Glossary A-Z

Wirkstoffe L

Lapatinib - TYVERB®

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

Lapatinib ist ein synthetisches, oral-aktives Chinazolin mit einer potentiellen antineoplastischen Aktivität. Lapatinib blockiert reversibel die Phosphorylierung des epidermalen Wachstumsfaktor-Rezeptors (EGFR), ErbB2 und die Erk-1, Erk-2 und AKT Kinasen; es hemmt die Cyclin D Proteinspiegel in humanen Tumorzelllinien und Xenotransplantaten. EGFR und ErbB2 sind am Wachstum verschiedener Tumorarten beteiligt.

Indikationen/Anwendungsmöglichkeiten gemäss Compendium®:

- In Kombination mit Capecitabine zur Behandlung von Patientinnen mit fortgeschrittenem oder metastasierendem Brustkrebs, bei Überexpression von ErbB2 (HER2) mit Progression nach einer vorhergehenden Trastuzumab-Therapie im metastasierten Stadium.

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 to Wikipedia
Link zu PharmaWiki
Link to European Medicines Agency (EMEA)
Link to Physicians Desk Reference (PDR)

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

Tyrosin Kinase Inhibitor

Larotrectinib - VITRAKVI® (USA; EU)

According to the NCI website, ***larotrectinib sulfate*** is an orally available, tropomyosin receptor kinase (Trk) inhibitor, with potential antineoplastic activity. Upon administration, larotrectinib binds to Trk, thereby preventing neurotrophin-Trk interaction and Trk activation, which results in both the induction of cellular apoptosis and the inhibition of cell growth in tumors that overexpress Trk. Trk, a receptor tyrosine kinase activated by neurotrophins, is mutated in a variety of cancer cell types and plays an important role in tumor cell growth and survival. Check for active clinical trials using this agent. (NCI Thesaurus) Patient information

Indikationen/Anwendungsmöglichkeiten gemäss PDR (in der Schweiz nochnicht zugelassen; Stand 18. April 2020)

- For the treatment of neurotrophic receptor tyrosine kinase-positive solid tumors.
  NOTE: Larotrectinib has been designated an orphan drug by the FDA for the treatment of solid tumors with NTRK-fusion proteins.
  NOTE: Verify the presence of a NTRK gene fusion in the tumor specimen; in clinical studies, identification of positive NTRK gene fusion status was prospectively determined using next generation sequencing or fluorescence in situ hybridization.[63780]
- For the treatment of metastatic or surgically unresectable neurotrophic receptor tyrosine kinase (NTRK) gene fusion-positive solid tumors with no known acquired resistance mutation, in patients with no satisfactory alternative treatments or who have progressed following treatment.

More Information in English:

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

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Lazertinib

According to the NCI website, Lazertinib is an orally available third-generation, selective inhibitor of certain forms of the epidermal growth factor receptor (EGFR) with activating mutations, including the resistance mutation T790M, exon 19 deletions (Del19), and the L858R mutation, with potential antineoplastic activity. Upon administration, lazertinib specifically and irreversibly binds to and inhibits selective EGFR mutants, which prevents EGFR mutant-mediated signaling and leads to cell death in EGFR mutant-expressing tumor cells. Lazertinib may inhibit programmed cell death-1 ligand 1 (PD-L1) and inflammatory cytokines in specific cancer cells harboring certain EGFR mutations. Compared to some other EGFR inhibitors, lazertinib may have therapeutic benefits in tumors with T790M- or L858R-mediated drug resistance. In addition, lazertinib penetrates the blood-brain barrier (BBB). This agent shows minimal activity against wild-type EGFR (wtEGFR), and does not cause dose-limiting toxicities,

which occur during the use of non-selective EGFR inhibitors and inhibit wtEGFR. EGFR, a receptor tyrosine kinase (RTK) mutated in many tumor cell types, plays a key role in tumor cell proliferation and tumor vascularization. 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

LCAR-B38M

According to the NCI website autologous bi-epitope BCMA-targeted CAR T cells JNJ-68284528 is a preparation of autologous T lymphocytes that are transduced, ex vivo, with a lentiviral vector expressing a chimeric antigen receptor (CAR) containing two bispecific anti-B-cell maturation antigen (BCMA) variable fragments of llama heavy-chain murine antibodies fused to the signaling domain of 4-1BB (CD137), with potential immunostimulating and antineoplastic activities. The antigen-binding region of the CAR is a non-scFv structure targeting two distinct regions of BCMA. Upon intravenous administration back into the patient, the autologous bi-epitope BCMA-targeted CAR T cells JNJ-68284528 are directed to cells expressing BCMA, bind to two different epitopes on BCMA and induce selective toxicity in BCMA-expressing tumor cells. BCMA, a tumor-associated antigen (TAA) and a receptor for both a proliferation-inducing ligand (APRIL) and B-cell activating factor (BAFF), is a member of the tumor necrosis factor receptor superfamily (TNFRSF) and plays a key role in plasma cell survival. BCMA is overexpressed on malignant plasma cells. Check for active clinical trials using this agent. (NCI Thesaurus)

Link to National Cancer Institute

LCL161

According to the NCI website SMAC mimetic LCL161 is an orally bioavailable second mitochondrial-derived activator of caspases (SMAC) mimetic and inhibitor of IAP (Inhibitor of Apoptosis Protein) family of proteins, with potential antineoplastic activity. SMAC mimetic LCL161 binds to IAPs, such as X chromosome-linked IAP (XIAP) and cellular IAPs 1 and 2. Since IAPs shield cancer cells from the apoptosis process, this agent may restore and promote the induction of apoptosis through apoptotic signaling pathways in cancer cells. IAPs are overexpressed by many cancer cell types and suppress apoptosis by binding and inhibiting active caspases-3, -7 and -9, which play essential roles in apoptosis (programmed cell death), necrosis and inflammation. 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

Inhibitor of apoptosis

Lenalidomid - REVLIMID®

Lenalidomid ist ein Thalidomid-Analogon mit einer potentiellen antineoplastischen Aktivität. Lenalidomid hemmt die TNF-alpha-Produktion, stimuliert T-Zellen, reduziert den Serumspiegel der Zytokine VEGF (vaskulärer endothelialer Wachstumsfaktor) und bFGF (basischer Fibroblastenwachstumsfaktor) und hemmt die Angiogenese. Dieses Mittel fördert auch den Unterbruch des Zellzyklus in der G1-Phase und die Apoptose von Krebszellen.

Indikationen/Anwendungsmöglichkeiten gemäss Compendium®:

- Revlimid in Kombination mit Bortezomib und Dexamethason ist indiziert zur Behandlung von erwachsenen Patienten mit unbehandeltem multiplem Myelom.
- Revlimid ist indiziert zur Behandlung von erwachsenen Patienten mit multiplem Myelom als Erhaltungstherapie nach autologer Stammzelltransplantation.
- Revlimid in Kombination mit Dexamethason oder Revlimid in Kombination mit Melphalan und Prednison, jeweils gefolgt von einer Revlimid Erhaltungstherapie, ist indiziert zur Behandlung erwachsener Patienten mit unbehandeltem multiplem Myelom, die nicht transplantierbar sind.
- Revlimid in Kombination mit Dexamethason ist indiziert zur Behandlung von Patienten mit multiplem Myelom, die wenigstens eine vorangegangene medikamentöse Therapie erhalten haben.
- Revlimid ist indiziert zur Behandlung von Patienten mit transfusionsabhängiger Anämie infolge von myelodysplastischem Syndrom mit niedrigem oder intermediärem Risiko 1 in Verbindung mit einer zytogenetischen Deletion 5q-Anomalie mit oder ohne weitere zytogenetische Anomalien.
- Revlimid ist indiziert zur Behandlung von Patienten mit rezidiviertem oder refraktärem Mantelzell-Lymphom (MCL) nach vorangegangener Therapie, welche Bortezomib und Chemotherapie/Rituximab umfasste.
- Revlimid in Kombination mit Rituximab (Anti-CD20-Antikörper) ist indiziert zur Behandlung von erwachsenen Patienten mit einem rezidivierten oder refraktären follikulären Lymphom (Grad 1-3A) (siehe «Eigenschaften/Wirkungen»)

Merkblätter für Patientinnen und Patienten

Link zur Fachinformation des Compendium®z:

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Info for Patients presented by Scott Hamilton from Chemocare.com

Monoclonal Antibodies

Lenvatinib – KISPLYX®; LENVIMA®

According to the NCI website, lenvatinib mesylate is a synthetic, orally available inhibitor of vascular endothelial growth factor receptor 2 (VEGFR2, also known as KDR/FLK-1) tyrosine kinase with potential antineoplastic activity. Lenvatinib mesylate blocks VEGFR2 activation by VEGF, resulting in inhibition of the VEGF receptor signal transduction pathway, decreased vascular endothelial cell migration and proliferation, and vascular endothelial cell apoptosis. Check for active clinical trials using this agent. (NCI Thesaurus)

Indikationen/Anwendungsmöglichkeiten für Kisplyx gemäss Compendium®

- Kisplyx ist indiziert in Kombination mit Everolimus zur Behandlung von erwachsenen Patienten mit fortgeschrittenem Nierenzellkarzinom (RCC) nach einer vorhergehenden, gegen den vaskulären endothelialen Wachstumsfaktor (VEGF) gerichteten Behandlung.

Link zur Fachinformation von Compendium.ch®

Indikationen/Anwendungsmöglichkeiten für Lenvima® gemäss Compendium®

- *Differenziertes Schilddrüsenkarzinom (DTC)*

Lenvima ist indiziert für die Behandlung von Patienten mit Radiojod-refraktärem, lokal fortgeschrittenem oder metastasierendem, progredientem, differenziertem Schilddrüsenkarzinom.

- *Hepatozelluläres Karzinom (HCC)*

Lenvima ist indiziert zur Erstlinien-Therapie von Patienten mit fortgeschrittenem oder nicht-resezierbarem Leberzellkarzinom (siehe Abschnitt «Klinische Wirksamkeit»).

Link zur Fachinformation von Compendium.ch®

Merkblätter 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

Wiki

Link zu PharmaWiki
Link to Physicians Desk Reference (PDR)
Link to European Medicines Agency (EMEA):

- Human medicine European public assessment report (EPAR): Kisplyx

- Human medicine European public assessment report (EPAR): Lenvima
Info for Patients presented by Scott Hamilton from Chemocare.com

Letrozol - FEMARA® plus diverse Generika

Letrozol ist ein nicht-steroidaler Inhibitor der Östrogensynthese mit antineoplastischer Aktivität. Als Aromatasehemmer der dritten Generation wirkt Letrozol selektiv und hemmt reversibel die Aromatase, was zur Wachstumshemmung von Östrogen-abhängigen Brustkrebs-Zellen führen kann. Aromatase ist ein Cytochrom P-450 Enzym, das im endoplasmatische Retikulum der Zellen vieler Gewebe lokalisiert ist, einschließlich denen des prämenopausalen Ovars, der Leber und der Brust, katalysiert die Aromatisierung von Androstendion und Testosteron zu Östron und Östradiol, dem letzten Schritt in der Östrogen Biosynthese.

Indikationen/Anwendungsmöglichkeiten gemäss Compendium®:

- Adjuvante Behandlung von postmenopausalen Frauen mit frühem Mammakarzinom (Estrogen- oder Progesteronrezeptorstatus positiv oder unbekannt).
- Adjuvante Behandlung von postmenopausalen Frauen mit frühem Mammakarzinom (Estrogen- oder Progesteronrezeptorstatus positiv oder unbekannt), welche eine adjuvante Therapie mit Tamoxifen von 5 Jahren erhalten haben (erweiterte adjuvante Therapie).
- Behandlung des fortgeschrittenen Mammakarzinoms bei postmenopausalen Frauen mit Estrogen- oder Progesteron-positivem oder mit unbekanntem Rezeptorstatus, wobei die Postmenopause physiologisch oder nach einem künstlichen Eingriff eingetreten sein kann.

Merkblätter für Patientinnen und Patienten

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Information des Médicaments: Info prof. Info patient

Informazione sul medicamento: info per il paziente

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Link to National Cancer Institute
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Link zu PharmaWiki
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More information for patients:

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Info for Patients presented by Scott Hamilton from Chemocare.com

Leucovorin- LEUCOVORIN Teva

Leucovorin, According to the NCI website Leucovorin is an active metabolite of folic acid (also called folinic acid and citrovorum factor), which does not require metabolism by dihydrofolate reductase, the molecular target of folate antagonist-type chemotherapeutic drugs. Leucovorin calcium counteracts the toxic effects of these medications, 'rescuing' the patient while permitting the antitumor activity of the folate antagonist. This agent also potentiates the effects of fluorouracil and its derivatives by stabilizing the binding of the drug's metabolite to its target enzyme, thus prolonging drug activity.

Indikationen/Anwendungsmöglichkeiten gemäss Compendium®:

- Als spezifisches Antidot des Folsäure-Antagonisten Methotrexat zur Verminderung der hämatopoetischen Toxizität bzw. zur Aufhebung der Wirkung dieses Zytostatikums.
- Bei Behandlungen mit hohen Dosen Methotrexat ist die nachfolgende Gabe von Calciumfolinat («Rescue») unbedingt erforderlich, da das Zytostatikum bei den entsprechenden Indikationen in

toxischen Dosen verabreicht wird.
- Calciumfolinat hat sich in der Therapie des fortgeschrittenen kolorektalen Karzinoms (Stadium: Dukes C) in der Kombination mit 5-Fluorouracil (5-FU) als wirksam erwiesen.

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 zur Fachinformation des Compendium®
Info for Patients presented by Scott Hamilton from Chemocare.com

Leuprolide/Leuprorelin - ELIGARD®, LUCRIN Depot®; LEUPRORELIN Sandoz®

Eligard®, Lucrin Depot®, Leuprorelin Sandoz®; According to the NCI website Leuprolide binds to and activates gonadotropin-releasing hormone (GnRH) receptors. Continuous, prolonged administration of leuprolide in males results in pituitary GnRH receptor desensitization and inhibition of pituitary secretion of follicle stimulating hormone (FSH) and luteinizing hormone (LH), leading to a significant decline in testosterone production; in females, prolonged administration results in a decrease in estradiol production. This agent reduces testosterone production to castration levels and may inhibit androgen receptor-positive tumor progression.

Indikationen/Anwendungsmöglichkeiten gemäss Compendium®:

Eligard®:

- Prostatakarzinom: symptomatische palliative Therapie des fortgeschrittenen hormonabhängigen Prostatakarzinoms.

Lucrin Depot® (nur Indikationen bei Karzinom aufgeführt):

- *Prostatakarzinom:* Symptomatische Behandlung des fortgeschrittenen hormonabhängigen Prostatakarzinoms. Als alternative Behandlung, wenn Orchiektomie oder Östrogengaben entweder für den Patienten nicht indiziert oder nicht zumutbar sind.
- *Mammakarzinom:* Adjuvante Therapie des frühen operablen Mammakarzinoms und Therapie des fortgeschrittenen, metastasierenden Mammakarzinoms bei prämenopausalen Frauen mit Rezeptor-positiven Tumoren, bei denen eine Hormontherapie angezeigt ist.

Leuprorelin Sandoz®:

- Symptomatische Behandlung des fortgeschrittenen hormonabhängigen Prostatakarzinoms; als alternative Behandlung, wenn Orchiektomie oder Östrogengaben entweder für den Patienten nicht indiziert oder nicht zumutbar sind.

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 zur Fachinformation des Compendium®
Info for Patients presented by Scott Hamilton from Chemocare.com

Lisocabtagen Maraleucel

According to the NCI website lisocabtagene maraleucel is a preparation of a defined ratio of CD4+ and CD8+ autologous T lymphocytes transduced with a lentiviral vector expressing a chimeric antigen receptor (CAR) containing an anti-CD19 single chain variable fragment (scFv) fused to the signaling domain of 4-1BB (CD137), the zeta chain of the TCR/CD3 complex (CD3-zeta), and a truncated form of the human epidermal growth factor receptor (EGFRt), with potential immunostimulating and antineoplastic activities. Upon intravenous administration, lisocabtagene maraleucel is directed to and induce selective toxicity in CD19-expressing tumor cells. CD19 antigen is a B-cell specific cell surface antigen expressed in all B-cell lineage malignancies. Devoid of both ligand binding domains and tyrosine kinase activity, the expressed EGFRt both facilitates in vivo detection of the administered, transduced T cells and can promote elimination of those cells through a cetuximab-induced antibody dependent cellular cytotoxicity (ADCC) response. The 4-1BB costimulatory signaling domain enhances both proliferation of T cells and antitumor

activity. Check for active clinical trials using this agent. (NCI Thesaurus)

Link to National Cancer Institute

Wiki

Lomustine

Ceenu®, According to the NCI website lomustine is a nitrosourea with antineoplastic activity. Lomustine alkylates and crosslinks DNA, thereby inhibiting DNA and RNA synthesis. This agent also carbamoylates DNA and proteins, resulting in inhibition of DNA and RNA synthesis and disruption of RNA processing.

Indikationen/Anwendungsmöglichkeiten:

Palliative Behandlung allein oder in Kombination mit anderen Zytostatika von:
– Hirntumoren, Hirnmetastasen,
– Morbus Hodgkin (Lymphogranulom) Stadien IIB bis IV.
Ceenu allein oder in Kombination mit anderen Zytostatika kann auch zur palliativen Behandlung von:
– Bronchialkarzinomen (metastasierend und/oder inoperabel),
– Non-Hodgkin-Lymphom Stadien II bis IV,
– malignem Melanom,
– Nierenkarzinomen (metastasierend und/oder inoperabel),
– gastrointestinalen Tumoren (metastasierend und/oder inoperabel),
angewendet werden.

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 to Wikipedia

Link zu PharmaWiki

Link to Physicians Desk Reference (PDR)

Im Arzneimittel-Kompendium der Schweiz® nicht verzeichnet

Info for Patients presented by Scott Hamilton from Chemocare.com

Alkylating Agentsv

Loncastuximab Tesirine

According to the NCI website loncastuximab tesirine is an antibody-drug conjugate (ADC) consisting of an anti-CD19 humanized monoclonal antibody conjugated, via a cleavable linker comprised of valine-alanine and maleimide, to a cytotoxic, cross-linking agent pyrrolobenzodiazepine (PBD) dimer, which targets DNA minor grooves, with potential antineoplastic activity. Upon administration, the monoclonal antibody portion of loncastuximab tesirine targets the cell surface antigen CD19 on various cancer cells. Upon antibody/antigen binding and internalization, the cytotoxic PBD moiety is released. The imine groups of the PBD moiety bind to the N2 positions of guanines on opposite strands of DNA. This induces interstrand cross-links in the minor groove of DNA and inhibits DNA replication, which inhibits the proliferation of CD19-overexpressing tumor cells. CD19, a transmembrane receptor and tumor-associated antigen (TAA), is expressed on a number of B-cell-derived cancers. 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

Wiki

Lorlatinib - LORVIQUA®

According to the NCI website, lorlatinib is an orally available, ATP-competitive inhibitor of the receptor tyrosine kinases, anaplastic lymphoma kinase (ALK) and C-ros oncogene 1 (Ros1), with potential antineoplastic activity. Upon administration, lorlatinib binds to and inhibits both ALK and ROS1 kinases. The kinase inhibition leads to disruption of ALK- and ROS1-mediated signaling and eventually inhibits tumor cell growth in ALK- and ROS1-overexpressing tumor cells. In addition, lorlatinib is able to cross the blood brain barrier. ALK belongs to the insulin receptor superfamily and plays an important role in nervous system development; ALK dysregulation and gene rearrangements are associated with a series of tumors. ROS1, overexpressed in certain cancer cells, plays a key role in cell growth and survival of cancer cells. Check for active clinical trials using this agent. (NCI Thesaurus)

Indikationen/Anwendungsmöglichkeiten gemäss Compendium®:

- Lorviqua kann angewendet werden zur Behandlung von erwachsenen Patienten mit Anaplastische-Lymphom-Kinase (ALK)-positivem, metastasiertem nicht kleinzelligem Lungenkarzinom (non small cell lung cancer, NSCLC) nach Progression unter vorangegangener Behandlung mit mindestens zwei ALK-Tyrosinkinaseinhibitoren (TKI).

Merkblätter für Patientinnen und Patienten **(Stand 6.4.20: noch nicht erhältlich)**

Link zur Fachinformation des Compendium®:

Medikamenteniformation: Für den Arzt Patienteninformation

More information in English:

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Info for Patients presented by Scott Hamilton from Chemocare.com

Loxo-305

According to the NCI website BTK inhibitor LOXO-305 is an orally available, selective, non-covalent Bruton's tyrosine kinase (BTK) inhibitor with potential antineoplastic activity. Upon oral administration, BTK inhibitor LOXO-305 selectively and reversibly binds to BTK. This prevents both the activation of the B-cell antigen receptor (BCR) signaling pathway and BTK-mediated activation of downstream survival pathways, thereby inhibiting the growth of malignant B cells that overexpress BTK. Reversible binding of LOXO-305 may preserve antitumor activity in the presence of certain acquired resistance mutations, including C481 mutated BTK, and limit toxicity associated with inhibition of other non-BTK kinases. BTK, a member of the Src-related BTK/Tec family of cytoplasmic tyrosine kinases, is overexpressed or mutated in B-cell malignancies; it plays an important role in the development, activation, signaling, proliferation and survival of B lymphocytes. Check for active clinical trials using this agent. (NCI Thesaurus)

Link to National Cancer Institute

BTK inhibitor

Lumretuzumab

Lumretuzumab is a humanized and glycoengineered anti-HER3 monoclonal antibody.

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

Conclusions Combination treatment with lumretuzumab, pertuzumab and paclitaxel was associated with a high incidence of diarrhea. Despite the efforts to alter dosing, the therapeutic window remained too narrow to warrant further clinical development. (see paper)

Lurbinectedin

According to the NCI website lurbinectedin is a synthetic tetrahydropyrrolo [4, 3, 2-de]quinolin-8(1H)-one alkaloid analogue with potential antineoplastic activity. Lurbinectedin covalently binds to residues lying in the minor groove of DNA, which may result in delayed progression through S phase, cell cycle arrest in the G2/M phase and cell death. Check for active clinical trials using this agent. (NCI Thesaurus)

According to WIKI in "Small-cell carcinoma"

Lurbinectedin demonstrated an overall response rate (ORR) of 35.2% in relapsed small cell lung cancer trial. Overall survival rate for patients with sensitive disease (chemotherapy-free interval of 90 days or longer) was 15.2 months, and 5.1 months for patients with resistant disease (chemotherapy-free interval of less than 90 days). [47] Lurbinectedin is currently available in the U.S. under Expanded Access Program (EAP).[48]

Summary of the following 2 studies**:**

Trigo J, et al "Lurbinectedin as a second-line treatment for patients with small-cell lung cancer: A single-arm, open-label, phase 2 basket trial" *Lancet Oncol* 2020; 21: 645-54.

Arrieta O, et al "New opportunities in a challenging disease: Lurbinectedin for relapsed small-cell lung cancer" *Lancet Oncol* 2020; 21: 605-607.

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

Luspatercept – REBLOZYL® (FDA orphan drug status in 2013 and fast track designation in 2015)

According to the NCI website Luspatercept is a soluble, recombinant fusion protein composed of a modified form of the extracellular domain of human activin receptor type IIb (ActRIIb) and linked to the human IgG1 Fc domain, with red blood cell stimulating activity. Upon subcutaneous administration, luspatercept inhibits several ligands in the transforming growth factor (TGF)-beta superfamily. This prevents activation of a variety of TGF-beta superfamily members involved in late stage erythropoiesis and results in an increased differentiation and proliferation of erythroid progenitors. Luspatercept acts at a different, later stage than erythropoietin. This agent ultimately enhances red blood cell production and prevents anemia. Check for active clinical trials using this agent. (NCI Thesaurus)

According to WIKI Luspatercept is a drug for the treatment of anemia in beta thalassemia and myelodysplastic syndromes.

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Link to National Cancer Institute

Wiki

Link to European Medicines Agency (EMEA)