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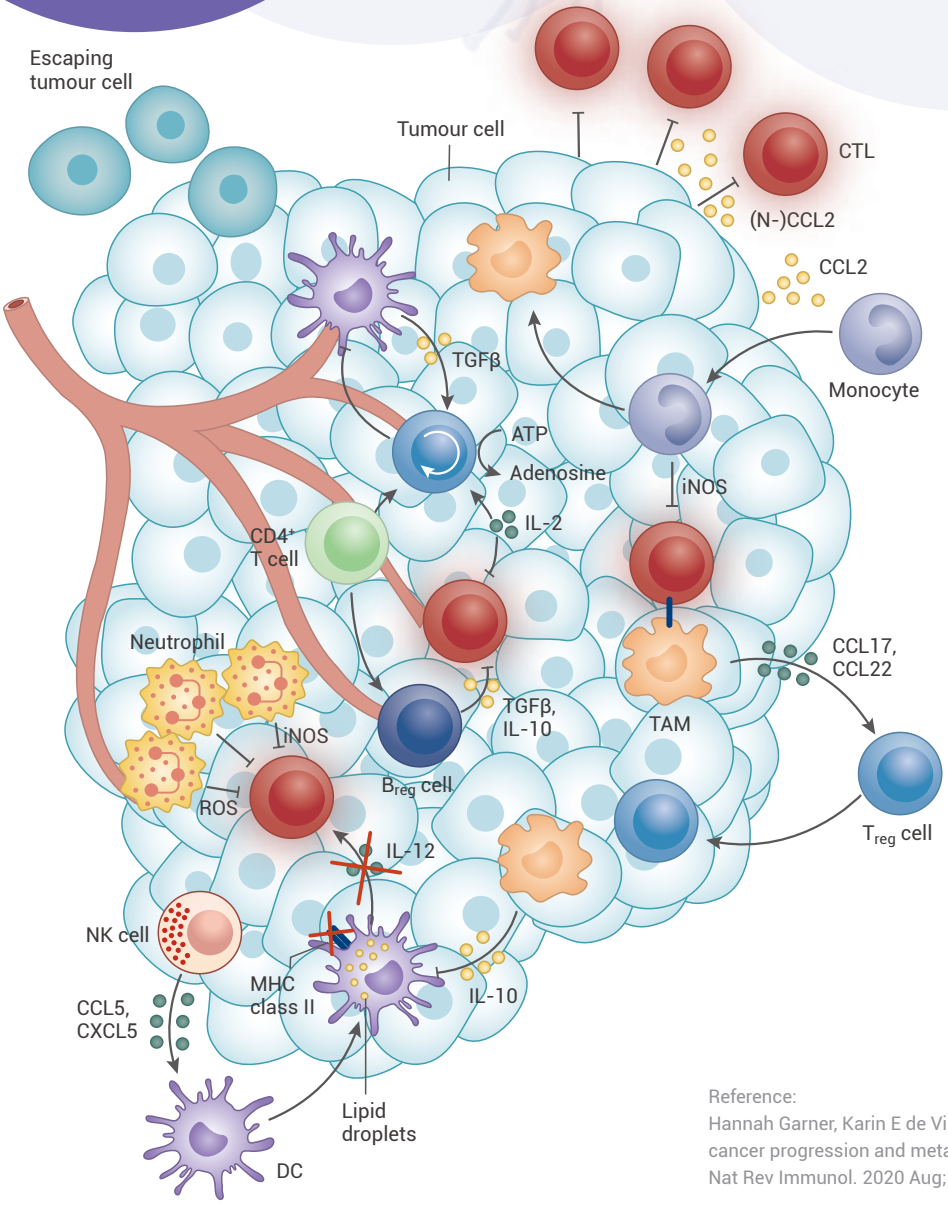
Cancer Research

Product Handbook



Reference:
Hannah Garner, Karin E de Visser. Immune crosstalk in cancer progression and metastatic spread: a complex conversation. Nat Rev Immunol. 2020 Aug;20(8):483-497.

Cancer

Introduction

With the increase of morbidity and mortality worldwide, cancer has become the leading cause of death and a global public health problem. According to statistics of the WHO Report on Cancer 2020, there were an estimated 18.1 million new cases and 9.6 million deaths from cancer.

In 2018, lung cancer was the most frequently diagnosed (11.6% in all cases) cancer and the leading cause of death (18.4% of all deaths) from cancer. Breast cancer, colon cancer, prostate cancer, stomach cancer and liver cancer are cancers with top incidences and mortalities. Figure 2 shows the top 10 types of cancers in the world, of which lung cancer and breast cancer are mentioned in this handbook.

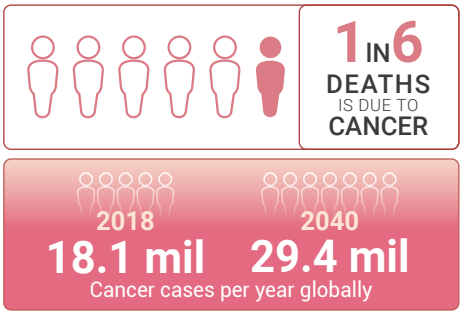


Figure 1. Global cancer burdens [1]

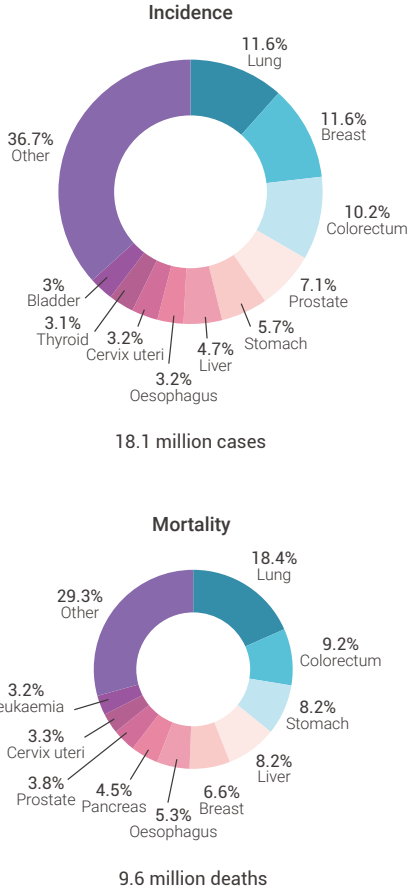


Figure 2. The leading cancer types in 2018 [1]

The traditional hallmarks of cancer include sustaining proliferative signaling, evading growth suppressors, resisting cell death, enabling replicative immortality, inducing angiogenesis, and activating invasion and metastasis. Nowadays, reprogramming of energy metabolism and evading immune destruction is included. In this handbook, some hot topics in cancer such as cancer metabolism, cancer immunotherapy, and cancer stem cells are involved and related small molecule chemicals are listed partially.

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Cancer Metabolism

Cancer has different metabolic pattern from most normal tissues. Metabolic targets of aberrant metabolism cancer cells present new therapeutic perspectives, and a great process has been made.

Warburg effect or aerobic glycolysis indicates that cancer cells consume tremendous amounts of glucose and metabolize them into lactate despite the presence of oxygen. Lactate and pyruvate generated in aerobic glycolysis can guarantee the sufficient biomass for lipids, nucleotides and amino acids of proliferating cancer cells. What's more, a high level of lactate offers an acidic immunosuppressive environment for cancer cells. Enzymes and signaling pathways involved in glycolysis such as glutaminase, PI3K/AKT/mTOR signaling pathway, isocitrate dehydrogenase (IDH) are promising targets for cancer therapy.

Except for carbohydrate metabolism, the metabolisms of other molecules are abnormal in cancer cells. Because of the absence of the ability to synthesizesome non-essential amino acids, an extra supply of them is necessary for the survival of cancer cells. Targeting these molecules such as phosphoglycerate dehydrogenase (PHGDH) in amino acid dependence is potential cancer therapy. The majority of cancer cells can synthesize lipid de novo, which ensures a continuous supply of raw materials to build a cell membrane. Acetyl-coA carboxylase (ACC) and fatty acid synthase (FASN) are key enzymes in lipid synthesis and might be effective targets of cancer therapy. Nucleotide metabolism is also hyperactive in many cancer cells. In fact, the clinical success of antimetabolite chemotherapies for treating cancer benefits from the increased demand of nucleotides for nucleotide biosynthesis and DNA replication.

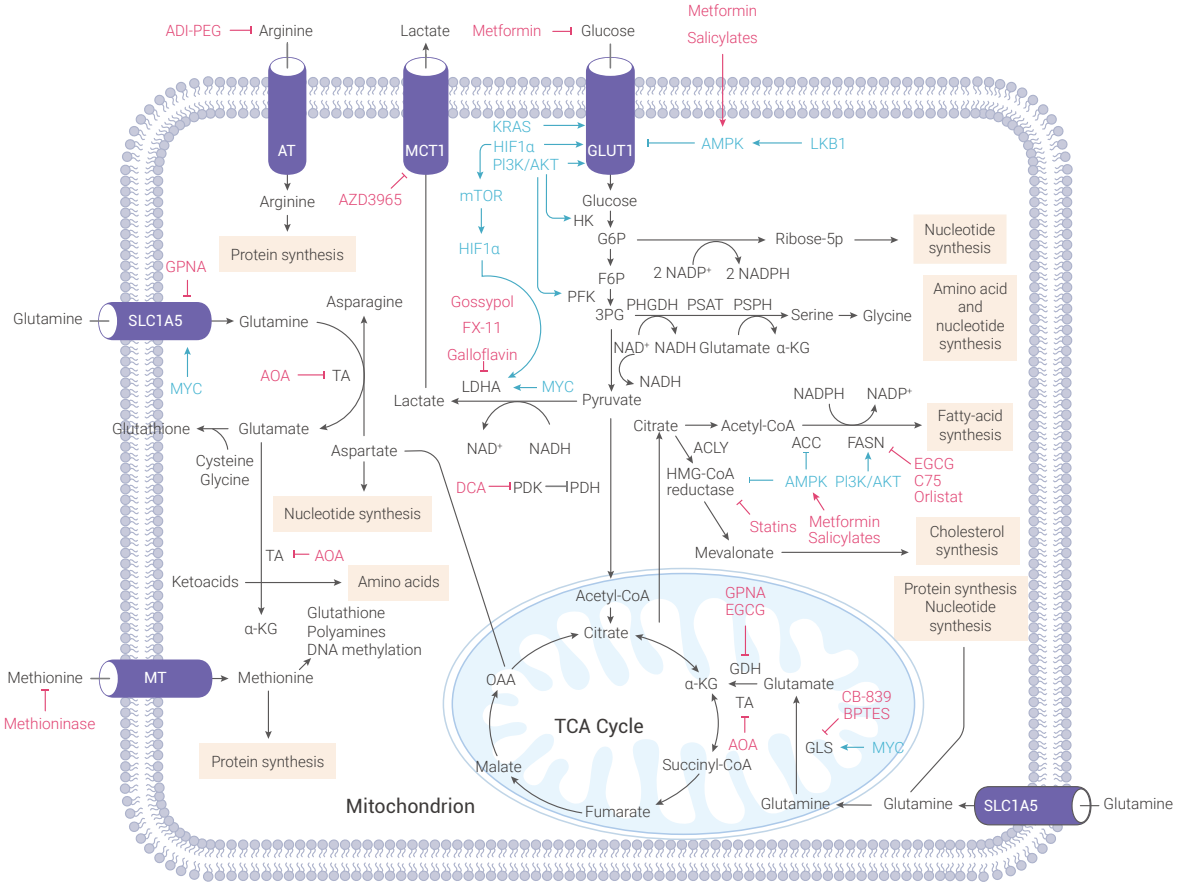


Figure 3. Main metabolic pathways deregulated in cancers and corresponding targeting drugs [2]

Compounds

Cat. No.	Product Name	Description
HY-17394	Cisplatin	An antineoplastic chemotherapy agent by cross-linking with DNA.
HY-B0627	Metformin	The most commonly prescribed drug for type 2 diabetes and inhibits growth of certain types of cancer.
HY-12248	Telaglenastat (CB-839)	A first-in-class glutaminase 1 (GLS1) inhibitor.
HY-100017	BAY-876	An orally active GLUT1 inhibitor and inhibits glycolytic metabolism and ovarian cancer growth.
HY-18767	Ivosidenib (AG-120)	An orally active IDH1 inhibitor for AML therapy.
HY-19992	3-Bromopyruvic acid	A hexokinase II inhibitor, and an effective antitumor agent on the hepatoma cells.
HY-135841	CM10	An ALDH1A inhibitor. Regulates metabolism and has anti-cancer activity.
HY-A0210	Cerulenin	The best known natural inhibitor of fatty acid synthase (FASN).
HY-120394	TVB-3166	A FASN inhibitor and inhibits in-vivo xenograft tumor growth.
HY-Y0445A	Sodium dichloroacetate	A metabolic regulator that increases ROS generation and promotes cancer cell apoptosis.
HY-119502	Camalexin	Induces ROS production. Anticancer activities.
HY-122312	BAY-8002	An orally active inhibitor of MCT1. Anti-tumor activity.

Compound Screening Libraries

Cat. No. : HY-L012

Metabolism/Protease Compound Library

A unique collection of Metabolism/Protease-related small molecules that acts as a useful tool for drug discovery of metabolism-related diseases.

Cat. No. : HY-L058

Glycolysis Compound Library

A unique collection of glycolysis-related small molecules for glucose metabolism research and anti-cancer drug discovery.

Cat. No. : HY-L064

Glutamine Metabolism Compound Library

A unique collection of glutamine metabolism -related small molecules targeting the mainly proteins and enzymes involved in glutamine metabolism pathway.

Cancer Immunotherapy

The immune system can distinguish between self and non-self. Tumor cells have the ability to avoid recognition and elimination by the immune system, allowing malignant cancers progression. Over the last few decades, tremendous progress has been made in the understanding of immune escape mechanisms of tumors, which in turn offers strategies of immunotherapy for cancer. Immunotherapy for cancer has attracted increasing interest and gained impressive clinical benefits. A variety of proteins/receptors are now being investigated as potential targets for cancer immunotherapy, in which immune checkpoints and tumor microenvironment (TME) is outstanding.

Immune checkpoints are regulators of the immune system including stimulatory checkpoint molecules and inhibitory checkpoint molecules. Stimulatory checkpoint molecules such as CD28, TCR are necessary for activation of T cells, and inhibitory checkpoint molecules such as CTLA4 and PD1 can cause immunosuppression. Targeting the inhibitory checkpoints using antibodies or small molecules is a promising therapy for cancer treatment.

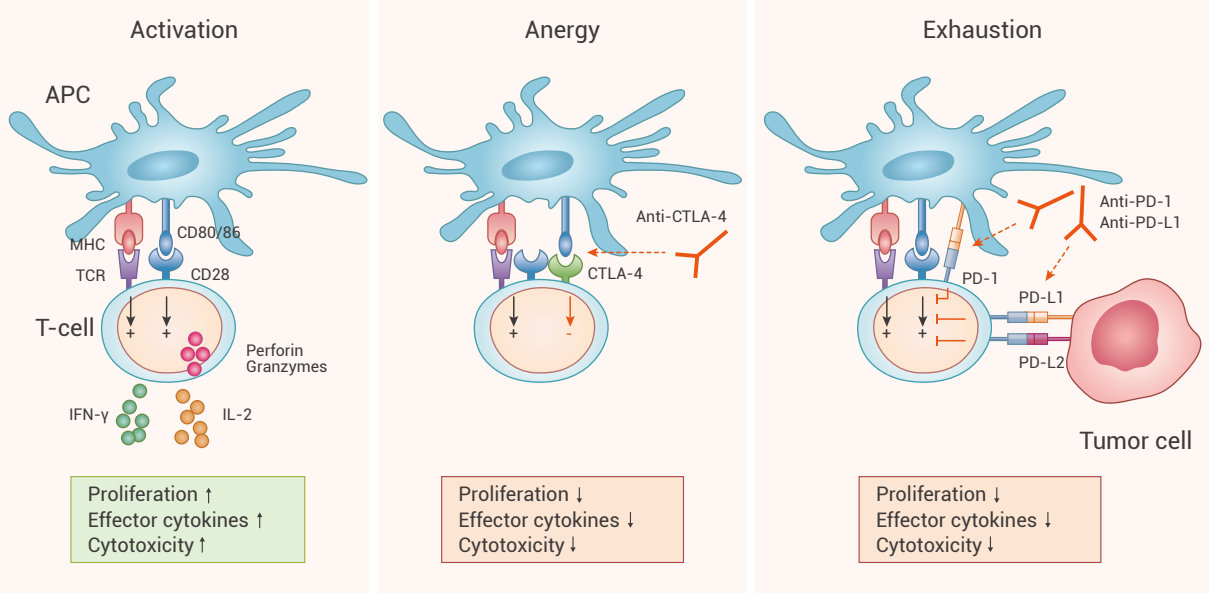


Figure 4. Schematic of mechanism of CTLA-4 and PD-1 [3]

The tumor microenvironment (TME) is the cellular environment in which the tumor exists, including surrounding blood vessels, the extracellular matrix (ECM), other non-malignant cells, and also signaling molecules. Researchers have recognized that normal cells in TME are stromal cells, cancer associated fibroblasts (CAFs), immune cells, endothelial cells, etc. Except for toxic T cells, B cells, there are also regulatory T cells, tumor-associated macrophages (TAMs), myeloid derived suppressor cells (MDSCs). The growth factors secreted by stromal cells and CAF can not only promote growth and survival of malignant cells but also function as negative regulators of the immune response.

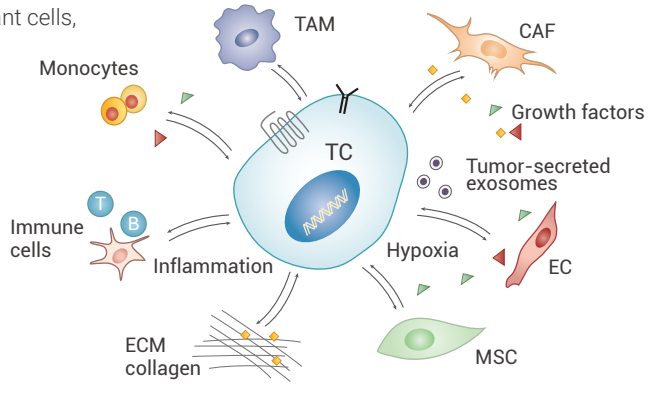


Figure 5. Diagram of tumor microenvironment [4]

All the components synergize an immunosuppressive TME. Molecules associated with TME such as cytokine receptors, metabolic enzymes are critical targets in cancer immunotherapy. These targets include RORγt, Chemokine receptor (CXCR), Sting, IDO, TLR, etc.

		Compounds
Cat. No.	Product Name	Description
HY-P9901	Ipilimumab	A humanized monoclonal IgG1κ antibody against CTLA-4. Approved by FDA for the treatment of melanoma.
HY-P9904	Atezolizumab	A selective humanized monoclonal IgG1 antibody against PD-L1. Approved by FDA for the treatment of NSCLC.
HY-19745	BMS-202	A potent PD-1/PD-L1 complex inhibitor with antitumor activity.
HY-102011	BMS-1166	A potent PD-1/PD-L1 checkpoint inhibitor that antagonizes the inhibitory effect of PD-1/PD-L1 immune checkpoint on T cell activation.
HY-104037	Cintirorgon (LYC-55716)	A first-in-class RORγ agonist that decreases tumor growth, and improves survival.
HY-126321	RORγt agonist 1	A potent, orally bioavailable RORγt agonist for cancer immunotherapy.
HY-19855	AZD-5069	A potent CXCR2 antagonist, used for cancer study.
HY-119339	SX-682	An orally bioavailable inhibitor of CXCR1 and CXCR2. SX-682 enhances T cell activation and antitumor immunity.
HY-13226	Galunisertib (LY2157299)	A selective TGF-βRI kinase inhibitor with anti-tumor activity.
HY-19928	Vactosertib (EW-7197)	An orally active ALK5 inhibitor and has potently antimetastatic activity and anticancer effect.
HY-10964	Vadimezan (DMXAA)	The tumor vascular disrupting agent. An agonist of STING.
HY-130115	IACS-8803	A potent STING agonist with robust systemic antitumor efficacy.
HY-130116	IACS-8779	A potent STING agonist with robust systemic antitumor efficacy.
HY-16724	Indoximod (NLG-8189)	An IDO pathway inhibitor with anti-tumor activity.
HY-15689	Epacadostat	A selective IDO1 inhibitor for treating advanced cancer.
HY-18770B	Navoximod (GDC-0919)	A potent IDO pathway inhibitor for solid tumors.
HY-101560	Linrodostat (BMS-986205)	A selective IDO1 inhibitor with potent pharmacodynamic activity in advanced cancers.

Compounds

Cat. No.	Product Name	Description
HY-101111	PF-06840003	A selective IDO-1 inhibitor and inhibits tumor growth.
HY-101978	CPI-444	A selective A2AR antagonist, which induces antitumor responses.
HY-101980	AZD4635	An orally active A2AR antagonist with anti-tumor activity.
HY-101979	Numidargistat (CB-1158)	An orally active inhibitor of arginase. Immuno-oncology agent.
HY-N0242	Fraxinellone	A PD-L1 inhibitor for cancer treatment. Inhibits HIF-1α protein synthesis without affecting HIF-1α protein degradation.
HY-P9902	Pembrolizumab	A humanized antibody that inhibits the programmed cell death 1 (PD-1) receptor, used in cancer immunotherapy.
HY-125506	NP-G2-044	A potent, orally active fascin inhibitor that blocks tumor metastasis and increases antitumor immune response.
HY-136927	MSA-2	A potent and orally available non-nucleotide STING agonist that induces tumor regression with durable antitumor immunity.
HY-136198	SRX3207	An orally active and first-in-class dual Syk/PI3K inhibitor that relieves tumor immunosuppression.
HY-126147	J22352	A PROTAC-like and highly selective HDAC6 inhibitor that induces anticancer effects.
HY-129601	MYCi975	An orally active MYC inhibitor that disrupts MYC/MAX interaction. Anti-tumor efficacy.
HY-119367	ODM-203	A potent FGFR and VEGFR inhibitor that exhibits strong anti-tumor activity and induces anti-tumor immunity.
HY-16961	Sitravatinib	An orally bioavailable receptor tyrosine kinase (RTK) inhibitor that shows potent single-agent antitumor efficacy.
HY-123291	SM-276001	A selective TLR7 agonist induces antitumor immune responses.

Compound Screening Library

Cat. No. : HY-L031

Small Molecule Immuno-Oncology Compound Library

A unique collection of bioactive tumor immunology compounds that target some important checkpoints such as PD1/PD-L1, CXCR, STING, IDO, TLR, etc.

Cancer Stem Cells

Heterogeneity is one of the most relevant features of cancer cells within different tumor types and is responsible for treatment failure and recurrence. Cancer stem cells (CSCs) are a population of cells with stem cell properties that are considered to be the root cause of tumor heterogeneity because of their ability to self-renewal and differentiate into all of cancer cell types.

CSCs are generally considered insensitivity to traditional chemotherapy drugs. Conventional therapy kills non-CSCs but leaves CSCs untouched, leading to tumor relapse. Killing the CSCs may result in eventual tumor eradication (Figure 6). For CSC therapy, promoting CSCs differentiation into non-CSCs, inhibiting self-renewal property of CSCs, CSCs microenvironment is promising targets. What's more, molecules or pathways directly related to drug resistance of CSCs such as multidrug resistance proteins and anti-apoptotic pathways have also been explored.

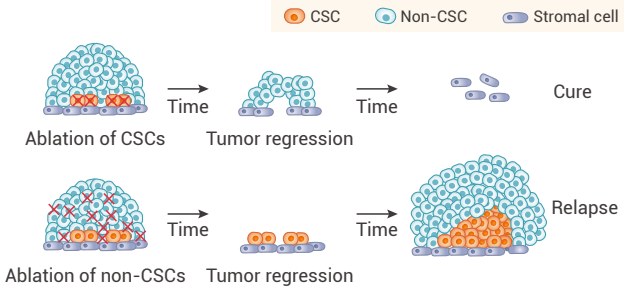


Figure 6. Diagram of cancer recurrence based on CSCs [5]

To the date, the most exploited signaling pathways associated with the self-renewal of CSCs are the Hedgehog signalling, Notch signalling and Wnt/β-catenin signaling pathways. As for promoting the differentiation of CSCs, bone morphogenic protein (BMP) signaling and P13K/mTOR signaling are among the most studied signaling pathways.

Compounds

Cat. No.	Chemical Name	Description
HY-10440	Vismodegib (GDC-0449)	A hedgehog pathway inhibitor for advanced basal cell carcinoma.
HY-135145	CB-103	A first-in-class notch pathway inhibitor with anti-tumor activity.
HY-15531	Venetoclax (ABT-199)	A potent Bcl-2 inhibitor and effectively targets CSC population.
HY-100431	IMR-1	A Notch inhibitor and inhibits tumor growth.
HY-15721	FH535	An inhibitor of Wnt/β-catenin and PPAR. Anti-tumor activities.
HY-12020	TW-37	A potent Bcl-2 inhibitor that acts as an anticolorectal cancer agent.
HY-16591	Birinapant (TL32711)	A potent XIAP and cIAP1 antagonist. Induction of tumor cell death.
HY-12289	Defactinib (VS-6063)	A FAK inhibitor with antiangiogenic and antineoplastic activities.
HY-13917	PND-1186	A potent FAK inhibitor with anti-cancer activity.

Compound Screening Library

Cat. No. : HY-L017	
Stem Cell Signaling Compound Library	A unique collection of compounds for stem cell signaling research.

Cancer Pathways

Notch, Hippo, Hedgehog, Wnt, and TGF-β/BMP/FGF signaling pathways are highly conserved cell signaling systems present in almost all multicellular organisms. They are networks that act coordinately to play crucial roles in cell proliferation, apoptosis, differentiation, and finally in organ development. Disruptions of genes in one pathway can have effects in related pathways and may result in cancer.

Compounds

Cat. No.	Product Name	Description
HY-101275	EMT inhibitor-1	An inhibitor of of Hippo, TGF-β, and Wnt signaling pathways with antitumor activities.
HY-B0146	Verteporfin (CL 318952)	Inhibits the Hippo pathway and blocks the translocation of YAP/TAZ into the nucleus, thus inhibiting cancer cell growth and survival.
HY-12419	BMS-983970	An oral pan-Notch inhibitor for the treatment of multiplecancers.
HY-N0133	Tangeretin (NSC53909)	A Notch-1 inhibitor with anti-tumor activity.
HY-16582A	Sonidegib (Erismodegib)	A potent Smo antagonist for locally advanced basal cell carcinoma.
HY-10440	Vismodegib (GDC-0449)	A Hedgehog pathway inhibitor. Approved by FDA for the treatment of locally advanced basal cell carcinoma.
HY-18959	CWP232228	A highly potent Wnt/β-catenin signaling inhibitor that inhibits the growth of breast and liver cancer stem cells (CSCs).
HY-103021	LY3200882	A highly selective TGF-β R1 (ALK5) inhibitor that acts as an immune modulatory agent.
HY-101568	Roblitinib (FGF-401)	A highly selective FGFR4 inhibitor with anti-tumor activity.
HY-N2112	Glaucocalyxin A	Inhibits GLI1 via regulating PI3K/Akt pathway. Antitumor effect.

Compound Screening Libraries

Cat. No. : HY-L018

TGF-beta/Smad Compound Library

A unique collection of TGF-beta/Smad signaling pathway compounds used for TGF-beta/Smad-related drug screening and disease research.

Cat. No. : HY-L020

Wnt/Hedgehog/Notch Compound Library

A unique collection of Wnt/Hedgehog/Notch signaling pathway-related small molecules used for stem cell research and anti-cancer drug screening.

NSCLC

As mentioned above, lung cancer is the most frequently diagnosed cancer and the leading cause of cancer death in the world. Lung cancers are a group of distinct diseases with cellular and genetic heterogeneity. Approximately 85% of lung cancer is non-small cell lung cancer (NSCLC), of which lung adenocarcinoma (LUAD; ~50%) and lung squamous cell carcinoma (LUSC; ~40%) are the most common subtypes. A series of genetic mutations are identified in lung cancer, such as KRAS, BRAF, EGFR, TP53, AKT, etc. Most of them are promising therapeutic targets for lung cancer.

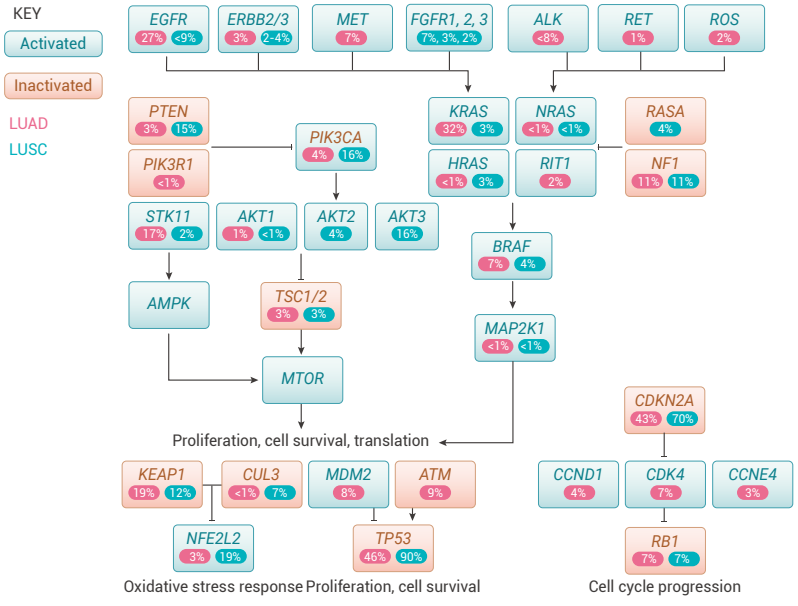


Figure 7. Targetable mutations involved in LUAD and LUSC [6]

Compounds

Cat. No.	Chemical Name	Description
HY-50896	Erlotinib	A directly acting EGFR inhibitor for the treatment of NSCLC.
HY-50895	Gefitinib	A selective and orally active EGFR inhibitor for treatment of NSCLC.
HY-15772	Osimertinib	An irreversible and mutant selective EGFR inhibitor for the treatment of $EGFR^{T790M}$ NSCLC.
HY-50878	Crizotinib	An orally bioavailable ALK and c-Met inhibitor for the treatment of advanced ALK^+/$ROS1^+$ NSCLC.
HY-13011	Alectinib	An orally available ALK inhibitor for the treatment of advanced ALK^+ NSCLC.
HY-114277	Sotorasib (AMG-510)	The first $KRAS^{G12C}$ inhibitor in clinical development and leads to the regression of $KRAS^{G12C}$ tumors.
HY-130149	Adagrasib (MRTX849)	A mutation-selective covalent inhibitor of $KRAS^{G12C}$ with potential antineoplastic activity.

Compounds

Cat. No.	Chemical Name	Description
HY-130260	KRAS inhibitor-4	A potent KRAS inhibitor and developed as an anticancer agent.
HY-19980A	PRIMA-1	A mutant p53 reactivator with anti-tumor activity.
HY-122054	BPK-29	A specific ligand that disrupts NR0B1-protein interactions. Impairs anchorage-independent growth of KEAP1-mutant cancer cells.
HY-136173	TNO155	An orally active allosteric wild-type SHP2 inhibitor for the study of RTK-dependent malignancies, especially advanced solid tumors.
HY-112823	Almonertinib	An orally available, irreversible EGFR inhibitor with high selectivity for EGFR-sensitizing and T790M resistance mutations.
HY-136174	RBN-2397	A potent, accross species and orally active NAD^+ competitive PARP7 inhibitor for the study of advanced or metastatic solid tumors.
HY-123986	CTPI-2	A third-generation mitochondrial citrate carrier SLC25A1 inhibitor.
HY-116000	Glumetinib	A highly selective, orally bioavailable, ATP-competitive c-Met inhibitor. Antitumor activity.
HY-131067	EMI56	The derivative of EMI1, displays greater potency toward mutant EGFR than EMI1. Inhibits EGFR triple mutants.
HY-12215	Lorlatinib	A selective, orally active, brain-penetrant and ATP-competitive ROS1/ALK inhibitor. Anticancer activity.
HY-131066	EMI48	The derivative of EMI1, displays greater potency toward mutant EGFR than EMI1. Inhibits EGFR triple mutants.
HY-19637	SW044248	A non-canonical topoisomerase I inhibitor, and selectively toxic for certain NSCLC cell lines.
HY-112299	TAS6417	An EGFR inhibitor for NSCLC.
HY-19730	Olmutinib	An orally available and irreversible third EGFR inhibitor that binds to a cysteine residue near the kinase domain. Used for NSCLC.

Compound Screening Library

Cat. No. : HY-L025

Anti-Cancer Compound Library

A unique collection of bioactive anti-cancer compounds that target kinases, cell cycle key components, tumorigenesis related signaling pathways, etc.

Breast Cancer

Just like lung cancer, breast cancer is also a very heterogeneous disease with distinct clinical implications and different molecular subtypes. The major subtypes of breast cancer are approximated by the joint expression of three tumor markers: estrogen receptor (ER), progesterone receptor (PR), and human epidermal growth factor receptor 2 (HER2). As shown in figure 8, the four main subtypes of breast cancer are described as Luminal A ($HR^+/HER2^-$), Luminal B ($HR^+/HER2^+$), HER2 enriched ($HR^-/HER2^+$), and basal-like (triple-negative, $HR^-/HER2^-$), in which HR represents hormone receptor including ER and PR [7]. Except for the different expression pattern of HR and HER2, there are also many genetic mutations (such as *PTEN, BRCA1, BRCA2, TP53, XRCC2, XRCC3, ATM, CHEK2, PALB2, RAD51, XPD*) have been implicated in breast cancer. Targeting the related proteins has a role in breast cancer research and management.

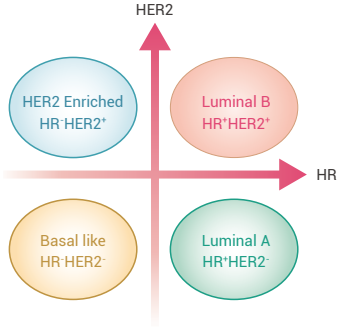


Figure 8. Subtypes of breast cancer according to HR/HER2 [7]

Compounds

Cat. No.	Product Name	Description
HY-P9907	**Trastuzumab**	A humanized monoclonal antibody for the treatment of metastatic $HER2^+$ breast cancer.
HY-P9912	**Pertuzumab**	A humanized monoclonal antibody for the treatment of metastatic $HER2^+$ breast cancer.
HY-13757A	**Tamoxifen**	An orally active ER modulator. Approved by FDA for the treatment of breast cancer.
HY-119377	**UPGL00004**	An allosteric GAC inhibitor that inhibits the proliferation of highly aggressive triple-negative breast cancer cell lines.
HY-10162	**Olaparib**	An orally active PARP inhibitor. Approved by FDA for the treatment of $BRCA^+/HER2^-$ breast cancer.
HY-124691	**D-I03**	A selective RAD52 inhibitor that suppresses growth of BRCA1- and BRCA2-deficient cells.
HY-50767	**Palbociclib**	A selective CDK4 and CDK6 inhibitor that has the potential for $ER^+/HER2^-$ breast cancer research.
HY-N0656A	**(+)-Usnic acid**	Inhibits mTORC1/2. Anti-cancer activity.
HY-15842	**SF1670**	A specific PTEN inhibitor for the research of breast cancer.
HY-10029	**Nutlin-3a**	Inhibits MDM2-p53 interactions and has the potential for the study of ovarian carcinomas.

PROTACs

PROTACs or PROteolysis TArgeting Chimeric Molecules are structurally comprised of two recognition motifs linked by a linker. One recognition motif is a small molecule ligand for the protein of interest, the other recognizes a specific E3 ligase. A PROTAC can recruit an E3 ligase to a target protein and result in the degradation of the protein through ubiquitination proteasome pathway. PROTACs are an emerging and promising approach for the development of targeted therapy drugs and many PROTACs with high potency have been frequently reported.

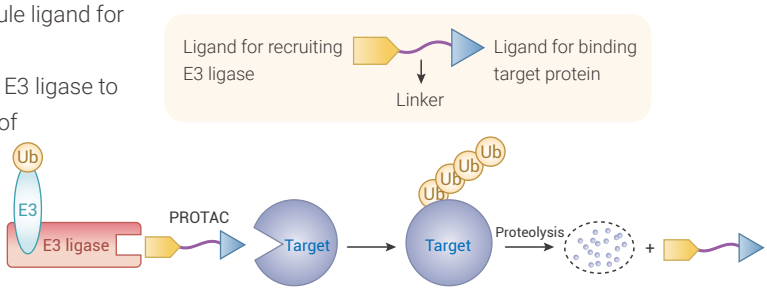


Figure 9. Structure of PROTACs [8]

All of the components of PROTACs (target protein ligand, the E3 ligase ligand and the linker) attract a lot of attention in the research filed of PROATCs. Nowadays, epigenetic tools (BET bromodomain), nuclear receptors (such as RAR, ER, and AR) and kinases (CDK, RIPK2) are reported successfully targeted by PROTACs. The E3 ligases highlighted in PROTACs are VHL, Cereblon, IAP, MDM2, etc. The following list are some PROTACs related products of MedChemExpress (MCE).

300+
PROTAC
Heterobifunctional nanomolecules that structurally comprised of two functional motifs linked by a linker.

5,000+
PROTAC Linker
Connects two functional motifs of a PROTAC, a target protein binder and an E3 ligase recruiter.

80+
Ligand for E3 Ligase
Binds to a pocket or surface of the E3 ligase, to provide a suitable starting point for the design of the bifunctional PROTACs.

20+
Target Protein Ligand-Linker Conjugate
Incorporates a ligand for the target protein and a linker. When binding to an E3 ligand, the conjugate will be a PROTAC to induce ubiquitylation and subsequent degradation of target proteins.

500+
E3 Ligase Ligand-Linker Conjugate
One part of PROTACs, incorporates a ligand for the E3 ubiquitin ligase and a linker.

5+
PROTAC-linker Conjugate for PAC
Comprises an antibody conjugated via a linker to a PROTAC.

60+
Ligand for Target Protein for PROTAC
Leads to attachment of a PROATC to target proteins for ubiquitylation and subsequent degradation.

20+
SNIPER
Induces IAP-mediated ubiquitylation and proteasomal degradation of target proteins.

PROTACs

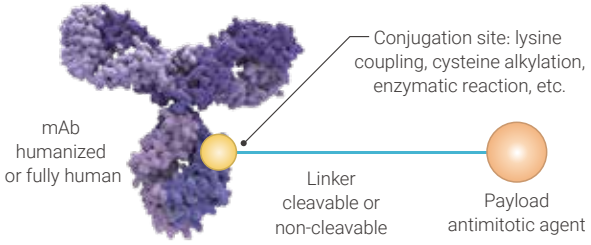
Cat. No.	Product Name	Description
HY-112588	dBET6	A highly potent PROTAC BET degrader with antitumor activity.
HY-128756	SIAIS178	A highly potent PROTAC BCR-ABL degrader with antitumor activity.
HY-111556	BSJ-03-123	A potent PROTAC CDK6 degrader with antitumor activity.
HY-100972	ARV-771	A potent PROTAC BET degrader with antitumor activity.
HY-101838	dBET1	A potent PROTAC BRD4 degrader with antitumor activity.
HY-133557	XZ739	A CRBN-dependent PROTAC BCL-XL degrader. Antitumor activity.
HY-128600	ERD-308	A potent PROTAC ER degrader with antitumor activity.
HY-130492	ARCC-4	A potent PROTAC AR degrader with antitumor activity.
HY-107425	MZ 1	A PROTAC BRD4 degrader that rapidly induces reversible, long-lasting, and selective removal of BRD4 over BRD2 and BRD3.
HY-16954	ARV-825	A BRD4 degrader based on PROTAC technology. ARV-825 binds to BD1 and BD2 of BRD4.
HY-122826	ZXH-3-26	A selective PROTAC BRD4 degrader.
HY-122562	MT-802	A potent PROTAC BTK degrader that has potential to treat C481S mutant chronic lymphocytic leukemia (CLL).
HY-114312	MD-224	A first-in-class and highly potent PROTAC human murine double minute 2 (MDM2) degrader.
HY-130604	DT2216	A potent and selective PROTAC BCL-XL degrader that inhibits various BCL-XL-dependent leukemia and cancer cells.
HY-114305	A1874	A nutlin-based and BRD4-degrading PROTAC. Effective in inhibiting many cancer cell lines proliferation.
HY-101519	BETd-260	A potent PROTAC BET degrader that potently suppresses cell viability and robustly induces apoptosis in HCC cells.
HY-123937	THAL-SNS-032	A selective PROTAC CDK9 degrader with antitumor activity.
HY-129602	SD-36	A potent and efficacious PROTAC STAT3 degrader that exerts robust anti-tumor activity.

Antibody-Drug Conjugates (ADCs)

Antibody-Drug Conjugates (ADCs) are potent biopharmaceutical cancer-targeted drugs comprised of a humanized or human monoclonal antibody conjugated with cytotoxic drugs (payloads) via a chemical linker.

ADCs exhibit high selectivity and toxicity to the tumor, and become one of the fastest-growing classes of therapeutics. To date, several ADCs (Mylotarg, Adcetris, Kadcyla, Besponsa, Lumoxiti, Polivy) have been approved for tumor treatment and hundreds of ADCs are currently in clinical trials.

Except for specific antigen and antibody, linkers and payloads are also very important factors for the efficacy of ADCs. The following products including cytotoxins, linkers and drug-linker conjugates for ADCs are available in MedChemExpress (MCE).



Key factors

-High potency
-Low immunogenicity
-Low cytotoxicity to off-target cells
- High cancer cell specificity
- Long circulating life

Figure 9. Structure of ADCs [9]

HY-19609 Calicheamicin	HY-B0015 Paclitaxel	HY-15162 Monomethyl auristatin E	HY-19792 Mertansine	Antineoplastic agents. Used as cytotoxins of antibody-drug conjugates (ADCs).
HY-42973 DBCO-NHS ester	HY-100216 SPDP	HY-12362 Val-cit-PAB-OH		Cleavable ADC linkers. Cleavable linkers utilize inherent properties of tumor cells for selective release of the cytotoxin from the ADCs.
HY-D0975 Sulfo-SMCC sodium	HY-42149 NH2-PEG2-C2-Boc	HY-79369 Succinic anhydride		Nonclaevable ADC linkers for the synthesis of ADCs. After entering cells, the antibody is partially degraded, and the nonclaevable linker and toxic molecule are still connected together.
HY-15575 VcMMAE	HY-101070 SMCC-DM1	HY-126681 SC-VC-PAB-MMAE		Drug-Linker Conjugates consist of active toxic molecules and linkers for the synthesis of ADCs.
HY-L023 Toxins for Antibody-Drug Conjugate Research Library				A unique collection of ADC cytotoxins for the synthesis of ADCs.

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