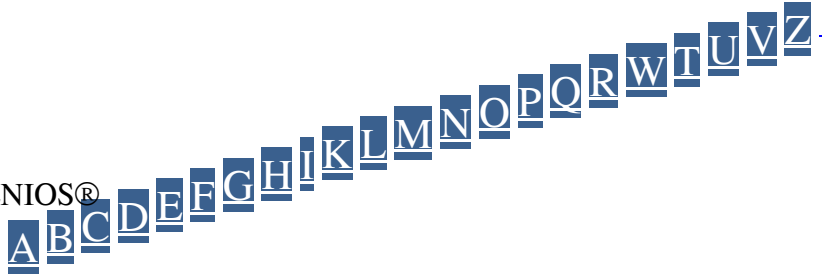


Glossary A-Z

Wirkstoffe A

Abemaciclib – VERZENIOS®

[Navigation überspringen](#)



According to the NCI website Abemaciclib is an orally available cyclin-dependent kinase (CDK) inhibitor that targets the CDK4 (cyclin D1) and CDK6 (cyclin D3) cell cycle pathway, with potential antineoplastic activity. Abemaciclib specifically inhibits CDK4 and 6, thereby inhibiting retinoblastoma (Rb) protein phosphorylation in early G1. Inhibition of Rb phosphorylation prevents CDK-mediated G1-S phase transition, thereby arresting the cell cycle in the G1 phase, suppressing DNA synthesis and inhibiting cancer cell growth. Overexpression of the serine/threonine kinases CDK4/6, as seen in certain types of cancer, causes cell cycle deregulation.

Indikationen gemäss Compendium.ch®

Verzenios ist angezeigt zur Behandlung von postmenopausalen Frauen mit Hormonrezeptor (HR)-positivem, humanem epidermalen Wachstumsfaktor-Rezeptor-2 (HER2)-negativem lokal fortgeschrittenem oder metastasiertem Brustkrebs:

- in Kombination mit einem Aromatasehemmer als initiale endokrine Therapie;
- in Kombination mit Fulvestrant bei Frauen, nach vorheriger endokriner Therapie;
- als Monotherapie nach Progression der Erkrankung nach endokriner Therapie und einem oder zwei Chemotherapie-Regimen bei metastasierter Erkrankung, wenn eine Chemotherapie nicht geeignet ist.

Link zur Fachinformation von Compendium.ch®:

Medikamenteninformation: [Für den Arzt](#)

More Information in English:

[Link to Drug Information Portal, a service of the U.S. National Library of Medicine, National Institutes of Health](#)

[Link to MedlinePlus, a service of the U.S. National Library of Medicine, National Institutes of Health](#)

[Link to National Cancer Institute](#)

[Link zu Wiki](#)

[Link zu PharmaWiki](#)

[Link to Physicians Desk Reference \(PDR\)](#)

[Link to European Medicines Agency \(EMA\)](#)

[Info for Patients presented by Scott Hamilton from Chemocare.com](#)

Abirateron - ZYTIGA®

Zytiga® ist ein oral verfügbares Azetat-Salz des antiandrogenen Steroids Abirateron. Es hemmt die enzymatische Aktivität der Steroid-17alpha-Monooxygenase (17alpha-hydroxylase/C17,20 Lyase Komplex), einem Mitglied der Zytochrom p450 Familie. Dieses katalysiert die 17alpha-Hydroxylierung der Steroid-Zwischenprodukte, die an der Testosteron Synthese beteiligt sind. Die Verabreichung des Wirkstoffes kann die Bildung von Testosteron sowohl in den Hoden als auch in den Nebennieren bis auf ein Kastrationsniveau unterdrücken.

Indikationen/Anwendungsmöglichkeiten gemäss Arzneimittelkompendium:

- Zur Behandlung in Kombination mit LHRH Agonisten und Prednison oder Prednisolon bei Patienten mit fortgeschrittenem metastasierenden Prostatakarzinom bei

Progredienz nach Behandlung mit Docetaxel.

- Zur Behandlung in Kombination mit LHRH Agonisten und Prednison oder Prednisolon bei asymptomatischen oder leicht symptomatischen Patienten mit metastasierendem, kastrationsresistentem Prostatakarzinom (mCRPC) ohne viszerale Metastasen und ohne Lebermetastasen, nach Versagen der Androgendeprivationstherapie, wenn eine Chemotherapie klinisch nicht indiziert ist.
- Zur Behandlung in Kombination mit Prednison oder Prednisolon und Androgendeprivationstherapie (ADT) bei Patienten mit neu diagnostiziertem Hochrisiko-metastasiertem hormonsensitivem Prostatakarzinom (mHSPC) (siehe «Klinische Wirksamkeit»).

Link zur Fachinformation des Arzneimittel-Kompodium der Schweiz:

Medikamenteninformation:

[Für den Arzt](#)

[Patienteninformation](#)

Information des Médicaments:

[Info prof.](#)

[Info patient](#)

Informazione sul medicamento:

[info per il paziente](#)

More Information in English:

[Link to National Cancer Institute](#)

[Link zu Wiki](#)

[Link zu PharmaWiki](#)

[Link to Physicians Desk Reference \(PDR\)](#)

[Link to European Medicines Agency \(EMA\)](#)

Acalabrutinib – CALQUENCE® (USA)

According to the NCI website Acalabrutinib orally available inhibitor of Bruton's tyrosine kinase (BTK) with potential antineoplastic activity. Upon administration, acalabrutinib inhibits the activity of BTK and prevents the activation of the B-cell antigen receptor (BCR) signaling pathway. This prevents both B-cell activation and BTK-mediated activation of downstream survival pathways. This leads to an inhibition of the growth of malignant B cells that overexpress BTK. BTK, a member of the src-related BTK/Tec family of cytoplasmic tyrosine kinases, is overexpressed in B-cell malignancies; it plays an important role in B lymphocyte development, activation, signaling, proliferation and survival.

Indicated according to Chemocare for:

- The treatment of adult patients with mantle cell lymphoma (MCL) who have received at least one prior therapy.
- The treatment of adult patients with chronic lymphocytic leukemia (CLL) or small lymphocytic lymphoma (SLL).

More Information in English:

[Link to Drug Information Portal, a service of the U.S. National Library of Medicine, National](#)

Institutes of Health

[Link to MedlinePlus, a service of the U.S. National Library of Medicine, National Institutes of Health](#)

[Link to National Cancer Institute](#)

[Link zu Wiki](#)

[Link zu PharmaWiki](#)

[Link to Physicians Desk Reference \(PDR\)](#)

[Link to European Medicines Agency \(EMA\)](#)

[Info for Patients presented by Scott Hamilton from Chemocare.com](#)

ACTR087

According to the NCI website **anti-ACTR/4-1BB/CD3zeta-viral vector-transduced autologous T lymphocytes ACTR087** are autologous T lymphocytes that are genetically modified and transfected with a viral vector expressing the ACTR gene, a proprietary gene encoding for an antibody-coupled T cell receptor (ATCR), with potential antineoplastic activity.

[Link to National Cancer Institute](#)

ADP-A2M4 (MAGE-A4 C1032 T cells)

According to the NCI website, autologous genetically modified MAGE-A4 C1032 T cells are Autologous human T-lymphocytes transduced with a retroviral vector encoding a T-cell receptor (TCR) specific for the human melanoma antigen A4 (MAGE-A4), with potential immunostimulatory and antineoplastic activities. Upon leukapheresis, isolation, transduction, expansion ex vivo, and reintroduction into the patient, the autologous genetically-modified MAGE-A4 C1032 T cells bind to tumor cells

expressing MAGE-A4. This may result in both inhibition of growth and increased cell death of MAGE-A4-expressing tumor cells. The tumor-associated antigen MAGE-A4, a member of the MAGE-A family of cancer testis antigens, is overexpressed by a variety of cancer cell types. Check for [active clinical trials](#) using this agent. ([NCI Thesaurus](#))

More Information in English:

[AdisInsight](#)

[Link to National Cancer Institute](#)

Adriamycin

Vgl. DOXORUBICIN

Afatinib - GIOTRIF®

Afatinib ist ein oral verfügbarer dualer Tyrosin-Kinase-Rezeptor (RTK) Inhibitor mit einer potentiell antineoplastischen Aktivität. Afatinib bindet sich irreversibel an die humanen epidermalen Wachstumsfaktoren 1 und 2 (EGFR-1; HER2) und hemmt sie auch irreversibel. Dies kann zu einer Hemmung des Tumorwachstums sowie der Gefässneubildung (Angiogenese) führen. EGFR/HER2 sind RTKs, die zur EGFR Superfamilie gehören. Beide spielen eine bedeutende Rolle bei der Proliferation der Tumorzellen und bei der Gefässversorgung des Tumors und werden in vielen Krebszellen überexprimiert.

Indikationen/Anwendungsmöglichkeiten gemäss Compendium®:

- Giotrif ist als Monotherapie für Patienten mit nicht-kleinzelligem Lungenkarzinom (NSCLC, Stadium IIIb/IV) mit aktivierenden Mutationen des EGFR (Exon 19 Deletionen, Exon 18 G719X Substitutionen, Exon 20 S768I Substitutionen sowie Exon 21 L858R Substitutionen und L861Q Substitutionen) indiziert, die nicht mit EGFR-TKIs vorbehandelt sind.
- Giotrif ist zur Behandlung von Patienten mit einem lokal fortgeschrittenen oder metastasierten Plattenepithelkarzinom der Lunge indiziert, deren Karzinom während oder nach einer platinhaltigen Chemotherapie fortgeschritten ist und die für eine Immuntherapie nicht geeignet sind.

[Merkblätter für Patientinnen und Patienten](#)

Link zur Fachinformation des Compendium®:

Medikamenteninformation: [Für den Arzt](#) [Patienteninformation](#)

Information des Médicaments: [Info prof.](#) [Info patient](#)

Informazione sul medicamento: [info per il paziente](#)

More Information in English

[Link to Drug Information Portal, a service of the U.S. National Library of Medicine, National Institutes of Health](#)

[Link to National Cancer Institute](#)

[Link zu Wiki](#)

[Link zu PharmaWiki](#)

[Link to Physicians Desk Reference \(PDR\)](#)

[Link to European Medicines Agency \(EMA\)](#)

[Info for Patients presented by Scott Hamilton from Chemocare.com](#)

Receptor tyrosine kinases (RTK)s are very important signaling pathway, which not only include growth factor receptors such as [EGFR\(HER\)](#), [VEGFR](#), [PDGFR](#), [FGFR](#), [IGF-1R](#), Mast/stem cell growth factor receptor ([c-Met](#)) and [HER2](#), but also other gene products which are expressed by the oncogenes such as SRC, Bcr, c-Met and Abl as well. [Read more at selleckbio about Receptor Tyrosine Kinase Signaling Pathway](#)

[Tyrosin Kinase Inhibitor](#)

Afuresertib

According to the NCI website Afuresertib is an orally bioavailable inhibitor of the serine/threonine protein kinase Akt ([protein kinase B](#)) with potential antineoplastic activity. Afuresertib binds to and inhibits the activity of Akt, which may result in inhibition of the [PI3K/Akt signaling pathway](#) and tumor cell proliferation and the induction of tumor cell apoptosis. Activation of the PI3K/Akt signaling pathway is frequently associated with tumorigenesis and dysregulated PI3K/Akt signaling may contribute to tumor resistance to a variety of antineoplastic agents. Check for active clinical trials or closed clinical trials using this agent.

[Link to Drug Information Portal, a service of the U.S. National Library of Medicine, National Institutes of Health](#)

[Link to National Cancer Institute](#)

[The IUPHAR/BPS Guide to PHARMACOLOGY](#)

AK-104 (ANTI-PD1/ANTI-CTLA4 BISPECIFIC ANTIBODY AK104)

According to the NCI website, anti-PD-1/CTLA-4 bispecific antibody AK104 is a bispecific antibody directed against the human negative immunoregulatory checkpoint receptors programmed cell death protein 1 (PD-1; PDCD1; CD279) and cytotoxic T-lymphocyte-associated antigen 4 (CTLA4; CTLA-4), with potential immune checkpoint inhibitory and antineoplastic activities. Upon administration, anti-PD-1/CTLA4 bispecific antibody AK104 targets and binds to both PD-1 and CTLA4 expressed on tumor-infiltrating T lymphocytes (TILs), and inhibits the PD-1- and CTLA4-mediated downregulation of T-cell activation and proliferation. This restores immune function and activates a sustained cytotoxic T-lymphocyte (CTL)-mediated immune response against tumor cells. Both PD-1 and CTLA4 are selectively expressed on TILs in the tumor microenvironment (TME) and negatively regulate the activation and effector functions of T cells. They play key roles in the downregulation of the immune system and tumor evasion from host immunity. Dual checkpoint blockade of PD-1 and CTLA4 with AK104 may enhance T-cell activation and proliferation more than the blockade of either immune checkpoint receptor alone. Check for [active clinical trials](#) using this agent. ([NCI Thesaurus](#))

More Information in English:

[Inxight: Drugs \(NIH\)](#)

[AdisInsight](#)

[Link to Drug Information Portal, a service of the U.S. National Library of Medicine, National Institutes of Health](#)

[Link to National Cancer Institute](#)

Aldesleukin/Interleukin-2

Proleukin®; According to the NCI Aldesleukin is a recombinant analog of the endogenous cytokine interleukin-2 (IL-2) with immunoregulatory and antineoplastic activities. Aldesleukin binds to and activates the IL-2 receptor, followed by heterodimerization of the cytoplasmic domains of the IL-2R beta and gamma(c) chains; activation of the tyrosine kinase Jak3; and phosphorylation of tyrosine residues on the IL-2R beta chain, resulting in an activated receptor complex.

Indikationen/Anwendungsmöglichkeiten gemäss Arzneimittelkompendium:

Metastasierendes Nierenkarzinom bei nephrektomierten Patienten.

Die Risikofaktoren, die zu reduziertem Ansprechen und mittlerem Überleben führen, sind:

- reduzierter Allgemeinzustand (ECOG 1 oder mehr bzw. Karnofsky-Index $\leq 80\%$),
 - metastatischer Befall in mehr als einem Organ,
 - ein Intervall von weniger als 24 Monaten zwischen Primärdiagnose und Ansetzen der Proleukintherapie.
- Ansprechraten und mittlere Überlebenszeit werden mit zunehmender Anzahl vorhandener Risikofaktoren geringer. Patienten mit allen drei Risikofaktoren sollten nicht mit Proleukin behandelt werden.

[Link to MedlinePlus, a service of the U.S. National Library of Medicine, National Institutes of Health](#)

[Link to National Cancer Institute](#)

[Link zu Wiki](#)

[Link zu PharmaWiki](#)

[Link to Physicians Desk Reference \(PDR\)](#)

[Link to European Medicines Agency \(EMA\)](#)

[Link zur Fachinformation des Arzneimittel-Kompendium der Schweiz](#)
[Info for Patients presented by Scott Hamilton from Chemocare.com](#)

[Interleukine](#)

Alectinib - ALECENSA®

According to the NCI website Alectinib is an orally available inhibitor of the receptor tyrosine kinase anaplastic lymphoma kinase (ALK) with antineoplastic activity. Upon administration, alectinib binds to and inhibits ALK kinase, ALK fusion proteins as well as the gatekeeper mutation ALKL1196M known as one of the mechanisms of acquired resistance to small-molecule kinase inhibitors. The inhibition leads to disruption of ALK-mediated signaling and eventually inhibits tumor cell growth in ALK-overexpressing tumor cells.

Indikationen/Anwendungsmöglichkeiten gemäss Compendium®

- Alecensa ist für die Behandlung von Patienten mit lokal fortgeschrittenem oder metastasiertem ALK (Anaplastic-lymphoma-kinase)-positivem nicht-kleinzelligem Lungenkarzinom (NSCLC) indiziert.

Medikamenteninformation:

[Für den Arzt gemäss Compenium®](#)

[Patienteninformation gemäss Compendium®](#)

[Link to Drug Information Portal, a service of the U.S. National Library of Medicine, National Institutes of Health](#)

[Link to National Cancer Institute](#)

[Link zu Wiki](#)

[Link zu PharmaWiki](#)

[Link to Physicians Desk Reference \(PDR\)](#)

More info for patients:

[Link to MedlinePlus, a service of the U.S. National Library of Medicine, National Institutes of Health](#)

[Info for Patients presented by Scott Hamilton from Chemocare.com](#)

[ALK-Inhibitors](#)

Alemtuzumab

MabCampath® - According to the NCI website Alemtuzumab is a recombinant DNA-derived humanized monoclonal antibody directed against the cell surface glycoprotein CD52. Alemtuzumab is an IgG1 kappa with human variable framework and constant regions, and complementarity-determining regions derived from a rat monoclonal antibody. This agent selectively binds to CD52, thereby triggering a host immune response that results in lysis of CD52 + cells. CD52 is a glycoprotein expressed on the surface of essentially all normal and malignant B and T cells, a majority of monocytes, macrophages and natural killer (NK) cells, a subpopulation of granulocytes, and tissues of the male reproductive system.

Indikationen/Anwendungsmöglichkeiten gemäss Arzneimittelkompendium:

MabCampath ist als Monotherapie zur Behandlung von Patienten mit chronischer lymphatischer B-Zell Leukämie (B-CLL) indiziert.

Kombinationstherapien mit MabCampath wurden nicht hinreichend untersucht.

Nicht im Compendium in dieser Indikation aufgeführt

[Link to National Cancer Institute](#)

[Link to Wikipedia](#)

[Link zu PharmaWiki](#)

[Link to Physicians Desk Reference \(PDR\)](#)

[Link to European Medicines Agency \(EMA\)](#)

[Link zur Fachinformation des Arzneimittel-Kompendium der Schweiz](#)

[Info for Patients presented by Scott Hamilton from Chemocare.com](#)

[Monoclonal antibodies for tumors](#)

[Monoclonal Antibodies](#)

Alitretinoin

Toctino®; According to the NCI website alitretinoin is an orally- and topically-active naturally-occurring retinoic acid with antineoplastic, chemopreventive, teratogenic, and embryotoxic activities. Alitretinoin binds to and activates nuclear retinoic acid receptors (RAR) and retinoid X receptors (RXR); these activated receptors act as transcription factors, regulating gene expression that results in the inhibition of cell proliferation, induction of cell differentiation, and apoptosis of both normal cells and tumor cells.

Indikationen/Anwendungsmöglichkeiten gemäss Arzneimittelkompendium:

(According to MedlinePlus Alitretinoin is used to treat skin lesions associated with Kaposi's sarcoma. It helps stop the growth of Kaposi's sarcoma cells.

This medication is sometimes prescribed for other uses; ask your doctor or pharmacist for more information.)

Toctino ist indiziert bei Erwachsenen mit therapierefraktärem, schwerem chronischem Handekzem, die eine ausgebaute lokale Behandlung für mindestens 4 Wochen erhalten und nicht darauf angesprochen haben. Die Vorbehandlung schliesst die Vermeidung von Kontakten mit der auslösenden Noxe, Hautschutz und potente topische Kortikosteroide ein.

[Link to MedlinePlus, a service of the U.S. National Library of Medicine, National Institutes of Health](#)

[Link to National Cancer Institute](#)

[Link zu Wiki](#)

[Link zu PharmaWiki](#)

[Link to Physicians Desk Reference \(PDR\)](#)

[Link to European Medicines Agency \(EMA\)](#)

[Link zur Fachinformation des Arzneimittel-Kompendium der Schweiz](#)

[Info for Patients presented by Scott Hamilton from Chemocare.com](#)

[Chemotherapy](#)

ALLO-647

According to the NCI website, ALLO-647 is a monoclonal antibody directed against the cell surface glycoprotein CD52 (CAMPATH-1 antigen; Cambridge pathology 1 antigen), with potential immunodepleting activity. Upon administration, anti-CD52 monoclonal antibody ALLO-647 selectively targets and binds to CD52, thereby triggering a host immune response that results in the lysis of CD52-positive lymphocytes. This leads to immunodepletion and may prevent graft-versus-host disease (GvHD). CD52 is a glycoprotein expressed on the surface of many immune cells, including essentially all B and T lymphocytes. Check for [active clinical trials](#) using this agent. ([NCI Thesaurus](#))

[Link to National Cancer Institute](#)

ALLO-501 - allogeneic CD19-specific universal CAR19-expressing T lymphocytes

According to the NCI website, ALLO-501 are a preparation of allogeneic, frozen, 'off-the-shelf', universal transcription activator-like effector nuclease (TALEN)-engineered, gene-edited T lymphocytes expressing a chimeric antigen receptor (CAR) targeting the tumor-associated antigen (TAA) CD19, and containing a RQR8 transgene, with potential immunostimulating and antineoplastic activities. Using TALEN technology, the T-cell receptor (TCR) alpha chain and CD52 genes are deleted from the CAR19 T cells. Upon infusion, allogeneic universal CD19-specific CAR-modified T cells (UCART19) specifically target and bind to CD19-expressing tumor cells, thereby selectively lysing CD19-expressing tumor cells. CD19 antigen is a B-cell specific cell surface antigen expressed in all B-cell lineage malignancies. Deletion of the CD52 gene makes the modified donor T cells resistant to the anti-CD52 monoclonal antibody alemtuzumab, which is used during lymphodepletion. The knockout of the TCR alpha gene eliminates TCR expression and is intended to abrogate the potential induction of graft-versus-host disease (GvHD) by the donor T cells. The gene-edited allogeneic, frozen UCART19 have reduced production times and provide off-the-shelf CAR-T cells when compared to autologous CAR-T cells, which use the patient's own cells and are produced on an individual basis. The protein expressed by the RQR8 transgene contains epitopes from CD34 and CD20, which allows tracking of the UCART19 cells with a clinically-approved anti-CD34 antibody. Additionally if the UCART19 cells cause unacceptable side effects, the CD20 portion of the protein permits selective depletion of the UCART19 cells when the anti-CD20 monoclonal antibody rituximab is administered. Check for [active clinical trials](#) using this agent.

([NCI Thesaurus](#))

[Link to National Cancer Institute](#)

Alpelisib - PIQRAY®

According to the NCI website Alpelisib is an **orally** bioavailable phosphatidylinositol 3-kinase (PI3K) inhibitor with potential antineoplastic activity. Alpelisib specifically inhibits PIK3 in the PI3K/AKT kinase (or protein kinase B) signaling pathway, thereby inhibiting the activation of the PI3K signaling pathway. This may result in inhibition of tumor cell growth and survival in susceptible tumor cell populations. Activation of the PI3K signaling pathway is frequently associated with tumorigenesis. Dysregulated PI3K signaling may contribute to tumor resistance to a variety of antineoplastic agents.

Indikation gemäss Compendium®

- Piqray wird in Kombination mit Fulvestrant angewendet für die Behandlung von postmenopausalen Frauen mit Hormon-Rezeptor (HR)-positivem, humanen epidermalen Wachstumsfaktor-Rezeptor-2 (HER2)-negativem fortgeschrittenem Brustkrebs mit einer PIK3CA-Mutation nach Fortschreiten der Erkrankung, wenn die Patienten zuvor eine endokrine Therapie einschliesslich eines Aromatase Inhibitors erhalten haben.

Arzneimittelinformation gemäss Compendium®

- [Für Medizinalpersonen](#)
- [Für Patientinnen und Patienten](#)

More Information in English:

[Link to Drug Information Portal, a service of the U.S. National Library of Medicine, National Institutes of Health](#)

[Link to MedlinePlus, a service of the U.S. National Library of Medicine, National Institutes of Health](#)

[Link to National Cancer Institute](#)

[Link zu Wiki](#)

[Link zu PharmaWiki](#)

[Link to Physicians Desk Reference \(PDR\)](#)

[Link to European Medicines Agency \(EMA\)](#)

[Info for Patients presented by Scott Hamilton from Chemocare.com](#)

Altretamine

According to the NCI website Altretamine is a synthetic cytotoxic s-triazine derivative similar in structure to alkylating agent triethylenemelamin with antineoplastic activity. Although the precise mechanism by which altretamine exerts its cytotoxic effect is unknown, N-demethylation of altretamine may produce reactive intermediates which covalently bind to DNA, resulting in DNA damage.

Indikationen/Anwendungsmöglichkeiten:

Ovarian cancer

[Link to MedlinePlus, a service of the U.S. National Library of Medicine, National Institutes of Health](#)

[Link to National Cancer Institute](#)

[Link zu Wiki](#)

[Link to Physicians Desk Reference \(PDR\)](#)

[Info for Patients presented by Scott Hamilton from Chemocare.com](#)

[Zytostatikum](#)

AMG 160 (anti-PSMAxanti-CD3-BiTE-bispecific-T-cell-engager)

According to the NCI website, anti-PSMA/CD3 BiTE antibody AMG 160 is a half-life extended (HLE), bispecific T-cell engager (BiTE) antibody composed of two single-chain variable fragments (scFv), one directed against the tumor-associated antigen (TAA) human prostate specific membrane antigen (PSMA), fused to one that is directed against the CD3 antigen found on T lymphocytes, with potential immunostimulating and antineoplastic activities. Upon administration of anti-PSMA/CD3 BiTE antibody AMG 160, this bispecific antibody binds to both CD3 on cytotoxic T lymphocytes (CTLs) and PSMA found on PSMA-expressing tumor cells. This activates and redirects CTLs to PSMA-expressing tumor cells, which results in the CTL-mediated cell death of PSMA-expressing tumor cells. PSMA, a tumor

associated antigen, is overexpressed on the surface of metastatic and hormone-refractory prostate cancer cells.

More Information in English:

[AdisInsight](#)

[Link to National Cancer Institute](#)

AMG 330 - anti-CD33/CD3 BiTE antibody

According to the NCI website, **AMG 330** is a proprietary recombinant bispecific T-cell engager (BiTE) antibody composed of two single-chain variable fragments (scFv), one directed against the tumor-associated antigen (TAA) CD33 fused to one that is directed against the CD3 antigen found on T-lymphocytes, with potential immunostimulating and antineoplastic activities. Upon administration of anti-CD33/CD3 BiTE antibody AMG 330, this bispecific antibody binds to both the CD3 antigen on cytotoxic T-lymphocytes (CTLs) and the CD33 antigen found on CD33-expressing tumor cells. This activates and redirects CTLs to CD33-expressing tumor cells, which results in the CTL-mediated cell death of CD33-expressing tumor cells. CD33, a myeloid differentiation antigen, is expressed on normal non-pluripotent hematopoietic stem cells and overexpressed on neoplastic cells in patients with acute myeloid leukemia. Check for [active clinical trials](#) using this agent. ([NCI Thesaurus](#))

[Link to National Cancer Institute](#)

AMG 673 - anti-CD33/CD3 BiTE antibody

According to the NCI website **AMG 673** or **anti-CD33/CD3 BiTE antibody AMG 673** is a bispecific T-cell engager (BiTE) antibody composed of two single-chain variable fragments (scFv), one directed against the tumor-associated antigen (TAA) CD33 fused to one that is directed against the CD3 antigen found on T lymphocytes, with potential immunostimulating and antineoplastic activities. Upon administration of anti-

CD33/CD3 BiTE antibody AMG 673, this bispecific antibody binds to both the CD3 antigen on cytotoxic T lymphocytes (CTLs) and the CD33 antigen found on CD33-expressing tumor cells. This activates and redirects CTLs to CD33-expressing tumor cells, which results in the CTL-mediated cell death of CD33-expressing tumor cells. CD33, a myeloid differentiation antigen, is expressed on normal non-pluripotent hematopoietic stem cells and overexpressed on a variety of cancer cell types, including acute myeloid leukemia (AML). It plays a key role in tumor initiation, proliferation and progression. Check for [active clinical trials](#) using this agent.

[Link to National Cancer Institute](#)

Amifostine

Ethiol®; According to the NCI amifostine trihydrate is the trihydrate form of a phosphorylated aminosulphydryl compound. After dephosphorylation of amifostine by alkaline phosphatase to an active free sulphydryl (thiol) metabolite, the thiol metabolite binds to and detoxifies cytotoxic platinum-containing metabolites of cisplatin and scavenges free radicals induced by cisplatin and ionizing radiation.

Indikationen/Anwendungsmöglichkeiten gemäss Arzneimittelkompendium:

Chemotherapie

Reduktion des Infektionsrisikos in Verbindung mit Neutropenie (z.B. neutropenisches Fieber), zurückzuführen auf die Kombinationsmedikation Cyclophosphamid und Cisplatin bei Patientinnen mit fortgeschrittenem Ovarialkarzinom (FIGO-Stadium III oder IV) unter 70 Jahren.

Reduktion der Nephrotoxizität einer Cisplatin enthaltenden Chemotherapie bei Patientinnen mit fortgeschrittenem Ovarialkarzinom (FIGO-Stadium III oder IV) unter 70 Jahren.

Strahlentherapie

Verminderung der akuten und späten Xerostomie (Mundtrockenheit) bei Patienten mit Kopf- und Hals-Karzinomen, die eine standardisierte Strahlentherapie erhalten sollen, die mindestens 75% der Ohrspeicheldrüsen einschliesst.

[Link to MedlinePlus, a service of the U.S. National Library of Medicine, National Institutes of Health](#)

[Link to National Cancer Institute](#)

[Link zu Wiki](#)

[Link zu PharmaWiki](#)

[Link to Physicians Desk Reference \(PDR\)](#)

[Link zur Fachinformation des Arzneimittel-Kompendium der Schweiz](#)

[Info for Patients presented by Scott Hamilton from Chemocare.com](#)

[Zytoprotektion](#)

Amrubicin

The hydrochloride salt of a third-generation synthetic 9-amino-anthracycline with antineoplastic activity.

[Link to National Cancer Institute](#)

[Link zu Wiki](#)

[Link zu PharmaWiki](#)

[Link to European Medicines Agency \(EMA\)](#)

AMV564

According to the NCI website **AMV564 anti-CD33 antigen/CD3 receptor bispecific monoclonal antibody AMV564** is an anti-CD33/anti-CD3 bispecific tetravalent antibody, with potential immunostimulatory and antineoplastic activities. Anti-CD33/CD3 tetravalent bispecific monoclonal antibody AMV564 possesses two antigen-recognition and binding sites, one for the CD3 complex, a group of T-cell surface glycoproteins that complex with the T-cell receptor (TCR), and one for CD33, a tumor-associated antigen (TAA) overexpressed on the surface of a variety of tumor cell types. Upon infusion of AMV564, this bispecific antibody binds to CD3-expressing T cells and CD33-expressing tumor cells, thereby crosslinking CD33-expressing tumor cells and cytotoxic T-lymphocytes (CTLs). This may result in a potent CTL-mediated cell lysis of CD33-expressing cells. CD33, a glycoprotein expressed by a variety of cancers, including the majority of acute myeloid leukemias (AMLs), and normal non-pluripotent hematopoietic stem cells, plays a key role in tumor initiation, proliferation and progression. Check for [active clinical trials](#) using this agent.

[Link to National Cancer Institute](#)

Anagrelide - XAGRID®, THROMBOREDUCTIN® ANAGRELID NORDIC®

According to the NCI website, anagrelide hydrochloride is the salt of a synthetic quinazoline derivative; anagrelide hydrochloride reduces platelet production through a decrease in megakaryocyte maturation. Anagrelide inhibits cyclic AMP phosphodiesterase, as well as ADP- and collagen-induced platelet aggregation. At therapeutic doses, it does not influence white cell counts or coagulation parameters. Anagrelide is used for treatment of essential thrombocythemia to reduce elevated platelet counts and the risk of thrombosis. Check for [active clinical](#) trials using this agent. ([NCI Thesaurus](#))

Indikationen/Anwendungsmöglichkeiten gemäss Compendium®:

- Anagrelid Nordic ist zur Behandlung der essentiellen Thrombozythämie bei Risikopatienten vorgesehen ([vgl. Fachinformation Compendium®](#))
- Thromboreductin ist zur Behandlung der essentiellen Thrombozythämie bei Risikopatienten vorgesehen ([vgl. Fachinformation Compendium®](#))
- Xagrid ist zur Behandlung der Essentiellen Thrombozythämie vorgesehen ([vgl. Fachinformation Compendium®](#))

XAGRID®: [Merkblätter für Patientinnen und Patienten](#)

[Link to MedlinePlus, a service of the U.S. National Library of Medicine, National Institutes of Health](#)

[Link to National Cancer Institute](#)

[Link zu Wiki](#)

[Link zu PharmaWiki](#)

[Link to Physicians Desk Reference \(PDR\)](#)

[Link to European Medicines Agency \(EMA\)](#)

[Info for Patients presented by Scott Hamilton from Chemocare.com](#)

Anastrozol - ARIMIDEX® und diverse Generika

Arimidex® sowie diverse Generika - A nonsteroidal inhibitor of estrogen synthesis that resembles paclitaxel in chemical structure.

Indikationen/Anwendungsmöglichkeiten gemäss Compendium®:

- Adjuvante Behandlung beim Mammakarzinom mit Östrogen- oder Progesteron-Rezeptor-positivem oder mit unbekanntem Hormon-Rezeptor-Status (Stadium I und II) bei postmenopausalen Frauen.
- Behandlung des fortgeschrittenen Mammakarzinoms bei postmenopausalen Frauen.

[Merkblatt für Patientinnen und Patienten](#)

Link zur Fachinformation des Compendium®:

Medikamenteninformation: [Für den Arzt](#) [Patienteninformation](#)

Information des Médicaments: [Info prof.](#) [Info patient](#)

Informazione sul medicamento: [info per il paziente](#)

[Link to National Cancer Institute](#)

[Link to Wikipedia](#)

[Link zu PharmaWiki](#)

[Link to Physicians Desk Reference \(PDR\)](#)

[Info for Patients presented by Scott Hamilton from Chemocare.com](#)

Apalutamide - ERLEADA®

According to the NCI website, Apalutamide is a small molecule and androgen receptor (AR) antagonist with potential antineoplastic activity. Apalutamide binds to AR in target tissues thereby preventing androgen-induced receptor activation and facilitating the formation of inactive complexes that cannot be translocated to the nucleus. This prevents binding to and transcription of AR-responsive genes. This ultimately inhibits the expression of genes that regulate prostate cancer cell proliferation and may lead to an inhibition of cell growth in AR-expressing tumor cells. Check for [active clinical trials](#) using this agent. ([NCI Thesaurus](#)) [Patient Information](#)

Indikationen/Anwendungsmöglichkeiten gemäss Compendium®

-

ERLEADA in Kombination mit einer Androgendeprivationstherapie (ADT) ist indiziert für die Behandlung von erwachsenen Patienten mit nicht-metastasiertem, kastrationsresistentem Prostatakarzinom (NM-CRPC), bei denen ein hohes Risiko für eine Entwicklung von Metastasen besteht (insbesondere PSADT ≤10 Monate; siehe «Klinische Wirksamkeit»).

[Link zur Fachinformation von Compendium.ch®](#)

[Link zur Patienteninfo von Compendium®](#)

[Merkblätter für Patientinnen und Patienten](#) (am 16.6.20 noch nicht erhältlich gewesen)

More Information in English:

[Link to Drug Information Portal, a service of the U.S. National Library of Medicine, National Institutes of Health](#)

[Link to MedlinePlus, a service of the U.S. National Library of Medicine, National Institutes of Health](#)
[Link to National Cancer Institute](#)

[Wiki](#)

[Link zu PharmaWiki](#)

[Link to Physicians Desk Reference \(PDR\)](#)

[Link to European Medicines Agency \(EMA\)](#)

[Info for Patients presented by Scott Hamilton from Chemocare.com](#)

Apatinib (Rivoceranib)

According to the NCI website, apatinib is an orally bioavailable, small-molecule receptor tyrosine kinase inhibitor with potential antiangiogenic and antineoplastic activities. Apatinib selectively binds to and inhibits vascular endothelial growth factor receptor 2, which may inhibit VEGF-stimulated endothelial cell migration and proliferation and decrease tumor microvessel density. In addition, this agent mildly inhibits c-Kit and c-SRC tyrosine kinases. Check for [active clinical trials](#) using this agent. ([NCI Thesaurus](#))

More Information in English:

[Inxight: Drugs \(NIH\)](#)

[AdisInsight](#)

[Link to Drug Information Portal, a service of the U.S. National Library of Medicine, National Institutes of Health](#)

[Link to National Cancer Institute](#)

[Link to European Medicines Agency \(EMA\)](#)

APR-246

According to the NCI website **PRIMA-1 analog APR-246** is a methylated derivative and structural analog of PRIMA-1 (p53 re-activation and induction of massive apoptosis), with potential antineoplastic activity. Upon administration, PRIMA-1 analog APR-246 covalently modifies the core domain of mutated forms of cellular tumor antigen p53 (p53) through the alkylation of thiol groups. These modifications restore both the wild-type conformation and function to mutant p53, which reconstitutes endogenous p53

activity, leading to cell cycle arrest and apoptosis in tumor cells. This agent may work synergistically with other antineoplastic agents. p53, a tumor suppressor and transcription factor normally activated upon DNA damage, is frequently mutated and overexpressed in cancer cells; it plays a key role in both DNA repair and the induction of apoptosis. Check for [active clinical trials](#) using this agent.

[Link to National Cancer Institute](#)

ARV-110 - androgen receptor degrader

According to the NCI website, ARV-110 is an orally available selective androgen receptor (AR)-targeted protein degrader, using the proteolysis targeting chimera (PROTAC) technology, with potential antineoplastic activity. ARV-110 is composed of an AR ligand attached to an E3 ligase recognition moiety. Upon oral administration, ARV-110 targets and binds to the AR ligand binding domain. E3 ligase is recruited to the AR by the E3 ligase recognition moiety and the AR target protein is tagged by ubiquitin. This causes ubiquitination and degradation of AR by the proteasome. This prevents the expression of AR target genes and halts AR-mediated signaling. This results in an inhibition of proliferation in AR-overexpressing tumor cells. In addition, the degradation of the AR protein releases the ARV-110 is released and can bind to additional AR target proteins. AR plays a key role in the proliferation of castration-resistant prostate cancer cells (CRPC). Check for [active clinical trials](#) using this agent. ([NCI Thesaurus](#))

[Link to National Cancer Institute](#)

Asciminib

According to the NCI website, Asciminib is An orally bioavailable, allosteric Bcr-Abl tyrosine kinase inhibitor with potential antineoplastic activity. Designed to overcome resistance, asciminib binds to the Abl portion of the Bcr-Abl fusion protein at a location that is distinct from the ATP-binding domain. This binding results in the inhibition of Bcr-Abl-mediated proliferation and enhanced apoptosis of Philadelphia chromosome-positive (Ph+) hematological malignancies. The Bcr-Abl fusion protein tyrosine kinase is an abnormal enzyme produced by leukemia cells that contain the Philadelphia chromosome. Check for [active clinical trials](#) using this agent. ([NCI Thesaurus](#))

More Information in English:

[Link to Drug Information Portal, a service of the U.S. National Library of Medicine, National Institutes of Health](#)

[Link to National Cancer Institute](#)

[Link to European Medicines Agency \(EMA\)](#)

Asparaginase - ONCASPAR Inj Lös 3750 IE/5ml

According to the NCI asparaginase is an enzyme isolated from the bacterium *Escherichia coli* or the bacterium *Erwinia carotovora* with antileukemic activity. Asparaginase hydrolyzes L-asparagine to L-aspartic acid and ammonia in leukemic cells, resulting in the depletion of asparagine, inhibition of protein synthesis, cell cycle arrest in the G1 phase, and apoptosis in susceptible leukemic cell populations.

Indikationen/Anwendungsmöglichkeiten gemäss MedlinePlus:

Asparaginase is used with other chemotherapy drugs to treat a certain type of acute lymphocytic leukemia (ALL; a type of cancer of the white blood cells). Asparaginase is an enzyme that interferes with natural substances necessary for cancer cell growth. It works by killing or stopping the growth of cancer cells.

Gemäss Compendium:

Oncaspar ist als Bestandteil einer antineoplastischen Kombinationstherapie bei akuter lymphatischer Leukämie (ALL) angezeigt.

[Fachinfo Compendium](#)

[Link to MedlinePlus, a service of the U.S. National Library of Medicine, National Institutes of](#)

Health**[Link to National Cancer Institute](#)****[Link zu Wiki](#)****[Link to Physicians Desk Reference \(PDR\)](#)****[Link to European Medicines Agency \(EMA\)](#)****[Info for Patients presented by Scott Hamilton from Chemocare.com](#)**

Atezolizumab - TECENTRIQ®

According to the NCI website Atezolizumab is a humanized, Fc optimized, monoclonal antibody directed against the protein ligand PD-L1 (programmed cell death-1 ligand 1), with potential immune checkpoint inhibitory and antineoplastic activities.

Indikationen gemäss Compendium.ch®*Nicht-kleinzelliges Lungenkarzinom (NSCLC)*

Tecentriq ist indiziert zur Behandlung von Patienten mit lokal fortgeschrittenem oder metastasiertem nicht-kleinzelligem Lungenkarzinom (NSCLC), nach vorausgegangener Chemotherapie.

Kleinzelliges Lungenkarzinom (SCLC)

Tecentriq ist in Kombination mit Carboplatin und Etoposid indiziert für die Erstlinientherapie von Patienten mit fortgeschrittenem kleinzelligem Lungenkarzinom (ES-SCLC, extensive-stage small cell lung cancer).

Metastasiertes Urothelkarzinom

Tecentriq ist indiziert für die Behandlung von erwachsenen Patienten mit lokal fortgeschrittenem oder metastasiertem Urothelkarzinom nach vorangegangener Platin-basierter Chemotherapie.

Triple-negatives Mammakarzinom

Tecentriq ist in Kombination mit Nab-Paclitaxel indiziert für die Behandlung von erwachsenen Patientinnen mit nicht-resezierbarem, lokal fortgeschrittenem oder metastasiertem triple-negativem Mammakarzinom (TNBC), deren Tumore eine PD-L1-Expression $\geq 1\%$ aufweisen und die keine vorherige Chemotherapie oder zielgerichtete systemische Therapie wegen ihrer fortgeschrittenen Erkrankung

erhalten haben (Nab-Paclitaxel Dosierung siehe «Dosierung/Anwendung»).

Link zur Fachinformation von Compendium.ch@:

Medikamenteninformation: [Für den Arzt](#)

More Information in English:

[Link to Drug Information Portal, a service of the U.S. National Library of Medicine, National Institutes of Health](#)

[Link to MedlinePlus, a service of the U.S. National Library of Medicine, National Institutes of Health](#)

[Link to National Cancer Institute](#)

[Link zu Wiki](#)

[Link zu PharmaWiki](#)

[Link to Physicians Desk Reference \(PDR\)](#)

[Link to European Medicines Agency \(EMA\)](#)

[Info for Patients presented by Scott Hamilton from Chemocare.com](#)

[Cancer immunotherapy](#)

AUTO3

According to the NCI website *autologous anti-CD19/CD22 CAR-T cells AUTO3* is a preparation of autologous T lymphocytes that have been transduced with a bicistronic retroviral vector encoding both an anti-CD19 chimeric antigen receptor (CAR) fused to OX40 co-stimulatory domain and an anti-CD22 CAR linked to the intracellular signaling domains of 4-1BB (CD137) and the zeta chain of the TCR/CD3 complex (TCRzeta; CD247; CD3zeta), optimized with a novel pentameric spacer derived from the collagen oligomeric matrix protein (COMP), with potential antineoplastic activity. Upon administration, the autologous anti-CD19/CD22 CAR T cells AUTO3 bind to and induce selectivity in tumor cells expressing CD19 and CD22. CD19 and CD22, both transmembrane phosphoglycoproteins expressed on the surface of cells in the B lineage, are often overexpressed on malignant B cells. By simultaneously targeting two B-cell antigens, this preparation may minimize relapse due to single antigen loss in patients with B-cell malignancies. Check for [active clinical trials](#) using this agent.

[Link to National Cancer Institute](#)

Avadomide

According to the NCI website, Avadomide is a novel, small molecule cereblon-modulating agent with potential antineoplastic, antiangiogenic and immunomodulatory activities. Upon oral administration, avadomide binds to and modulates cereblon to promote recruitment of the hematopoietic transcription factors Aiolos and Ikaros to the Cullin-4 RING E3 ubiquitin ligase complex. This binding results in the ubiquitination and rapid proteasomal degradation of Aiolos and Ikaros and the derepression of interferon (IFN)-stimulated genes, including DDX58 and IRF7, leading to apoptosis of certain tumor cells. Additionally, Aiolos degradation leads to derepression of the IL2 gene, thereby enhancing interleukin-2 production, costimulation of T lymphocytes and IL-2-induced T-cell proliferation. Avadomide may also promote the activation of natural killer (NK) cells, potentially enhancing tumor cell killing. Aiolos and Ikaros are transcriptional repressors known to play an important role in normal B and T cell function. Check for [active clinical trials](#) using this agent. ([NCI Thesaurus](#))

[Link to Drug Information Portal, a service of the U.S. National Library of Medicine, National Institutes of Health](#)

[Link to National Cancer Institute](#)

[Link to European Medicines Agency \(EMA\)](#)

Avelumab - BAVENCIO®

According to the NCI website Avelumab is a human immunoglobulin G1 (IgG1) monoclonal antibody directed against the human immunosuppressive ligand programmed death-ligand 1 ([PD-L1](#)) protein, with potential immune checkpoint inhibitory and antineoplastic activities. Upon administration, avelumab binds to PD-L1 and prevents the interaction of PD-L1 with its receptor programmed cell death protein 1 (PD-1). This inhibits the activation of PD-1 and its downstream signaling pathways. This may restore immune

function through the activation of cytotoxic T-lymphocytes (CTLs) targeted to PD-L1-overexpressing tumor cells. In addition, avelumab induces an antibody-dependent cellular cytotoxic (ADCC) response against PD-L1-expressing tumor cells. PD-1, a cell surface receptor belonging to the immunoglobulin superfamily expressed on T-cells, negatively regulates T-cell activation and effector function when activated by its ligand, and plays an important role in tumor evasion from host immunity. PD-L1, a transmembrane protein, is overexpressed on a variety of tumor cell types and is associated with poor prognosis.

Indikation gemäss Compendium

- Bavencio wird angewendet zur Behandlung von Patienten mit metastasiertem Merkelzellkarzinom (MCC).

Arzneimittelinformation

[Für den Arzt](#)

[Link to Drug Information Portal, a service of the U.S. National Library of Medicine, National Institutes of Health](#)

[Link to National Cancer Institute](#)

[Link to WIKI](#)

[Link zu PharmaWiki](#)

[Link to Physicians Desk Reference \(PDR\)](#)

[Link to European Medicines Agency \(EMA\)](#)

[Info for Patients presented by Scott Hamilton from Chemocare.com](#)

[The IUPHAR/BPS Guide to PHARMACOLOGY](#)

[Cancer immunotherapy](#)

axicabtagene ciloleucel – YESCARTA®

According to the NCI website *axicabtagene ciloleucel* is a preparation of autologous peripheral blood T-lymphocytes (PBTCL) that have been transduced with a gammaretroviral vector expressing a chimeric antigen receptor (CAR) consisting of an anti-CD19 single chain variable fragment (scFv) coupled to the costimulatory signaling domain CD28 and the zeta chain of the T-cell receptor (TCR)/CD3 complex (CD3 zeta), with potential immunostimulating and antineoplastic activities. Upon intravenous infusion and re-introduction of axicabtagene ciloleucel into the patient, these cells bind to and induce selective toxicity in CD19-expressing tumor cells. CD19 antigen is a B-cell specific cell surface antigen that is expressed in all B-cell lineage malignancies. CD3 zeta is one of several membrane-bound polypeptides found in the TCR/CD3 complex; it regulates both the assembly and cell surface expression of TCR complexes. CD28 is essential for CD4+ T-cell proliferation, interleukin-2 production, and T-helper type-2 (Th2) development. Check for [active clinical trials](#) using this agent.

Indikationen gemäss Compendium.ch®

YESCARTA ist eine gegen CD19 gerichtete, genetisch modifizierte autologe T-Zell-Immuntherapie und wird angewendet bei erwachsenen Patienten zur Behandlung von rezidiertem oder refraktärem diffus grosszelligem B-Zell-Lymphom (DLBCL) und primär mediastinalem grosszelligem B-Zell-Lymphom (PMBCL) nach zwei oder mehr systemischen Therapielinien.

Link zur Fachinformation von Compendium.ch®:

Medikamenteninformation: [Für den Arzt](#)

According to the NCI website Axicabtagene ciloleucel is approved to treat:

- **B-cell non-Hodgkin lymphoma**(NHL), including the following types:
 - Diffuse large B-cell lymphoma (DLBCL).
 - DLBCL in patients who had follicular lymphoma.
 - Primary mediastinal large B-cell lymphoma.
 - High-grade B-cell lymphoma.

It is used in adults whose disease has relapsed or has not gotten better after at least two other types of systemic treatment.

Axicabtagene ciloleucel is also being studied in the treatment of other types of cancer.

More Information in English:

[Link to Drug Information Portal, a service of the U.S. National Library of Medicine, National Institutes of Health](#)

[Link to National Cancer Institute](#)

[Link zu Wiki](#)

[Link zu PharmaWiki](#)

[Link to Physicians Desk Reference \(PDR\)](#)

[Link to European Medicines Agency \(EMA\)](#)

[Info for Patients presented by Scott Hamilton from Chemocare.com](#)

Axitinib - INLYTA®

Axitinib ist ein oral verfügbarer Tyrosin Kinase Hemmer. Er hemmt den Rezeptor für den vaskulären endothelialen Wachstumsfaktor (VEGF), ein gefässbildungsförderndes Zytokin sowie den Rezeptor für den von Blutplättchen freigesetzten Wachstumsfaktor (PDGF). Dies führt zu einer Hemmung der Blutgefässbildung.

Indikationen/Anwendungsmöglichkeiten gemäss Compendiums®:

Inlyta® ist indiziert zur Behandlung von Patienten mit fortgeschrittenem Nierenzellkarzinom (RCC) nach Versagen einer vorherigen systemischen Therapie.

[Merksblätter für Patientinnen und Patienten](#)

Link zur Fachinformation des Compendiums®:

Medikamenteninformation: [Für den Arzt](#) [Patienteninformation](#)

Information des Médicaments: [Info prof.](#) [Info patient](#)

Informazione sul medicamento: [info per il paziente](#)

More Information in English:

[Link to Drug Information Portal, a service of the U.S. National Library of Medicine, National Institutes of Health](#)

[Link to National Cancer Institute](#)

[Link zu Wiki](#)

[Link zu PharmaWiki](#)

[Link to Physicians Desk Reference \(PDR\)](#)

[Link to European Medicines Agency \(EMA\)](#)

[Info for Patients presented by Scott Hamilton from Chemocare.com](#)

[Tyrosin Kinase Inhibitor](#)

Azacitidine - VIDAZA®

According to the NCI website, azacitidine is a pyrimidine nucleoside analogue of cytidine with antineoplastic activity. Azacitidine is incorporated into DNA, where it reversibly inhibits DNA methyltransferase, thereby blocking DNA methylation. Hypomethylation of DNA by azacitidine may activate tumor suppressor genes silenced by hypermethylation, resulting in an antitumor effect. This agent is also incorporated into RNA, thereby disrupting normal RNA function and impairing tRNA cytosine-5-methyltransferase activity.

Indikationen/Anwendungsmöglichkeiten gemäss Compendium®:

Behandlung von Patienten, die für eine Transplantation hämatopoetischer Stammzellen nicht geeignet sind und eines der folgenden Krankheitsbilder aufweisen:

- Myelodysplastisches Syndrom mit intermediärem oder hohem Risiko gemäss International Prognostic Scoring System (IPSS) vom Typ refraktäre Zytopenie mit multilineärer Dysplasie (RCMD) oder refraktäre Anämie mit 5-19% Knochenmarksblasten (RAEB I und II)

- Chronisch myelomonozytäre Leukämie
- Akute myeloische Leukämie (AML) mit 20-30% Knochenmarksblasten und Mehrlinien-Dysplasie (gemäss Klassifikation der World Health Organisation (WHO) 2008)
- Akute myeloische Leukämie (AML) mit >30% Knochenmarksblasten, gemäss Klassifikation der WHO, bei älteren Patienten, die für eine intensive Chemotherapie nicht geeignet sind oder diese nicht vertragen.

[Link zur Fachinformation des Compendium®](#)

[Link to MedlinePlus, a service of the U.S. National Library of Medicine, National Institutes of Health](#)

[Link to National Cancer Institute](#)

[Link zu Wiki](#)

[Link zu PharmaWiki](#)

[Link to Physicians Desk Reference \(PDR\)](#)

[Link to European Medicines Agency \(EMA\)](#)

[Info for Patients presented by Scott Hamilton from Chemocare.com](#)