Below is the **Agent Table**, organized by target class.
Each entry includes **specific perturbation agents**, **mechanism**, **development stage**, **indications where studied**, and **source citations**.
All items are supported by the web-search results you requested.

AGENT TABLE — Therapeutic Agents Relevant to Suggested Pathways

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1. SDC1-TARGETING AGENTS (SYNDECAN-1)

Agent	Type	Mechanism	Development Stage / Evidence	Indications Studied	Citation
22B monoclonal antibody (anti-SDC1 mAb)	mAb	Binds SDC1; inhibits macropinocytosis; induces ADCC	**Preclinical (in vivo PDAC models)**: inhibits PDAC growth, synergizes with KRAS inhibitors, chemo, immunotherapy	Pancreatic cancer (PDAC)	[1]
SDC1-targeted α-emitter conjugate 211At-9E7.4	Radiolabeled mAb	SDC1-targeted α-particle therapy	**Preclinical**; durable tumor control in glioblastoma models	Glioblastoma	[2]

[1] https://aacrjournals.org/cancerres/article/84/17_Supplement_2/C019/747683/Abstract-C019-A-novel-SDC1-targeted-therapeutic

[2] https://www.thelancet.com/journals/ebiom/article/PIIS2352-3964%2824%2900237-8/fulltext

YAP1–SDC1 axis inhibitors (e.g., indirectly via KRAS inhibitor context)*	Not a single agent, but a mechanistic vulnerability	Target SDC1 re-expression after KRAS inhibition	**Preclinical / translational**	KRAS-mutant GI cancers	[3]
SDC1-targeted ADCs / theranostics	Proposed	Antibody-drug conjugates under development	**Conceptual / early preclinical**	PDAC	[4]

2. CD44-TARGETING AGENTS

Agent	Type	Mechanism	Development Stage	Indications Studied	Citation
Hyaluronic-acid (HA)–based CD44-targeting nanocarriers	Nanoparticle delivery platforms	CD44-dependent uptake to deliver cytotoxics or siRNA	**Preclinical**	Pancreatic and multiple solid tumors	[5]
CD44 shRNA / CRISPR (gene knockout)	Genetic intervention	Eliminates CD44; reduces invasion, increases chemosensitivity	**Preclinical**	Pancreatic cancer	[6]
CD44v3 targeting (splicing modulation)	RNA-targeted	Knockdown of oncogenic CD44V3 splice variant	**Preclinical**	Pancreatic cancer	[7]

[3] https://www.cell.com/cell-reports-medicine/fulltext/S2666-3791%2825%2900326-X

[4] https://aacrjournals.org/cancerres/article/84/17_Supplement_2/C019/747683/Abstract-C019-A-novel-SDC1-targeted-therapeutic

[5] https://www.frontiersin.org/journals/pharmacology/articles/10.3389/fphar.2021.800481/full

[6] https://www.researchsquare.com/article/rs-3677039/v1

[7] https://www.mdpi.com/1422-0067/23/20/12061

3. INTEGRIN-TARGETING AGENTS (αv, β1, β8, α2β1 etc.)

3A. Broad αv/β-family inhibitors

Agent	Type	Mechanism	Development Stage	Indications Studied	Citation
PLN-101095 (αvβ1/αvβ8 inhibitor)	Small molecule	Dual αvβ1/β8 inhibitor; blocks TGF-β activation and enhances T-cell infiltration	**Clinical trials (NCI listing)**	Solid tumors expressing αvβ1/β8	[8]
Peptide 5a (targets αvβ6/αvβ8)	Peptide–HSA conjugate	Blocks integrin-mediated activation of latent TGF-β	**Preclinical in multiple tumor models including PDAC**	PDAC, prostate, mammary tumors	[9]
ADWA-11 (anti-αvβ8 antibody)	mAb	Blocks TGF-β activation via αvβ8; enhances CD8 T cell cytotoxicity	**Preclinical**	Multiple solid tumors	[10]

3B. ITGA2-related or collagen-binding integrins

(Currently no selective α2β1 inhibitors in oncology trials; inhibition explored mainly preclinically)

Agent	Type	Mechanism	Stage	Notes	Citation
None clinically advanced	—	—	—	ITGA2 remains mechanistically implicated but **no dedicated clinical-stage inhibitors**	[11]

4. TIGIT-TARGETING IMMUNOTHERAPIES (NECTIN2/TIGIT AXIS)

Agent	Type	Mechanism	Development Stage	Indications	Citation

[8] https://www.cancer.gov/publications/dictionaries/cancer-drug/def/alphavbeta1-8-inhibitor-pln-101095

[9] https://link.springer.com/article/10.1186/s13046-025-03352-4

[10] https://www.cell.com/cell-reports/fulltext/S2211-1247%2821%2900685-9

[11] https://www.nature.com/articles/s41573-021-00284-4.pdf

Tiragolumab (anti-TIGIT mAb)	mAb	Blocks TIGIT to release T/NK-cell suppression	**Phase 1b / Phase 2 trials**	Solid tumors, CUP	[12] [13]
Domvanalimab (Fc-silent anti-TIGIT)	mAb	Anti-TIGIT, engineered Fc-silent design	**Phase II positive signal in GI cancer**	Gastroesophageal adenocarcinoma	[14]
AZD2936 (TIGIT/PD-1 bispecific)	Bispecific antibody	Dual inhibition of TIGIT + PD-1	**Phase I/II**	NSCLC	[15]
Multiple next-generation anti-TIGIT candidates	Various	Combination-focused TIGIT blockade	**Preclinical–clinical**	Multiple cancers	[16]

5. WNT5A–PATHWAY–TARGETING AGENTS

Agent	Type	Mechanism	Development Stage	Indications	Citation
Box5 (WNT5A antagonist peptide)	Synthetic peptide	Blocks WNT5A signaling, inhibits Ca^{2+} release	**Preclinical**	Melanoma; Fibrosis/AKI	[17] [18]
IWP2 (WNT secretion inhibitor)	Small molecule	Inhibits porcupine → blocks WNT ligands	**Preclinical**	WNT-dependent cancers; granulosa cell models	[19]

[12] https://jamanetwork.com/journals/jamaoncology/fullarticle/2810007

[13] https://clinicaltrials.gov/study/NCT06754501

[14] https://www.clinicaltrialsarena.com/news/gilead-arcus-anti-tigit-domvanalimab-phase-ii/

[15] https://www.astrazenecaclinicaltrials.com/study/D7020C00001/

[16] https://link.springer.com/article/10.1007/s00262-025-04128-7

[17] https://www.medchemexpress.com/box5.html

[18] https://www.cell.com/molecular-therapy-family/molecular-therapy/fulltext/S1525-0016%2825%2900490-3

[19] https://synapse.patsnap.com/drug/a2f22731036c468b9bad14d57980a229

BERA-Wnt5a siRNA	siRNA therapeutic	Silences Wnt5a to overcome resistance	**Preclinical (in vivo)**	Prostate cancer	[20]

6. LTB4 / LTA4H–PATHWAY–TARGETING AGENTS

(Relevant for inflammatory neutrophil-rich microenvironments and TAN-associated PDAC biology)

6A. LTB4R (BLT1/2) inhibitors

Agent	Type	Mechanism	Stage	Indications	Citation
LTB4R antagonists (class)	Small molecules	Block LTB4–BLT1/BLT2 signaling	**Preclinical–early translational**	Inflammatory diseases; cancer-associated inflammation	[21]
LTB4-IN-2	Small molecule	Potent BLT1 antagonist	**Preclinical (detailed evaluation)**	Neutrophil-driven inflammation	[22]

6B. LTA4H inhibitors

Agent	Type	Mechanism	Development Stage	Indications	Citation
LYS006 (LTA4H inhibitor)	Small molecule	Inhibits LTA4H → ↓LTB4 production	**Phase I completed; moving to Phase II**	Neutrophil-driven inflammatory diseases	[23]
Bestatin (classical LTA4H inhibitor)	Small molecule	Inhibits LTA4H and reduces tumor LTB4 levels	**Clinical sample–supported, preclinical PDX evidence**	Colorectal cancer	[24]

[20] https://aacrjournals.org/mct/article/21/10/1594/709526/Bioengineered-BERA-Wnt5a-siRNA-Targeting-Wnt5a

[21] https://synapse.patsnap.com/article/what-are-ltb4r-antagonists-and-how-do-they-work

[22] https://www.benchchem.com/pdf/Preclinical_Evaluation_of_LTB4_IN_2_A_Comprehensive_Technical_Guide.pdf

[23] https://ascpt.onlinelibrary.wiley.com/doi/pdf/10.1111/cts.13724

[24] https://www.thelancet.com/article/S2352-3964%2819%2930309-3/fulltext

Multiple selective LTA4H inhibitors (technical series)	Small molecules	Target epoxide hydrolase vs aminopeptidase activity	**Preclinical**	Inflammation and cancer	[25]

7. IGFBP3 / TMEM219-TARGETING AGENTS

Agent	Type	Mechanism	Development Stage	Indications	Citation
Anti-IGFBP3 antibodies (various, patent US20230039165A1)	mAbs	Block IGFBP3 binding to TMEM219	**Preclinical (patent-described)**	Cancer, autoimmune disorders	[26]
Anti-TMEM219 antibodies (Ent001 etc.)	mAbs	Block IGFBP3–TMEM219 interaction → prevent apoptosis	**Preclinical (intestinal stem cell, autoimmune models)**	Crohn's disease, IBD, potentially pancreas	[27]
ecto-TMEM219 recombinant protein	Soluble decoy receptor	Blocks IGFBP3 → prevents TMEM219-driven apoptosis	**Preclinical (intestinal and colitis models)**	IBD; stem-cell preservation	[28]
Pipeline biologics from Enthera (selective inhibitors)	Biologics	Restore IGFBP3/TMEM219 balance	**Preclinical–translational**	T1D, IBD	[29]

[25] https://pdf.benchchem.com/15574/The_Selectivity_of_LTA4H_Inhibitors_A_Technical_Guide.pdf

[26] https://patents.google.com/patent/US20230039165A1/en

[27] https://crcpediatrico.org/wp-content/uploads/2025/10/9.3Pharmacol-Res-2025.pdf

[28] https://www.jci.org/articles/view/185783

[29] https://www.entherapharmaceuticals.com/our-science/

HOW TO USE THIS TABLE

If you want, I can now:

✅ Generate **subset-specific tables** (e.g., only CAF–PDAC targeting agents)
✅ Generate **"model system suitability"** columns (e.g., organoids vs xenografts)
✅ Create a **ranked list** of agents most relevant to your PDAC responder/non-responder differentials
✅ Produce a **PDF-ready formatted appendix** for manuscripts or grants

Just tell me: **"Expand table with X"** or **"Rank agents by suitability for PDAC functional assays."**