CANCER DRUG PHARMACOLOGY TABLE

Cytotoxic Chemotherapy

Drugs are classified according to the BC Cancer Drug Manual Monographs, unless otherwise specified (see asterisks). Subclassifications are in brackets where applicable.

Alkylating Agents have reactive groups (usually alkyl) that attach to DNA or RNA, leading to interruption in synthesis of DNA, RNA, or proteins.

- bendamustine (nitrogen mustard)
- busulfan (alkyl sulfonate)
- carboplatin (platinum)
- carmustine (nitrosurea)
- chlorambucil (nitrogen mustard)
- cisplatin (platinum)
- cyclophosphamide (nitrogen mustard)
- dacarbazine (triazine)
- estramustine (nitrogen mustard with 17-beta-estradiol)
- hydroxyurea
- ifosfamide (nitrogen mustard)
- lomustine (nitrosurea)
- mechlorethamine (nitrogen mustard)
- melphalan (nitrogen mustard)
- oxaliplatin (platinum)
- procarbazine (triazine)
- streptozocin (nitrosurea)
- temozolomide (triazine)
- thiotepa (aziridine)
- treosulfan

Antimetabolites are structural analogues of naturally occurring molecules required for DNA and RNA synthesis. When substituted for the natural body substances, they disrupt DNA and RNA synthesis.

- azacitidine (pyrimidine analogue)
- capecitabine (pyrimidine analogue)
- cladribine (adenosine analogue)
- cytarabine (pyrimidine analogue)
- fludarabine (purine analogue)
- fluorouracil (pyrimidine analogue)
- gemcitabine (pyrimidine analogue)
- mercaptopurine (purine analogue)
- methotrexate (folate analogue)
- nelarabine (purine nucleoside analogue)
- pralatrexate (folate analogue)
- pemetrexed (folate analogue)
-
- pentostatin (purine analogue)
- raltitrexed (folate analogue)
- thioguanine (purine analogue)
- trifluridine-tipiracil (pyrimidine analogue/thymidine phosphorylase inhibitor)

Antimicrotubule Agents (Mitotic Inhibitors) inhibit cell mitosis by interfering with microtubule formation or function.

- cabazitaxel (taxane)
- docetaxel (taxane)
- eribulin
- ixabepilone
- paclitaxel (regular and nanoparticle, albumin-bound) (taxane)
- vinblastine (vinca alkaloid)
- vincristine (vinca alkaloid)
- vinorelbine (vinca alkaloid)

Miscellaneous Antineoplastics - Refer to BC Cancer monographs for pharmacology.

- arsenic trioxide
- asparaginase
- belzutifan
- bleomycin
- belinostat
- calaspargase pegol
- crisantaspase recombinant
- dactinomycin
- decitabine - cedazuridine
- iniparib
- lurbinectedin
- mitomycin
- mitotane
- pegaspargase
- porfimer
- romidepsin
- vorinostat

Topoisomerase Inhibitors (I and II) cause DNA strand breaks by disrupting the function of topoisomerase enzymes, which are responsible for regulating the 3-D structure of DNA.

Topoisomerase I

- irinotecan
- topotecan

Topoisomerase II

- amsacrine
- anthracyclines
  - daunorubicin
  - doxorubicin (regular and pegylated liposomal)
  - epirubicin
  - idarubicin
- etoposide
- mitoxantrone
- teniposide

Hormonal Therapies

Antiestrogens oppose the effects of estrogen.

- tamoxifen – partial estrogen antagonist (antagonist on breast tissue, agonist on endometrium, bone and lipids)
- fulvestrant – full estrogen antagonist (no agonist activity)

Antiandrogens opposes the effects of androgens.

Aromatase Inhibitors (AIs) prevent the final step in the conversion of androgens to estrogens in peripheral tissues.

- anastrozole
- exemestane
- letrozole

Luteinizing Hormone Releasing Hormone (LHRH) Agonists (also known as gonadotropin releasing hormone analogues) initially stimulate the release of

• apalutamide • bicalutamide • darolutamide • enzalutamide – more affinity for androgen receptors and Plus inhibits more steps in the androgen inhibition than other agents in this class • flutamide • nilutamide **Androgen Biosynthesis Inhibitors** • abiraterone - selectively inhibits the enzyme (CYP17) that converts pregnenolone and progesterone into testosterone precursors. **Androgens** • testosterone **-** The exact mode of action for androgen therapy in breast cancer is unclear. **Corticosteroids** are thought to act via apoptosis induction. • dexamethasone • prednisone **Somatostatin Analogues** inhibit exocrine and endocrine secretion of hormones, which is useful for hormone-secreting tumours (e.g., neuroendocrine). Additional mechanisms include modulation of biliary/GI motility and apoptosis inductions. • lanreotide • octreotide	luteinizing hormone, which leads to an increase in sex hormones (testosterone, estradiol). Chronic use leads to down-regulation of the LHRH receptors, leading to decreased testosterone in men and estrogen in women. • buserelin • goserelin • leuprolide **Luteinizing Hormone Releasing Hormone (LHRH) Antagonist** (also known as gonadotropin releasing hormone antagonist) reduce the release of luteinizing hormone, follicle-stimulating hormone, and consequently testosterone by the testes. • degarelix **Progestins** suppress the release of luteinizing hormone from the pituitary gland and subsequently decrease estrogen levels. Additional mechanisms include binding to progesterone, glucocorticoid, and androgen receptors, resulting in decreased number of estrogen receptors and decreased estrogen and progesterone levels peripherally in target tissues. • medroxyprogesterone • megestrol **Prolactin Lowering Agents** are dopamine antagonists that decrease hormone production and the size of prolactin-dependent pituitary adenomas by inhibiting the release and synthesis of prolactin from the anterior pituitary. • bromocriptine • cabergoline • quinagolide

Thyrotropin Stimulating Hormone Agonist is a recombinant thyrotropin used for serum thyroglobulin testing in thyroid cancer.

- thyrotropin alpha

Immunotherapies

Cytokines are proteins that are involved in the cell signaling that leads to immune responses at sites of inflammation, infection, and trauma. They induce various cellular responses, such as suppression of cell proliferation and augmentation of the cytotoxicity of lymphocytes.

- aldesleukin
- interferon
- peginterferon

Vaccine Therapy

- bacillus calmette-guerin (BCG)
  - a live, attenuated bacteria (Mycobacterium bovis) that exerts a variety of antitumour actions, including induction of a local granulomatous reaction, activation of histiocytes, and other direct and indirect stimulation of immune responses. The result is a local inflammatory response that destroys tumour cells.

Immunomodulatory Drugs (IMIDs) have multiple mechanisms of action, including inhibition of proliferation of certain hematopoietic tumour cells, enhancing numbers and activity of T, NK, and NK T cells, and inhibition of angiogenesis.

- lenalidomide
- pomalidomide
- thalidomide

Differentiating Agents are vitamin A derivatives. Their proposed mechanism of action is to overcome impaired cellular differentiation.

- acitretin
- alitretinoin
- bexarotene
- tretinoin

Other Immunotherapies

- imiquimod – TLR7 agonist
- Monoclonal antibodies could also be considered immunotherapies, particularly those that inhibit CTLA-4, PD-1 or PD-L1 (Checkpoint Inhibitors), or IL-6. They are covered on the pages that follow.

Targeted Therapies

Targeted therapies target receptors, ligands, or intracellular molecules involved in the signal transduction of cancer cells. The following table is a listing of targeted therapies with the target(s) listed in brackets. See the following page for more information on targets. Note that the relative affinity to particular targets is not always clear for each agent, and may differ when used in different indications. Some of the available literature refer to drugs by their target, such as EGFR-inhibitors or multikinase inhibitors for oral drugs with multiple targets (e.g., pazopanib,sorafenib, sunitinib).

abemaciclib (CDK 4/6)
acalabrutinib (BTK)
afatinib (EGFR, HER2, HER4)
AGS-16C3F (MMAF) (antibody conjugated with cytotoxic)
alectinib (ALK)
alemtuzumab (CD52)
amivantamab (low-fucose IgG1 bispecific antibody binding to EGFR and MET)
asciminib (ABL-TKI myristoyl pocket (STAMP)
atezolizumab (PD-L1)
avelumab (PD-L1)
axitinib (VEGFR 1, 2, & 3)
bevacizumab (VEGF)
belantamab mafodotin (IgG1) (antibody conjugated with cytotoxic)
binimetinib (MEK)
blinatumomab (CD3 & CD19)
bortezomib (26S proteosome)
bosutinib (BCR-ABL1 TKI)
brentuximab vedotin (CD30) (antibody conjugated with cytotoxic)
brigatininb (ALK) (ROS1) (EGFR) (IGF) (FLT3)
cabozantinib (MET, VEGF, FLT3)
capivasertib (AKT1, AKT2 and AKT3)
carfilzomib (26S proteosome)
carotuximab (aka TRC105) (CD105)
cemiplimab (PD-1)
elranatamab (IgG2 bispecific antibody against BCMA and CD3)
encorafenib (BRAF V600E, V600D,V600K, wt-BRAF, CRAF, JNK1, 2, 3, LIMK1, 2, MEK4 and STK3)
enfortumab vedotin (Nectin-4)(antibody drug conjugate)
entrectinib (NTRK gene fusion)
epcoritamab (bispecific antibody) (CD3 on T cells & CD20 on B-cell malignant cells)
erlotinib (EGFR)
everolimus (MTOR)
fedratinib (JAK2, FLT3)
gefitinib (EGFR)
gemtuzumab ozogamicin (CD33) (antibody conjugated with cytotoxic)
gilteritinib (FLT-3)
glofitamab (bispecific antibody binding to CD20 on B cells and CD3 on Tcells)
ibrutinib (BTK)
idelalisib (PI3Kδ)
imatinib (BCR-ABL1, PDGF, c-KIT)
inotuzumab ozogamicin (CD22) (antibody conjugated with cytotoxic)
ipilumumab (CTLA-4)
Isatuximab (IgG1 antibody) (CD38)
lapatinib (EGFR, HER2)
larotrectinib (tropomyosin receptor kinase,)
pembrolizumab (PD-1)
pertuzumab (HER2)
polatuzumab vedotin (CD79b) (antibody conjugated with cytotoxic)
ponatinib (BCR-ABL1, TKI)
pralsetinib (RET tyrosine kinase inhibitor)
ramucirumab (VEGFR2 and VEGF A, C, and D)
regorafenib (VEGFR-1, -2, & -3, TIE2, KIT, RET RAF-1, BRAF, BRAV600E, PDGFR, FGFR)
relatlimab (LAG3)
retifanlimab (PD-1)
ribociclib (CDK 4/6)
ripretinib (kinases KIT and PDGFRA)
rituximab (CD20)
ruxolitinib (JAK 1 & 2)
sacituzumab govitecan (IgG1.k antibody conjugated with cytotoxic)
selpercatinib (RET fusion positive)
selinexor (SINE)
siltuximab (IL-6)
sonidegib (Hh)
sorafinib (c-Raf,b-Raf, V600E,b-Raf,KIT,FLT-3, VEGFR -2, -3 & -beta)
sunitinib (VEGFR 1, 2, & 3, PDGFR α & β), KIT, FLT-3, CSF-1R, RET)
tarlatamab (bispecific antibody - binds to DLL3 on tumour cells and CD3 on T cells)
tebentafusp (fusion protein on CD3)

ceritinib (ALK) cetuximab (EGFR) cobimetinib (MEK) crizotinib (ALK, HGFR, C-Met, ROS1) dabrafenib (BRAF) dacomitinib (EGFR) daratumumab (CD38) dasatinib (BCR-ABL1, LYN, HCK, c-kit, EPH, PDGFβ) datopotamab deruxtecan (TROP2) (antibody conjugated with cytotoxic) denosumab (RANKL) dinutuximab (GD2) dostarlimab (IgG4) durvalumab (PD-L1)	NTRK gene fusion) lenvatinib (VEGFR, FGFR, PDGFRα, KIT, RET) lorlatinib (ALK & ROS1) midostaurin (FLT-3, KIT, PDGFR) mirvetuximab soravtansine (ADC: anti-FRα antibody with cleavable link to maytansoid antitubulin DM4) mogamulizumab (CCR4) nilotinib (BCR-ABL1, c-KIT, PDGFR) niraparib (PARP-1, PARP-2) nivolumab (PD-1) obinutuzumab (CD20) ofatumumab (CD20) olaparib (PARP-1, PARP-2, PARP-3) olaratumab (PDGFR α) osimertinib (EGFR) panitumumab (EGFR) palbociclib (CDK 4/6) pazopanib (VEGFR 1, 2, 3, c-KIT, PDGFR-α,-β, c-KIT,FGFR-1 and -3, IL-2, and c-Fms)	teclistamab (bispecific antibody targets CD3 on T and B cell maturation antigen (BCMA) temsirolimus (MTOR) tislelizumab (IgG4) (PD-1) tocilizumab (IL-6) trametinib (MEK 1 & 2) trastuzumab (HER2) trastuzumab emtansine (HER2) (antibody conjugated with cytotoxic) trastuzumab deruxtecan (HER2) (antibody conjugated with cytotoxic) tucatinib (HER2) vandetanib (VEGFR-2, EGFR, RET) vemurafenib (BRAF) venetoclax (BCL-2) vismodegib (Hh) zanubrutinib (BTK)

The last letters in the drug names in the table provide information about the classification of the drug:

- mab = monoclonal antibody
- zomib = proteasome inhibitor
- nib = kinase inhibitors
- olimus = MTOR inhibitor

Target Listing

AKT	AKT1, AKT2 and AKT3 (gene isoforms)	AKT is a serine/threonine protein kinase which regulates cell proliferation, metabolism, and angiogenesis in normal and malignant cells
ALK	Anaplastic Lymphoma Kinase	Translocations in this gene lead to oncogenic fusion proteins that play a role in many cancers, including non-small-cell lung cancer.
BCL-2	B-cell chronic lymphoma 2	BCL-2 is an anti-apoptotic protein
BCMA	B Cell maturation antigen	

BCR-ABL1	Breakpoint Cluster Region – Abelson Leukemia	The BCR-ABL1 gene is an acquired genetic change formed when two separate genes fuse together to become one abnormal gene. The abnormal BCR-ABL1 gene is formed when pieces of chromosome 9 (from ABL1 gene) and chromosome 22 (BCR gene) break off and trade places. The broken piece of chromosome 9 fuses to part of the BCR gene on chromosome 22. The abnormal fusion gene (BCR-ABL1) formed on chromosome 22 is called the Philadelphia (Ph) chromosome
BRAF	BRAF	The BRAF proto-oncogene belongs to RAF family of serine/threonine protein kinases. The BRAF protein plays a role in regulating the MAP kinase/ERK signaling pathway which regulate cell proliferation, apoptosis and cell differentiation. .
BTK	Bruton's Tyrosine Kinase	BTK gene encodes the protein for making Bruton's Tyrosine Kinase which is essential for development and maturation of B cells. BTK is an integral part of the B-cell antigen receptor (BCR) pathway, which is associated with the pathogenesis of several B-cell malignancies
CD	Cluster Of Differentiation Antigens	CDs are a group of antigens present on the surface of all cells in different combinations, which makes them useful for classifying cells. • CD3 is found on T cells • CD19 is found on B cells • CD20 is found on B cells • CD30 is expressed on Hodgkin's Lymphoma and anaplastic large cell lymphoma cells (16) • CD38 is highly expressed on myeloma cells, but is expressed at low levels on normal lymphoid and myeloid cells • CD52 is found on the surface of B and T lymphocytes, most monocytes, macrophages and NK cells, and certain granulocytes • CD105 (endoglin) expression is required for vascular endothelial cell proliferation. Targeting CD105 is a novel approach to inhibiting angiogenesis in cancer cells.
CDK 4/6	Cyclin-dependent kinases	CDK4/6 form complexes with cyclin D to promote phosphorylation of retinoblastoma (Rb) protein, which allows cell cycle progression.
C-Kit	Stem Cell Factor Receptor	C-Kit is involved in oncogenesis. 95% of GIST cells have c-Kit mutations.
CCR (1-10)	CC Chemokine receptors	There are 10 CC chemokine receptor subtypes. CCR4 is expressed on the surface of some T-cell malignancies, as well as on regulatory T-cells (Treg) and T helper cells (Th2)

CRAF	Cellular (RAF) Rapidly Accelerated Fibrosarcoma	Plays a critical role in mediating the cellular effects of growth factor signals.
CTLA-4	Cytotoxic T Lymphocyte-Associated Antigen 4	CTLA-4 acts as an immune response checkpoint by switching off T-cells. Agents that target CTLA-4 are referred to as Checkpoint Inhibitors.
DLL3	Delta like ligand	DLL3 is an inhibitory ligand highly expressed in SCLC and other neuroendocrine tumors. It is minimally expressed in normal tissues.
EGFR	Epidermal Growth Factor Receptor (also referred to as HER1)	EGFR is involved in cancer cell proliferation, blocking apoptosis, mobilizing cells to promote metastasis, and angiogeneisis.
EPH	Ephrin Receptor	EPH may be involved in the development of resistance to imatinib.
FGFR	Fibroblast Growth Factor Receptor	FGFR contributes to the maintenance of the tumour microenvironment.
FLT3	FMS-Like Tyrosine Kinase 3	Like other tyrosine kinase inhibitors, FLT3 competes for the ATP binding site in the active domain of the kinase, which inhibits the ability of the protein to be phosphorylated, and subsequently decreases activity of the protein.
FRα	Folate Receptor alpha	FRα is a membrane bound protein with a high affinity for binding and transporting folate into cells
GD2	Disialoganglioside	GD2 is a surface antigen found on the surface of neuroblastoma cells.
HER	Human Epidermal Growth Factor (also known as EGFR)	HER2 is overexpressed in about 20% of breast cancers, which leads to increased cell proliferation, cancer spread, and apoptosis inhibition.
Hh	Hedgehog Pathway	This pathway is normally dormant in adult tissues, but basal cell carcinomas have gene mutations that activate the Hh pathway, which promotes tumour survival and cancer spread.
IGF (1 &2)	Insulin-like growth factor	Produce insulin-like actions in some tissues, they are far less vvv than insulin in decreasing blood glucose concentrations. Their fundamental action is to stimulate growth.
Ig	Immunoglobulins	Are proteins produced by B lymphocytes and plasma cells. Each subclass possesses a unique manner of antigen binding and immune complex formation. Immoglobulins are also known as antibodies.
IgG (1,2,3,4)	Immunoglobulin G	IgG (1-4) provides the majority of antibody-based immunity and is the main type of antibody in blood and extracellular fluid.
JAK	Janus Associated kinase	JAK mediates the signaling pathway of cytokines and growth factors for hematopoiesis and immune function.
JNK	c-Jun N-terminal kinase	(Also known as stress-activated protein kinase, SAPK) is one of the 3 major members of the mitogen-activated protein kinase (MAPK) superfamily.

LAG3	Lymphocyte activation gene 3	LAG3 (also named CD233) suppresses T cell activation. LAG3 is expressed on several T cells ($CD4^+$, $CD8^+$ and Tregs) and negatively regulates T cell function.
LYN	Lck/Yes novel tyrosine kinase	LYN is involved in BCR-ABL signaling
LIMK	Lim Kinase	Are actin-binding kinases that phosphorylate members of the ADF/cofilin family of actin binding and filament severing proteins.
MEK	Mitogen-Activated Extracellular Signal-Regulated Kinase	MEK1 and MEK2 are involved in cell growth, differentiation, inflammation, and apoptosis.
MTOR	Mammalian Target of Rapamycin	Inhibit cell proliferation and angiogenesis.
Nectin-4	Adhesion protein on surface of cells	(Nectin Cell Adhesion Molecule 4) is a Protein Coding gene. In the antibody–drug conjugate (ADC), human anti-nectin-4 antibody is linked to the cytotoxic microtubule-disrupting agent monomethyl auristatin E
NTRK	Neurotrophic tyrosine kinase inhibitor	NTRK (NTRK1, NTRK2, NTRK3) gene fusions are oncogenic drivers of various tumour types. TRK proteins are receptor kinases that help regulate cell signaling and function in healthy tissues.
PARP	poly (ADP-ribose) polymerase	Binding to PARP inhibits single stranded DNA base excision repair and creates PARP-DNA complexes that lead to double-stranded DNA breaks, ultimately causing cell death.
PD-1 PD-L1	Programmed Death Receptor 1 and Programmed Death Receptor Ligand 1	PD-1 receptors are located on T-cells. When ligands bind to PD-1 receptors, they switch off T-cells, which fight cancer. Agents that target PD-1 are referred to Checkpoint Inhibitors.
PDGF	Platelet-Derived Growth Factor	PDGF contributes to maintenance of tumour microenvironments.
PI3Kδ	Phosphoinositide 3-kinase	Active in the signalling pathways of B-cell malignancies
Proteosome	Proteosome	Proteosomes degrade cellular proteins targeted for destruction. Inhibition of the proteasome results in cell cycle arrest and apoptosis.
RAF	Rapidly Accelerated Fibrosarcoma (see BRAF and CRAF)	RAF kinases are a family of three serine/threonine-specific protein kinases from the TKL (Tyrosine-kinase-like) group of kinases. RAF kinases participate in the RAS-RAF-MEK-ERK signal transduction cascade, also referred to as the mitogen-activated protein kinase (MAPK) cascade.
RANKL	Receptor Activator Of Nuclear Factor Kappa-B Ligand	RANKL activates osteoclasts, leading to bone resorption.
RET	Rearranged during transfection	RET is a proto-oncogene which encodes a receptor tyrosine kinase for members of the glial cell line-derived neurotrophic factor (GDNF) family of extracellular signalling molecules.
ROS1	c-ros oncogene1	ROS is a proto-oncogene which may mutate to become an oncogene.
SINE	Selective inhibitor of Nuclear Export	SINE target XPO1, arrest tumor suppressor proteins in the nucleus and induce apoptosis

STAMP	Specifically Targeting the ABL Myristoyl Pocket	Wild-type ABL has a myristoylated N-terminus which binds to an allosteric site, but the ABL fusion protein does not have the myristoylated domain. In wild-type ABL, when myristoylated N-terminus binds to the allosteric site, the kinase has reduced activity. The mutant fusion protein does not have the myristoylated N-terminus domain and is not subject to this form of regulation, and the fusion protein is constitutively active.
STK3	serine/threonine-protein kinase 3	The STK3 gene encodes a serine threonine protein kinase. STK receptors play a role in the regulation of cell proliferation, programmed cell death (apoptosis), cell differentiation, and embryonic development.
TLR7	Toll-like receptor 7	TLR7 stimulates innate and cell-mediated immunity to induce antitumour effects, including the increased production of inflammatory cytokines, such as tumour necrosis factor-α (TNFα), interferon-α, and interleukin-12.
TROP2	Anti-trophoblast cell surface antigen	TROP2 is a transmembrane glycoprotein involved in many cell signaling pathways often upregulated in cancer cells
VEGF VEGFR	Vascular Endothelial Growth Factor and Vascular Endothelial Growth Factor Receptor	VEGF and VEGFR are involved in the development of a tumour blood supply (angiogenesis).

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