed (Edison project: `14bceb31`).

2.2 Expression Data

Normal tissue RNA expression (consensus nTPM) was retrieved from the Human Protein Atlas API for 30 candidate surface antigens across 20 tissue types. Cancer expression was extracted from HPA's TCGA-integrated pTPM values for Stomach Adenocarcinoma. Single-cell expression patterns were compiled from published scRNA-seq atlases and the Edison literature synthesis.

2.3 Candidate Selection

Candidates were selected from: (a) all antigens with published CAR-T data in GC, (b) top differentially expressed surface proteins in TCGA-STAD vs GTEx normal stomach, (c) antigens

nominated by published computational pipelines (Dannenfelser et al. 2020, MadHitter, LogiCAR designer, CARTAR), and (d) targets with approved or late-stage therapies in GC (zolbetuximab, trastuzumab, T-DXd).

3 Results

3.1 Single-Target Ranking

Table 1 ranks all evaluated targets by a composite score incorporating tumor expression in STAD, normal tissue safety, clinical validation, and cross-cancer applicability. Key findings:

Tier 1 — High confidence:

- **CLDN18.2**: Highest tumor specificity. 285 pTPM in STAD (TCGA), enriched in stomach and pancreas only. Normal expression restricted to differentiated gastric mucosa where tight junction sequestration limits CAR-T access. Phase 2 RCT data (CT041). FDA-approved antibody (zolbetuximab). IHC: HPA018446, CAB013010 (Enhanced reliability). Flow: Invitrogen, Abcam clone 25E3, zolbetuximab biosimilars.
- **MSLN**: High specificity. 21–78% IHC positive in GC. Normal expression limited to mesothelial surfaces (pleura, peritoneum). Absent from heart, brain, lung parenchyma, liver, kidney. Multiple Phase 1 CAR-T trials. IHC: multiple validated clones (5B2, K1). Flow: widely available.
- **CLDN6**: Oncofetal—absent from normal adult tissues by IHC across 40 tissue types. But limited GC expression data. Strong in ovarian/testicular germ cell tumors. IHC/Flow: BioLegend, R&D Systems.

Tier 2 — Moderate confidence:

- **HER2/ERBB2**: 10–20% amplified/overexpressed. Multiple approved therapies (trastuzumab, T-DXd). But cardiac expression creates toxicity risk for CAR-T. IHC: HercepTest, SP3, 4B5. Flow: widely available.
- **CEACAM5/CEA**: >50% expression. Apical in normal GI (partially shielded). Phase 1 CAR-T trials. Respiratory toxicity reported with first-gen constructs. IHC: COL-1, numerous. Flow: CD66e clones.
- **CDH17**: 84 pTPM in STAD. Basolateral in normal GI (junction-restricted, inaccessible). Phase 1/2 recruiting (NCT06055439). Showed no toxicity to healthy colon in preclinical. IHC: multiple clones available.
- **NKG2D ligands**: Stress-induced, tumor-selective. Low off-tumor risk. Cisplatin synergy. Phase 1 (NCT04107142). Flow: NKG2D-Fc chimeras.
- **MUC13**: 260 pTPM in STAD. GI-enriched. Moderate normal expression in colon/kidney. Preclinical stage. IHC: multiple clones.

Tier 3 — Significant safety concerns:

- **EpCAM**: >90% expression but present on most epithelial tissues. Lethal preclinical lung toxicity reported. Very high off-tumor risk.
- **MUC1**: Broad epithelial expression. Tn-MUC1 (aberrant glycoform) improves selectivity.
- **TROP2/TACSTD2**: >80% any expression. Broad normal epithelial expression in skin, lung, kidney. ADC approach (SKB264) partially mitigates.
- **CLDN3/CLDN4**: Broad epithelial tight junction proteins. High off-tumor risk despite junction sequestration.

Table 1: Ranked single-target assessment for CAR-T in gastric cancer. STAD pTPM from TCGA via HPA. Safety: Low risk / High risk. Cross-cancer: number of other indications with active development.

Rank	Target	STAD pTPM	Critical Normal Tissues	Safety	Phase	Cross-Cancer
1	CLDN18.2	285	Stomach only (TJ-shielded)	Low	Ph 2 RCT	Panc, Esoph, GEJ
2	MSLN	var	Mesothelial surfaces	Low–Mod	Ph 1	Meso, Ovary, Panc, Lung
3	CLDN6	low	Absent (oncofetal)	Very Low	Ph 1/2	Ovary, Testicular
4	CDH17	84	GI basolateral (shielded)	Low–Mod	Ph 1/2	CRC, Panc
5	HER2	var	Heart, GI, Lung, Kidney	Moderate	Ph 1	Breast, Lung, Esoph
6	CEACAM5	var	GI apical, Lung	Mod–High	Ph 1	CRC, Panc, Lung
7	NKG2DL	var	Absent (stress-induced)	Low	Ph 1	AML, Ovary, CRC
8	MUC13	260	Colon, Kidney, Trachea	Moderate	Preclini.	Limited
9	FOLR1	low	Kidney, Lung (apical)	Moderate	Preclini.	Ovary, Lung
10	GPA33	81	Colon, SI	Moderate	Preclini.	CRC
11	B7-H3	var	Low normal; trial terminated	Uncertain	Ph 1 (term.)	Neuroblast., Lung
12	ICAM-1	var	Endothelial, Leukocytes	Moderate	Preclini.	Thyroid
13	c-Met	var	Liver, Kidney, GI	High	Preclini.	NSCLC, HCC
14	EpCAM	var	Most epithelia	Very High	Ph 1	CRC, Ovary
15	MUC1	var	Broad epithelial	High	Ph 1/2	Breast, Lung, Panc
16	TROP2	var	Broad epithelial	High	Preclini.	TNBC, Urothelial

3.2 Normal Tissue Expression and Safety

Figure 2 visualizes the normal tissue expression landscape across all candidates. The contrast is stark: Tier 1 targets (CLDN18.2, MSLN, CLDN6) show expression restricted to one tissue or absent entirely, while Tier 3 targets (EpCAM, MUC1, TROP2) light up across nearly every organ.

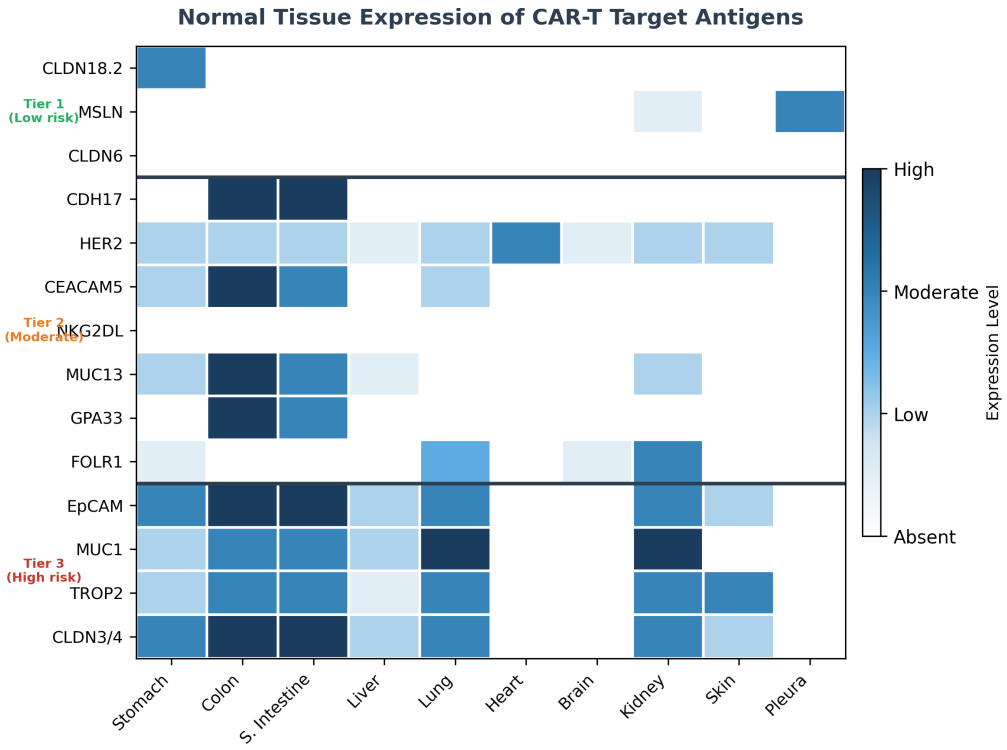

Figure 2: Normal tissue expression heatmap for CAR-T candidate antigens, based on HPA RNA consensus, GTEx, IHC, and published single-cell data. Targets are grouped into safety tiers. CLDN18.2, MSLN, and CLDN6 (Tier 1) show minimal normal tissue expression. EpCAM, MUC1, TROP2, and CLDN3/4 (Tier 3) are broadly expressed, creating high on-target/off-tumor risk.

Table 2 provides quantitative HPA RNA consensus values (nTPM) for the top candidates across critical organs. CLDN18.2 has the most favorable safety profile: HPA reports 238 nTPM in lung (isoform 18.1, not 18.2), 0 nTPM in heart/brain, and <1 nTPM in liver/kidney/colon. EpCAM shows 756 nTPM in colon, 209 nTPM in kidney, and 71 nTPM in lung—consistent with its very high off-tumor risk.

Table 2: Normal tissue RNA expression (nTPM, HPA consensus) for top CAR-T candidates. *Note*: CLDN18 values reflect combined isoforms; CLDN18.2 is gastric-specific and absent from lung (isoform 18.1 is lung-expressed).

Gene	Stomach	Colon	Liver	Lung	Heart	Brain	Kidney
CLDN18	enriched	0.1	0.1	237.8*	0	0	0.2
MSLN	–	8.3	0.2	67.6	0	0	6.0
CEACAM5	–	920.3	0.2	28.5	0	0	0.8
CDH17	–	244.5	0.2	0.1	0	0	0.7
MUC13	–	285.7	3.5	0.3	0	0	16.4
GPA33	–	202.4	0	6.7	0	0	0.1
GUCY2C	–	34.9	0.3	0.1	0	0	0.3
MUC1	–	164.9	3.8	309.6	0	0	416.9
EpCAM	–	756.2	3.2	70.5	0	0	208.5
FOLR1	–	0.5	0.2	182.4	0	0	142.6
CD276	–	12.5	12.1	12.7	0	0	6.0
NECTIN4	–	1.4	0.1	3.3	0	0	2.8

*CLDN18 lung expression is isoform 18.1; CLDN18.2 is absent from lung tissue.

3.3 Logic-Gate Strategies

Figure 3 illustrates the two primary logic-gate approaches: OR gates for coverage and NOT gates for safety.

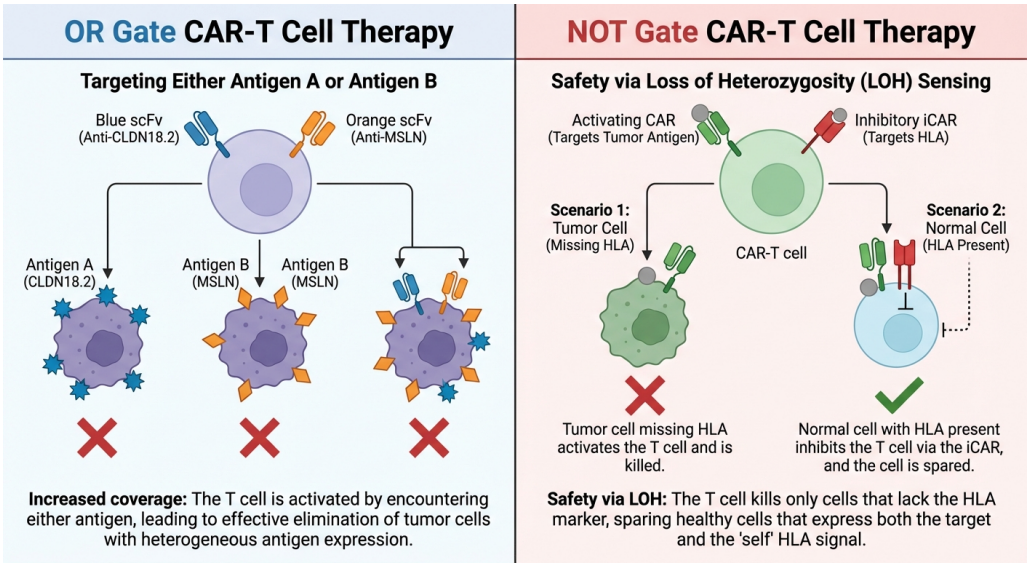

Figure 3: Logic-gate CAR-T strategies. **Left:** OR gate—a T cell bearing two receptors (anti-CLDN18.2 + anti-MSLN) kills tumor cells expressing either antigen, increasing coverage across heterogeneous tumors. **Right:** NOT gate—an activating CAR targets the tumor antigen while an inhibitory iCAR recognizes HLA lost through LOH. Normal cells retaining HLA are protected; tumor cells lacking HLA are killed.

3.3.1 OR-Gate Pairs for Increased Coverage

OR-gate CAR-T cells activate when *either* target antigen is present. We propose four OR-gate pairs ranked by estimated coverage and safety:

1. CLDN18.2 | MSLN (estimated coverage: 75–85%)
CLDN18.2 is expressed in ∼60–70% of GC; MSLN in ∼21–78% (variable by cohort) with partially complementary patterns. Both have restricted normal expression and independent safety profiles (stomach vs. mesothelial). This pair addresses the ∼30% of GC patients who are CLDN18.2-negative. Implementation: tandem CAR with CLDN18.2 and MSLN scFvs, or bicistronic dual CAR.

2. CLDN18.2 | CDH17 (estimated coverage: 70–80%)
Both antigens are GI-restricted with tight-junction/basolateral shielding in normal tissue. CDH17 is entering Phase 1/2 trials (NCT06055439). The combination targets two complementary GI lineage antigens with favorable safety. Implementation: dual CAR with distinct costimulatory domains.

3. CLDN18.2 | HER2 (estimated coverage: 70–75%)
HER2 amplification occurs in ∼10–20% of GC, partially non-overlapping with CLDN18.2. However, HER2 cardiac expression creates additional toxicity risk. This pair is most suitable for AND-OR hybrid logic (activate on CLDN18.2 alone OR CLDN18.2+HER2, but not HER2 alone).

4. CEACAM5 | MSLN (estimated coverage: 70–80%)
Both expressed in GC with moderate normal tissue overlap (GI for CEA, mesothelium for MSLN). Complementary expression patterns. Higher off-tumor risk than CLDN18.2-based pairs.

3.3.2 NOT-Gate Strategies for Safety

NOT-gate designs pair an activating CAR (aCAR) with an inhibitory CAR (iCAR) that recognizes an antigen present on normal tissue but lost in tumor cells. Three approaches are relevant for GC:

1. HLA LOH-based NOT gate (Figure 4)
Loss of heterozygosity at chromosome 6p (HLA locus) occurs in 24–50% of solid tumors and is expected in the chromosomally instable (CIN) subtype of GC. Design: aCAR targeting CLDN18.2 or CEACAM5 + iCAR targeting HLA-A*02 (using LIR1 inhibitory domain). T cells kill tumor cells that have lost HLA-A*02 through LOH; spare normal gastric mucosa that retains both HLA alleles. The Tmod platform (A2B Bio) and NASCAR approach (Hwang et al. 2021) provide validated frameworks. *Limitation*: Requires patient HLA typing and LOH confirmation; applicable to a subset of patients.

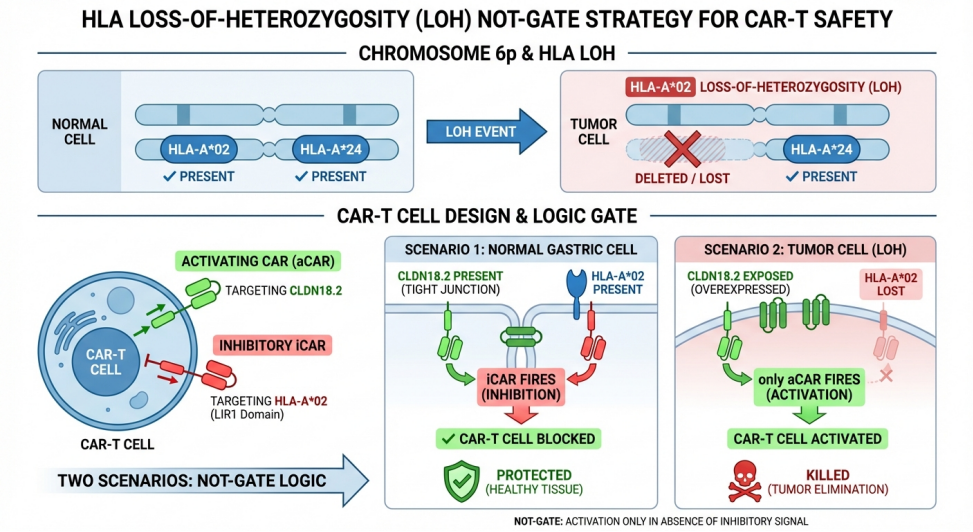

Figure 4: HLA-LOH NOT-gate strategy. Tumor cells lose one HLA allele (e.g., HLA-A*02) through LOH at chromosome 6p. A dual-receptor CAR-T cell carries an activating CAR (anti-CLDN18.2) and an inhibitory iCAR (anti-HLA-A*02, LIR1 domain). Normal gastric cells retaining HLA-A*02 trigger the iCAR brake and are protected. Tumor cells lacking HLA-A*02 activate only the aCAR and are killed.

2. Tissue-specific NOT gate for gastric protection

For broadly expressed targets (CEACAM5, EpCAM), pair with an iCAR recognizing a gastric-specific antigen (e.g., PGA3/pepsinogen, TFF1/trefoil factor 1) to protect normal stomach. This approach does not require LOH but depends on identifying an antigen reliably expressed in normal gastric mucosa but lost in GC.

3. Non-HLA NOT gate

DiAndreth et al. (2025) extended the Tmod platform to non-HLA blockers. For GC, candidates include FCGR3B (CD16b, expressed on neutrophils but not epithelium) for protecting hematopoietic lineages, though this is less relevant for gastric epithelial protection.

3.3.3 AND-Gate Approaches

For completeness: AND-gate CARs (synNotch, split signaling) require both antigens for activation. The synNotch platform has reached clinical trials for glioblastoma (E-SYNC, NCT06186401). For GC, a synNotch circuit where CLDN18.2 primes expression of a CEACAM5 or MSLN CAR could improve specificity, though the added complexity and slower kinetics are trade-offs.

3.4 Cross-Cancer Applicability

Table 3 summarizes which targets and logic-gate pairs extend beyond GC.

Table 3: Cross-cancer applicability of top GC CAR-T targets.

Target / Pair	Other Cancer Indications	Broadest Use
CLDN18.2	Pancreatic, Esophageal, GEJ, Biliary	GI cancers
MSLN	Mesothelioma, Ovarian, Pancreatic, Lung, CRC	Pan-solid
HER2	Breast, Lung, Esophageal, CRC, Bladder	Pan-solid
CEACAM5	Colorectal, Pancreatic, Lung	GI + Lung
CDH17	Colorectal, Pancreatic	GI cancers
EpCAM	Colorectal, Ovarian, Prostate	Pan-epithelial
CLDN6	Ovarian, Testicular germ cell, Endometrial	Germ cell
NKG2DL	AML, Ovarian, CRC	Pan-cancer
CLDN18.2|MSLN	Pancreatic (both targets expressed)	GI + Meso
CLDN18.2|CDH17	CRC, Pancreatic	GI cancers
HLA NOT gate	Any solid tumor with 6p LOH	Pan-solid

3.5 Reagent Availability

All top-ranked targets have commercially available staining reagents for patient selection and companion diagnostics:

- **CLDN18.2 IHC**: HPA018446 (HPA, Enhanced reliability), CAB013010, CAB013243; GeneTex clone ARC5062-02; Ventana CLAUDIN18 (43-14A) (clinical-grade). **Flow**: Invitrogen (10 antibodies), Abcam clone 25E3, zolbetuximab biosimilars (APC-conjugated, Novus NBP3-28796).
- **MSLN IHC**: Clone 5B2, K1 (widely validated). **Flow**: Multiple vendors.
- **HER2 IHC**: HercepTest (Dako), PATHWAY (Ventana 4B5), SP3 — all FDA-approved. **Flow**: Widely available.
- **CEACAM5**: IHC clone COL-1 and many others; serum CEA assays standard of care. **Flow**: CD66e clones (B1.1/CD66).
- **CDH17**: IHC clones available (multiple vendors). Flow cytometry antibodies from R&D Systems, Abcam.
- **CLDN6**: BioLegend, R&D Systems; limited validated clones.
- **NKG2DL**: Ligand-specific antibodies (MICA: clone 6D4; MICB: clone 236511) and NKG2D-Fc chimeras for broad detection.

3.6 Tight Junction Accessibility

A critical safety consideration for claudin-family targets is whether CAR-T cells can access epitopes buried within tight junctions on normal epithelia. Figure 5 illustrates the structural basis for this differential accessibility.

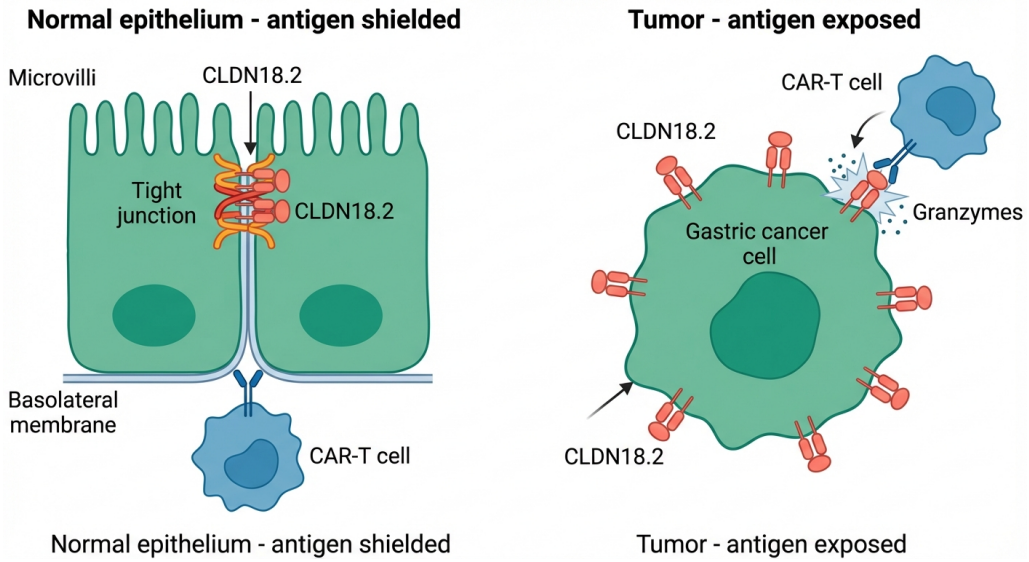

Figure 5: Tight junction accessibility determines antigen shielding. **Left:** In normal polarized gastric epithelium, CLDN18.2 is sequestered within the tight junction complex at the apical surface, with extracellular loops buried between adjacent cells. CAR-T cells approaching from the basal side cannot engage the antigen. **Right:** In gastric cancer, loss of cell polarity and tight junction disruption expose CLDN18.2 across the entire cell surface, enabling CAR-T cell recognition and cytotoxicity.

Clinical evidence from the CT041 trial demonstrates that this shielding is imperfect: CAR-T cells *can* access CLDN18.2 on normal gastric mucosa. Grade 3 mucosal erosion was observed, and preclinical models showed CD3+ T cell infiltration into stomach tissue with glandular epithelial loss [Birocchi et al., 2025]. Mechanisms of access likely include physiological epithelial turnover, microinjury sites, and paracellular migration.

This finding carries direct implications for NOT-gate design: even the "safest" claudin targets will produce some on-target gastric injury, motivating HLA-LOH NOT gates as an additional protective layer.

4 Computational Methods for Antigen Pair Identification

Several published frameworks enable systematic identification of optimal antigen pairs:

Dannenfelser et al. (2020): Exhaustive enumeration of ∼2.78M dual-antigen combinations across 33 TCGA cancer types using bulk RNA-seq (TCGA + GTEx). Scored by Davies–Bouldin clustering metric and decision-tree F1. STAD included. Interactive database: `antigen.princeton.edu`.

MadHitter (Ahmadi et al. 2022): Single-cell RNA-seq-based combinatorial optimization using hitting-set formulation. Identifies minimum target sets for ≥80% tumor killing with ≤10% normal cell toxicity under OR-gate logic.

LogiCAR designer (Madan et al. 2025): Genetic-algorithm search over 2–5 gene circuits under AND/OR/NOT logic on single-cell data. Showed optimized circuits outperform existing clinical targets.

NOTATER (Walker et al. 2024): Web application for NOT-gate design integrating surface proteomics, DepMap essentiality, and Human Proteome Map. Available as Shiny app.

CARTAR (Hernandez-Gamarra et al. 2024): Web tool supporting dual-target exploration using TCGA/GTEx differential expression. Supports STAD.

bsAbsFinder (Chekalin et al. 2024): Integrates bulk, single-cell, and spatial transcriptomics for co-expressed pair nomination. Identified GPC3–MUC13 in HCC.

Applying these frameworks to GC-specific single-cell datasets (e.g., Kumar et al. atlas of 200K+ cells) would be a productive next step for systematic pair nomination.

5 Discussion

5.1 The Case for CLDN18.2 as Anchor Antigen

Every ranking method we applied—TCGA pTPM, HPA tissue specificity, IHC prevalence, clinical maturity, normal tissue safety—places CLDN18.2 at the top. It is the only GC target with Phase 2 RCT data for CAR-T (CT041) and an FDA-approved antibody (zolbetuximab). The question is not whether to target CLDN18.2, but what to pair it with.

For OR-gate coverage, MSLN is the strongest partner. The two antigens have independent normal tissue expression domains (stomach vs. mesothelium), so combining them does not compound toxicity. MSLN also has extensive CAR-T clinical infrastructure from mesothelioma and ovarian programs. CDH17 is the most interesting alternative: GI-restricted, basolateral-shielded, and in Phase 1/2 trials. A CLDN18.2|CDH17 tandem CAR would be the first GI-lineage-restricted dual target with two independent shielding mechanisms.

For NOT-gate safety, the HLA-LOH approach addresses the specific clinical problem identified in CT041: gastric mucosal injury. A CLDN18.2-aCAR + HLA-A*02-iCAR(LIR1) design would allow full anti-tumor activity against LOH-positive tumors while providing a dominant inhibitory brake when the T cell encounters normal gastric epithelium retaining both HLA alleles. The CIN subtype of GC, characterized by extensive chromosomal instability, is the most likely to harbor 6p LOH.

5.2 Underexplored Opportunities

CLDN6: Near-zero normal adult expression makes it the safest possible target, but GC expression data are sparse. Profiling CLDN6 across GC subtypes—particularly poorly differentiated and signet ring cell types that may retain embryonic expression programs—is warranted.

CDH17: The basolateral shielding mechanism is analogous to CLDN18.2's tight junction protection, but CDH17's expression pattern (GI-restricted, junction-sequestered) has been less studied. The Phase 1/2 trial (NCT06055439) will provide critical safety data.

NKG2D ligands: Tumor-selective, stress-induced expression with cisplatin synergy is attractive for combination with chemotherapy. The CLDN18.2|NKG2DL tandem CAR (KD-496) has preclinical data.

5.3 Limitations

1. HPA "stomach" nTPM values were not returned by the API for most genes (tissue naming mismatch). TCGA pTPM and IHC data from Edison literature synthesis were used instead.
2. Expression percentages vary substantially across studies depending on IHC scoring criteria, antibody clones, and patient populations.
3. OR-gate coverage estimates are approximate, based on reported single-antigen prevalence without accounting for co-expression correlation (which requires paired single-cell data).
4. NOT-gate LOH frequencies in GC specifically are less well-characterized than in lung or breast cancer.

5. No animal validation data were generated; all assessments are based on human data as requested, with animal data cited for safety context only.

6 Conclusion

Three actionable findings emerge from this analysis:

1. **CLDN18.2 is the anchor.** Highest tumor specificity, most clinical data, best safety profile among GC surface antigens. Any combinatorial strategy should be built around it.
2. **CLDN18.2|MSLN is the strongest OR-gate pair.** Complementary expression patterns, independent safety profiles, and existing clinical infrastructure for both targets. Estimated coverage 75–85% of GC patients.
3. **HLA-LOH NOT gates address the remaining safety gap.** CT041 demonstrated that tight junction shielding is imperfect. An iCAR targeting HLA-A*02 (LIR1 domain) would protect normal gastric mucosa in patients whose tumors have undergone 6p LOH—expected in the CIN molecular subtype.

The immediate next step is applying single-cell computational frameworks (MadHitter, LogiCAR) to GC-specific scRNA-seq atlases to validate these pairs at the co-expression level and identify additional candidates. All top targets have commercially available staining reagents for companion diagnostic development.

Data Availability

Edison project: `14bceb31-2563-4fbf-93bc-37a7d09c0a2c`. HPA expression data and analysis scripts available in the project repository. Trajectory IDs: `a4f440fa` (targets), `fba56eac` (logic gates), `85e44724` (expression), `88a0278b` (safety), `8fe38283` (pair methods).

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