Glossary A-Z

Wirkstoffe S

Sacituzumab Govitecan - TRODELVY®

Navigation überspringen A B C D E F G H I K L M N O P Q R W T U V Z

According to the NCI website, *sacituzumab govitecan* is an antibody-drug conjugate containing the humanized monoclonal antibody, hRS7, against tumor-associated calcium signal transducer 2 (TACSTD2 or TROP2) and linked to the active metabolite of irinotecan, 7-ethyl-10-hydroxycamptothecin (SN-38), with potential antineoplastic activity. The antibody moiety of sacituzumab govitecan selectively binds to TROP2. After internalization and proteolytic cleavage, SN-38 selectively stabilizes topoisomerase I-DNA covalent complexes, resulting in DNA breaks that inhibit DNA replication and trigger apoptosis. TROP2, also known as epithelial glycoprotein-1 (EGP-1), is a transmembrane calcium signal transducer that is overexpressed by a variety of human epithelial carcinomas; this antigen is involved in the regulation of cell-cell adhesion and its expression is associated with increased cancer growth, aggressiveness and metastasis. Check for active clinical trials using this agent. (NCI Thesaurus)

Indikationen und Anwendungsmöglichkeiten gemäss Compendium:

TRODELVY ist indiziert zur Behandlung von erwachsenen Patienten mit nicht resezierbarem lokal fortgeschrittenem oder metastasiertem triple-negativem Mammakarzinom (*metastatic Triple-Negative Breast Cancer*, mTNBC), die mindestens zwei Vortherapien erhalten haben, davon mindestens eine im metastasierten Stadium (siehe «Klinische Wirksamkeit»).

November 23, 2021: Trodelvy® (sacituzumab govitecan) Granted European Commission Marketing Authorization for Treatment of Metastatic Triple-Negative Breast Cancer in Second Line -- Marketing Authorization Based on Phase 3 ASCENT Study Showing Trodelvy Significantly Improved Overall Survival vs. Physician's Choice of Chemotherapy in Metastatic Triple-Negative Breast 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

Wiki

Sapacitabine

According to the NCI website sapacitabine is an orally bioavailable pyrimidine analogue prodrug with potential antineoplastic activity. Sapacitabine is hydrolyzed by amidases to the deoxycytosine analogue CNDAC (2'-Cyano-2'-deoxyarabinofuranosylcytosine), which is then phosphorylated into the active triphosphate form. As an analogue of deoxycytidine triphosphate, CNDAC triphosphate incorporates into DNA strands during replication, resulting in single-stranded DNA breaks during polymerization due to beta-elimination during the fidelity checkpoint process; cell cycle arrest in the G2 phase and apoptosis ensue. The unmetabolized prodrug may exhibit antineoplastic activity as well.

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

Sargramostim

According to the NCI website sargramostim is a recombinant therapeutic agent which is chemically identical to or similar to endogenous human GM-CSF. Binding to specific cell surface receptors, sargramostim modulates the proliferation and differentiation of a variety of hematopoietic progenitor cells with some specificity towards stimulation of leukocyte production and may reverse treatment-induced neutropenias. This agent also promotes antigen presentation, up-regulates

antibody-dependent cellular cytotoxicity (ADCC), and increases interleukin-2-mediated lymphokine-activated killer cell function; it may also augment host antitumoral immunity..

Indikationen/Anwendungsmöglichkeiten gemäss MedlinePlus:
Sargramostim is a synthetic version of substances naturally produced by your body. It helps you to fight infections so you can receive your next chemotherapy cycle as scheduled.

Link to Drug Information Portal

Link to MedlinePlus

Link to National Cancer Institute

Link zu Wiki

Link to Physicians Desk Reference (PDR)

Info for Patients presented by Scott Hamilton from Chemocare.com

Granulocyte macrophage colony-stimulating factor

Savolitinib

According to the NCI website, *volitinib* (savolitinib) is an orally bioavailable inhibitor of the c-Met receptor tyrosine kinase with potential antineoplastic activity. Volitinib selectively binds to and inhibits the activation of c-Met in an ATP-competitive manner, and disrupts c-Met signal transduction pathways. This may result in cell growth inhibition in tumors that overexpress the c-Met protein. C-Met encodes the hepatocyte growth factor receptor tyrosine kinase and plays an important role in tumor cell proliferation, survival, invasion, and metastasis, and tumor angiogenesis; this protein is overexpressed or mutated in a variety of cancers. 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

Wiki

Seliciclib

According to the NCI website seliciclib is an orally bioavailable, small-molecule cyclin-dependent kinase (CDK) inhibitor with potential proapoptotic and antineoplastic activities. Seliciclib primarily inhibits CDK2/E, CDK2/A, CDK7 and CDK9 by competing for their ATP binding sites, leading to a disruption of cell cycle progression. In addition, this agent appears to interfere with CDK-mediated phosphorylation of the carboxy-terminal domain of RNA polymerase II, inhibiting RNA polymerase II-dependent transcription, which may result in the down-regulation of antiapoptotic proteins such as induced myeloid leukemia cell differentiation protein Mcl-1. CDKs, serine/threonine kinases that play an important role in cell cycle regulation, are overexpressed in various malignancies. Mcl-1 belongs to the Bcl-2 family of antiapoptotic proteins and is a protein crucial to the survival of a range of tumor cell types.

Link to Drug Information Portal
Link to MedlinePlus
Link to National Cancer Institute
Link zu Wiki

Cyclin-dependent-kinases

Selinexor - XPOVIO® (USA)

According to the NCI website, selinexor is an orally available, small molecule inhibitor of CRM1 (chromosome region maintenance 1 protein, exportin 1 or XPO1), with potential antineoplastic activity. Selinexor modifies the essential CRM1-cargo binding residue cysteine-528, thereby irreversibly inactivating CRM1-mediated nuclear export of cargo proteins such as tumor suppressor proteins (TSPs),

including p53, p21, BRCA1/2, pRB, FOXO, and other growth regulatory proteins. As a result, this agent, via the approach of selective inhibition of nuclear export (SINE), restores endogenous tumor suppressing processes to selectively eliminate tumor cells while sparing normal cells. CRM1, the major export factor for proteins from the nucleus to the cytoplasm, is overexpressed in a variety of cancer cell types. Check for active clinical trials using this agent. (NCI Thesaurus)

Indikation gemäss NCI Eebsite:

- **Multiple myeloma** that has relapsed (come back) or is refractory (does not respond to treatment). It is used with dexamethasone in adults who have received at least four previous treatments that included at least two proteasome inhibitors, at least two immunomodulating agents, and an anti-CD38 monoclonal antibody.

This use is approved under FDA's Accelerated Approval Program. As a condition of approval, a confirmatory trial(s) must show that selinexor provides a clinical benefit in these patients.

A Global Randomized Trial Planned for Selinexor to Treat COVID-19

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 to European Medicines Agency (EMEA)

Selpercatinib - Retevmo® (USA)

According to the NCI website, Selpercatinib is an orally bioavailable selective inhibitor of wild-type, mutant and fusion products involving the proto-oncogene receptor tyrosine kinase rearranged during transfection (RET), with potential antineoplastic activity. Upon oral administration, selpercatinib selectively binds to and targets wild-type RET as well as various RET mutants and RET-containing fusion products. This results in an inhibition of cell growth of tumors cells that exhibit increased RET activity. In addition, selpercatinib targets, binds to and inhibits vascular endothelial growth factor receptor 1

(VEGFR1) and 3 (VEGFR3), and fibroblast growth factor receptor 1 (FGFR1), 2 (FGFR2), and 3 (FGFR3). RET overexpression, activating mutations, and fusions result in the upregulation and/or overactivation of RET tyrosine kinase activity in various cancer cell types; dysregulation of RET activity plays a key role in the development and progression of these cancers. Check for active clinical trials using this agent. (NCI Thesaurus)

Patient information

According to the NCI website Selpercatinib is approved to treat:

- **Medullary thyroid cancer** that has a certain mutation in the RET gene and is advanced or metastatic. It is used in adults and children aged 12 years and older who need systemic therapy.[1]
- **Non-small cell lung cancer** that has a RET fusion gene and is metastatic. It is used in adults.[1]
- **Thyroid cancer** that has a RET fusion gene and is metastatic or advanced. It is used in adults and children aged 12 years and older who need systemic therapy, including those who received radioactive iodine and it did not work or is no longer working.[1]

[1]This use is approved under FDA's Accelerated Approval Program. As a condition of approval, confirmatory trial(s) must show that selpercatinib provides a clinical benefit in these patients.

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

More Information in English:

Inxight: Drugs (NIH)

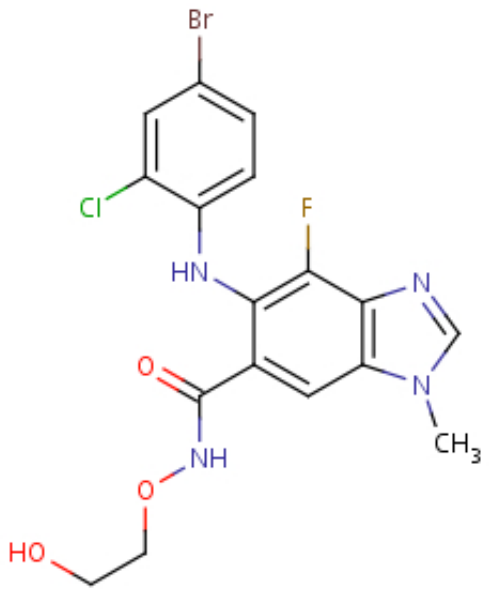
AdisInsight

Link to Drug Information Portal, a service of the U.S. National Library of Medicine, National Institutes of Health

Link to MedlinePlus, a service of the U.S. National Library of Medicine, National Institutes of Health
Link to National Cancer Institute

Wiki
Link to European Medicines Agency (EMEA)

Selumetinib



According to the NCI website Selumetinib is an orally bioavailable small molecule with potential antineoplastic activity. Selumetinib inhibits mitogen-activated protein kinase kinases (MEK or MAPK/ERK kinases) 1 and 2, which may prevent the activation of MEK1/2-dependent effector proteins and transcription factors, and so may inhibit cellular proliferation in MEK-overexpressing tumor cells. MEK 1 and 2 are dual-specificity kinases that are essential mediators in the activation of the RAS/RAF/MEK/ERK pathway, are often upregulated in various tumor cell types, and are drivers of diverse cellular activities, including cellular proliferation.

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

MaP-Kinase-Weg

Simlukafusp alfa / RO6874281

According to the NCI website, the anti-FAP/interleukin-2 fusion protein RO6874281 is a recombinant fusion protein comprised of a human monoclonal antibody directed against fibroblast activation protein-alpha (FAP) linked to an engineered, variant form of interleukin-2 (IL-2v), with potential immunostimulating and antineoplastic activities. Upon administration of RO6874281, the monoclonal antibody moiety recognizes and binds to FAP, thereby concentrating IL-2 in FAP-expressing tumor tissue. Subsequently, the IL-2 moiety of this fusion protein may stimulate a local immune response and activate natural killer (NK) cells and cytotoxic T-cells. FAP is a cell surface protein that is expressed on a wide variety of cancer cells. IL-2v cannot bind to IL-2 receptor-alpha (CD25, IL2Ra) and does not activate regulatory T-cells (Tregs). 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

Sintilimab

According to the NCI website, Sintilimab is a recombinant human monoclonal antibody directed against the negative immunoregulatory human cell surface receptor programmed cell death 1 (PD-1; PDCD1; PD1), with potential immune checkpoint inhibitory and antineoplastic activities. Upon administration, sintilimab binds to PD-1 and inhibits the binding of PD-1 to the PD-1 ligands programmed cell death-1 ligand 1 (PD-L1), and PD-1 ligand 2 (PD-L2). This prevents the activation of PD-1 and its downstream signaling pathways. This may restore immune function through the activation of both T cells and T-cell-mediated immune responses against tumor cells. PD-1, a transmembrane protein in the immunoglobulin (Ig) superfamily expressed on activated T cells, negatively regulates T-cell activation and effector function when activated by its ligands; it plays an important role in tumor evasion from host immunity. 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

Wiki
Link to European Medicines Agency (EMEA)

Sonidegib - ODOMZO®

Sonidegib - Odomzo® ist ein oral bioverfügbarer niedermolekularer „smoothened“ (Smo) Antagonist mit einer potentiellen antineoplastischen Aktivität. Sonidegib bindet selektiv an den Hedgehog (Hh)-Liganden Zelloberflächenrezeptor Smo, was zur Unterdrückung des Hh-Signalwegs und damit zur Hemmung von Tumorzellen, in denen dieser Signalweg abnormal aktiviert ist. Der Hh-Signalweg spielt eine wichtige Rolle beim Zellwachstum, der Differenzierung und Reparatur. Die unangemessene Aktivierung des Hh-Signalwegs und die unkontrollierte Zellproliferation, wie sie in einer Vielzahl von Krebsarten beobachtet wird, kann mit Mutationen im Hh-Ligand-Zelloberflächenrezeptor Smo assoziiert sein.

Indikationen/Anwendungsmöglichkeiten gemäss Compendium®:

- Odomzo ist indiziert zur Behandlung erwachsener Patienten mit fortgeschrittenem Basalzellkarzinom (BCC), das mit einer kurativen chirurgischen Behandlung oder einer radiologischen Therapie nicht behandelt werden kann.

Merkblätter für Patientinnen und Patienten **(Stichtag 26.3.20: noch nicht erhältlich)**

Link zur Fachinformation des Arzneimittel-Kompendiums der Schweiz:

Medikamenteniformation: 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 (EMEA)

More information 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

Smoothened agonist

Sorafenib - NEXAVAR®

Sorafenib - Nexavar® das Tosylatsalz von Sorafenib ist eine synthetische Verbindung, die auf die Signalkette für das Wachstum und die Tumorangiogenese abzielt. Sorafenib blockiert das Enzym Raf-

Kinase, ein kritischer Bestandteil des RAF/MEK/ERK-Signalweges, der die Zellteilung und Proliferation steuert; darüber hinaus hemmt Sorafenib die VEGFR-2/PDGFR-beta-Signalkaskade, wodurch die Tumor-Angiogenese blockiert wird.

Indikationen/Anwendungsmöglichkeiten gemäss Arzneimittel-Kompendium der Schweiz®:

- Zur Behandlung von Patienten mit Leberzellkarzinom.
- Behandlung von Patienten mit fortgeschrittenem Nierenzellkarzinom nach Nephrektomie und palliativer oder adjuvanter Vortherapie mit Cytokinen (IL-2, IFN).
- Behandlung von Patienten mit progredientem, lokal fortgeschrittenem oder metastasiertem, Radiojod-refraktärem, differenziertem Schilddrüsenkarzinom.

Merkblätter für Patientinnen und Patienten

Link zur Fachinformation des Compendium®:

Medikamenteniformation: 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 HealthLink to National Cancer InstituteLink zu Wiki
Link zu PharmaWiki
Link to Physicians Desk Reference (PDR)
Link to European Medicines Agency (EMEA)

More information 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

Receptor Tyrosine Kinase signaling Pathway
Receptor tyrosine kinases (RTK)s are very important signaling pathway, which not only include growth factor receptors such as **EGFR(HER)**, **VEGFR** , **PDGFR**, **FGGFR**, **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

Sotorasib/AMG-510

According to the NCI website, KRAS mutant-targeting AMG 510 (sotorasib) is an orally available agent that targets the specific KRAS mutation, p.G12C, with potential antineoplastic activity. Upon oral administration, KRAS mutant-targeting AMG 510 selectively targets the KRAS p.G12C mutant, at either the DNA, RNA or protein level, and prevents, through an as of yet not elucidated manner, expression of and/or tumor cell signaling through the KRAS p.G12C mutant. This may inhibit growth in KRAS p.G12C-expressing tumor cells. The KRAS p.G12C mutation is seen in some tumor cell types and plays a key role in tumor cell proliferation. 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

Wiki

Spartalizumab

According to the NCI website, Spartalizumab is a humanized monoclonal antibody directed against the negative immunoregulatory human cell surface receptor programmed death-1 (PD-1, PCD-1), with immune checkpoint inhibitory and antineoplastic activities. Upon administration, spartalizumab binds to PD-1 expressed on activated T-cells and blocks the interaction with its ligands, programmed cell death 1 ligand 1 (PD-L1, PD-1L1) and PD-1 ligand 2 (PD-L2, PD-1L2). The inhibition of ligand binding prevents PD-1-mediated signaling and results in both T-cell activation and the induction of T-cell-mediated immune responses against tumor cells. PD-1, an immunoglobulin (Ig) superfamily transmembrane protein and inhibitory receptor, negatively regulates T-cell activation. Check for active clinical trials using this agent. (NCI Thesaurus)

More Information in English:

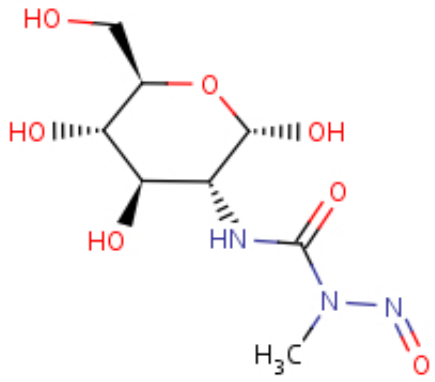
Inxight: Drugs (NIH)

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

Wiki
Link to European Medicines Agency (EMEA)

Streptozocin



According to the NCI website streptozocin is a methylnitrosourea antineoplastic antibiotic isolated from the bacterium Streptomyces achromogenes. Streptozocin alkylates DNA, forming inter-strand DNA cross-links and inhibiting DNA synthesis. Due to its glucose moiety, this agent is readily taken up by pancreatic beta cells, inducing diabetes mellitus at high concentrations.

Indikationen/Anwendungsmöglichkeiten gemäss MedlinePlus:
This medication is used to treat pancreatic islet cell cancer

Link to Drug Information Portal
Link to MedlinePlus
Link to National Cancer Institute
Link zu Wiki
Link zu PharmaWiki
Link to Physicians Desk Reference (PDR)
Info for Patients presented by Scott Hamilton from Chemocare.com
Zytostatikum

Sunitinib - SUTENT®

Sunitinib - Sutent® - ist das oral bioverfügbare Malat Salz eines Indolinon-basierten Tyrosinkinase-

Inhibitore mit einer potentiellen antineoplastischen Aktivität. Sunitinib blockiert die Tyrosinkinase-Aktivitäten des vaskulären endothelialen Wachstumsfaktor-Rezeptors 2 (VEGFR2) sowie den Plättchen-abhängigen Wachstumsfaktor-Rezeptor B (PDGFRB) und c-kit. Dadurch werden die Angiogenese und die Zellproliferation gehemmt. Dieses Mittel hemmt auch die Phosphorylierung der Fms bezogenen Tyrosinkinase 3 (FLT3).Es handelt sich um eine weitere Tyrosin-Rezeptor-Kinase, die von einigen leukämischen Zellen exprimiert wird.

Indikationen/Anwendungsmöglichkeiten gemäss Compendium®:

- Behandlung von Patienten mit fortgeschrittenem und/oder metastasierendem Nierenzellkarzinom (RCC).
- Behandlung von Patienten mit malignem gastrointestinalem Stromatumor (GIST) bei Resistenz oder Intoleranz auf Imatinib.
- Behandlung von Patienten mit nicht resezierbarem, gut differenziertem, fortgeschrittenem und/oder metastasierendem neuroendokrinen Pankreaskarzinom (pancreatic NET).

Merkblatt für Patientinnen und Patienten

Link zur Fachinformation des Compendium®:

Medikamenteniformation: 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

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More information for patients:

Link to MedlinePlus

Info for Patients presented by Scott Hamilton from Chemocare.com

Receptor Tyrosine Kinase signaling Pathway
Receptor tyrosine kinases (RTK)s are very important signaling pathway, which not only include growth factor receptors such as **EGFR(HER)**, **VEGFR** , **PDGFR**, **FGGFR**, **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

Sutimlimab

According to the NCI website, sutimlimab is a preparation of autologous CD8+ T lymphocytes targeting SLC45A2, a melanoma-associated antigen, with potential immunomodulating and antineoplastic activities. Following peripheral blood mononuclear cell (PBMC) collection and ex vivo expansion of SLC45A2-specific cytotoxic T-lymphocytes (CTLs), sutimlimab is re-infused into the patient, where they target and lyse SLC45A2-expressing tumor cells. While SLC45A2 is expressed by approximately 80% of cutaneous melanomas, its expression is limited in mature normal melanocytes, allowing high tumor selectivity and reduced potential for autoimmune toxicity. 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

Wiki
Link to European Medicines Agency (EMEA)

Sym021

According to the NCI website, the anti-PD-1 monoclonal antibody Sym021 is a humanized monoclonal antibody directed against the negative immunoregulatory human cell surface receptor programmed cell death 1 (PD-1 , PCD-1; PDCD1), with potential immune checkpoint inhibitory and antineoplastic activities. Upon administration, anti-PD-1 monoclonal antibody Sym021 binds to and inhibits PD-1 activation and its downstream signaling pathways. This may restore immune function through the activation of T cells and cell-mediated immune responses against tumor cells. PD-1, a transmembrane protein in the immunoglobulin superfamily (IgSF), is expressed on T cells and functions as an immune checkpoint that negatively regulates T-cell activation and effector function when activated by its ligands programmed cell death ligand 1 (PD-L1) or 2 (PD-L2). Activated PD-1 plays an important role in tumor evasion from host immunity. 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

Sym022

According to the NCI website, the anti-LAG-3 monoclonal antibody Sym022 is a recombinant, human Fc-inert monoclonal antibody targeting lymphocyte-activation gene 3 protein (LAG-3; LAG3), with potential immune checkpoint inhibitory and antineoplastic activities. Upon intravenous administration, monoclonal antibody Sym022 binds to human LAG-3 and blocks the interaction between LAG-3 and major histocompatibility complex class II (MHCII) molecules on the surface of antigen-presenting cells (APCs) and tumor cells. This prevents the negative regulation of T-cell activity that occurs via LAG-3-MHCII binding and enhances a cytotoxic T-lymphocyte (CTL)-mediated immune response against tumor cells. Additionally, Sym022 decreases LAG-3 surface levels through internalization and shredding. LAG-3 plays a key role in the activation of T cells and natural killer (NK) cells. 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

Sym0223

According to the NCI website, the anti-TIM-3 monoclonal antibody Sym023 is a recombinant, fully human monoclonal antibody against the inhibitory T-cell receptor T-cell immunoglobulin and mucin domain-containing protein 3 (TIM-3; TIM3; hepatitis A virus cellular receptor 2; HAVCR2), with

potential immune checkpoint inhibitory and antineoplastic activities. Upon administration, the anti-TIM-3 monoclonal antibody Sym023 binds to TIM-3 expressed on certain T cells, including tumor infiltrating lymphocytes (TILs). This abrogates T-cell inhibition, activates antigen-specific T lymphocytes and enhances cytotoxic T-cell-mediated tumor cell lysis, which results in a reduction in tumor cell proliferation. TIM-3, a transmembrane protein and immune checkpoint receptor, is associated with tumor-mediated immune suppression. Check for active clinical trials using this agent. (NCI Thesaurus)

More Information in English:

Inxight: Drugs (NIH)

AdisInsight
Link to National Cancer Institute