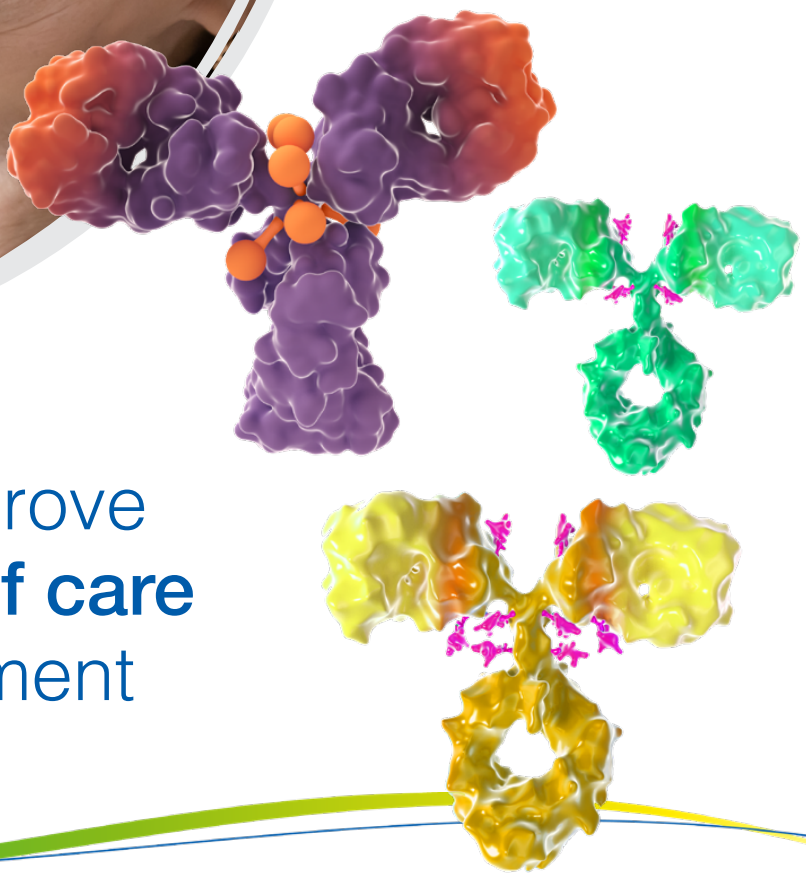


We seek to improve **standards of care** in cancer treatment

...because we focus our scientific and research efforts on areas of unmet medical need

WE ARE DAIICHI SANKYO

Daiichi-Sankyo

Passion for Innovation.
Compassion for Patients.™

Our purpose

To contribute to the enrichment of quality of life around the world.

Our mission

To create innovative pharmaceuticals addressing diverse medical needs.

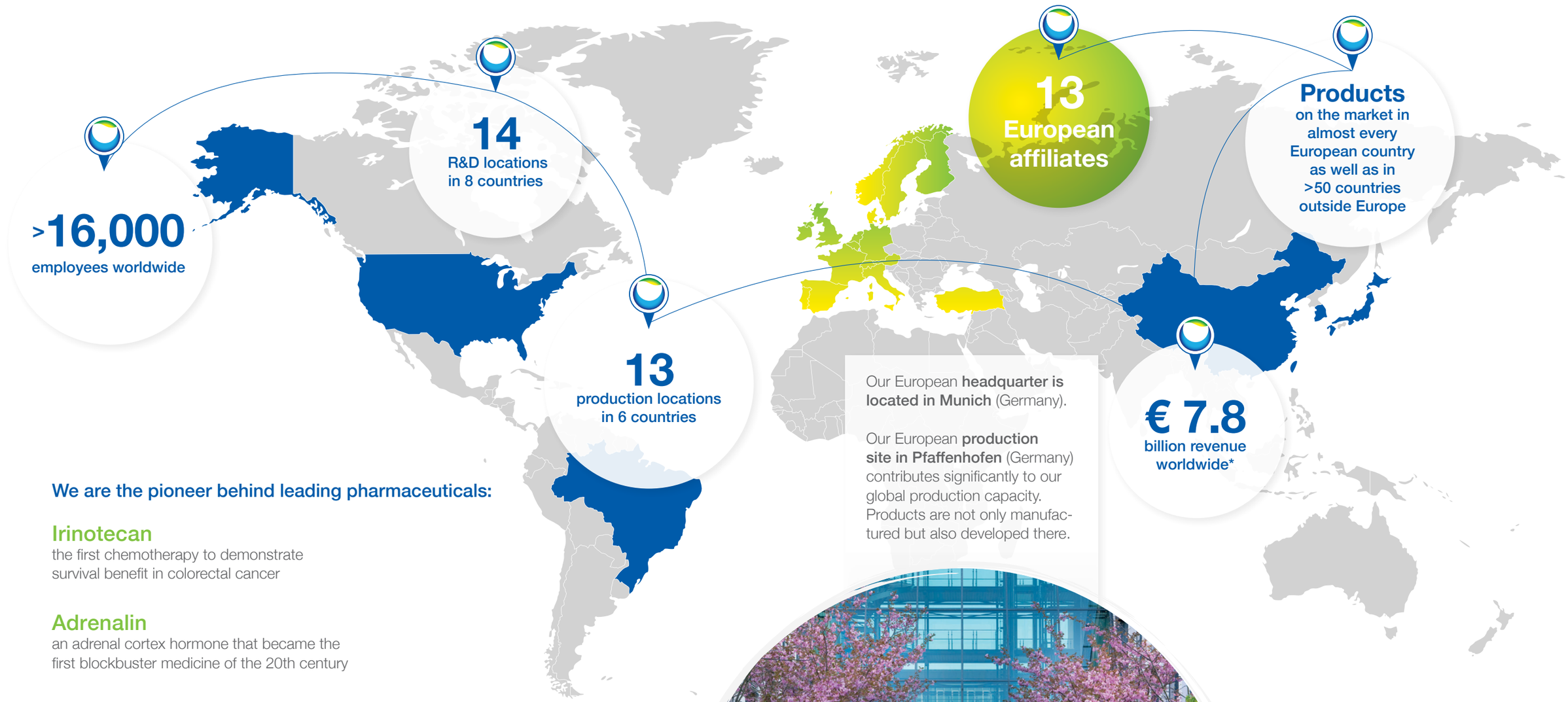
Our vision

To become an innovative global healthcare company contributing to the sustainable development of society.

WE ARE DAIICHI SANKYO

Daiichi-Sankyo

We are a global pharmaceutical company with over 120 years of scientific expertise



Our European **headquarter is located in Munich** (Germany).

Our European **production site in Pfaffenhofen** (Germany) contributes significantly to our global production capacity. Products are not only manufactured but also developed there.

We are the pioneer behind leading pharmaceuticals:

Irinotecan
the first chemotherapy to demonstrate survival benefit in colorectal cancer

Adrenalin
an adrenal cortex hormone that became the first blockbuster medicine of the 20th century

Pravastatin
a globally groundbreaking anti-hyperlipidemic agent

Edoxaban
an anticoagulant and direct factor Xa inhibitor, that helps reduce the risk of stroke and thromboembolism

* as of April 2021 R&D: Research & Development

Daiichi-Sankyo

We have more than 40 years of experience in bringing cancer therapies to patients

1977
Launched Krestin (polysaccharide-K) in Japan to treat gastrointestinal, lung and breast cancer

1985
Launched the first natural-type interferon beta preparations for brain tumour and skin cancer in Japan

1995
Received approval for irinotecan, the first chemotherapy to demonstrate survival benefits in colorectal cancer

2012
Launched RANMARK® (denosumab) in Japan for treatment of bone complications of multiple myeloma and bone metastases of solid tumours

2020
>20 compounds in development directed at a range of diseases with unmet medical needs, with a focus on ADC technology

2021
In January 2021, ENHERTU® (trastuzumab deruxtecan) received EMA approval for HER2-positive metastatic breast cancer in Europe

* Enhertu as monotherapy is indicated for the treatment of adult patients with unresectable or metastatic HER2-positive breast cancer who have received two or more prior anti-HER2-based regimens SmPC at www.ema.europa.eu

ADC: Antibody Drug Conjugate
EMA: European Medicines Agency
HER2: Human Epidermal growth factor Receptor 2

Daiichi-Sankyo

We have a passion for true innovation

Our oncology portfolio and research focus on innovation in areas of unmet medical need

Research & Development

R&D investment
>€ 11 billion
2021 - 2026*

R&D expenses of yearly revenue**
>20%
€ 1.84 billion

We put the patient at the heart of everything we do

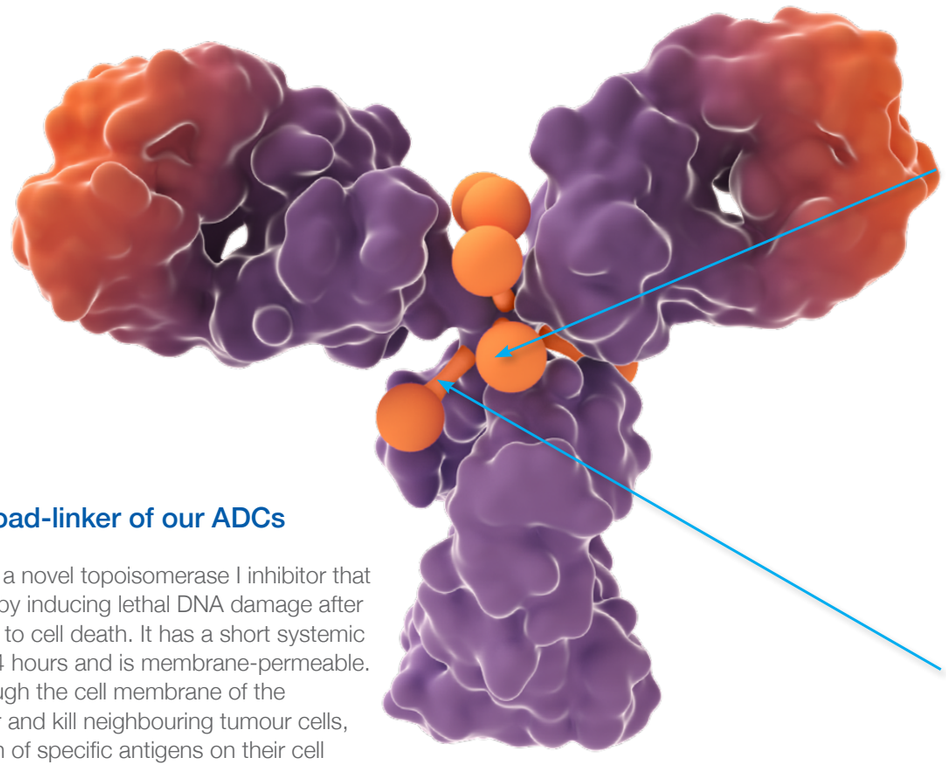
* https://www.daiichisankyo.com/files/investors/library/materials/2021/20210405_5th_MTP_E.pdf ** as of April 2021
R&D: Research & Development

Daiichi-Sankyo

Our strength: science & technology

DXd ADCs are composed of 3 components[1,6]:

- A monoclonal antibody attached to:
- A topoisomerase I inhibitor payload (an exatecan derivative) via
- A tetrapeptide-based cleavable linker



Deruxtecan, the payload-linker of our ADCs

The payload in our ADCs is a novel topoisomerase I inhibitor that exhibits potent cytotoxicity by inducing lethal DNA damage after intracellular release, leading to cell death. It has a short systemic half-life of approximately 1.4 hours and is membrane-permeable. Therefore, it can travel through the cell membrane of the target tumour cell and enter and kill neighbouring tumour cells, regardless of the expression of specific antigens on their cell surface. This dual action on both target and surrounding cancer cells, which is known as the bystander antitumour effect, is of great importance in molecularly heterogenous tumours.

Our DXd ADCs are designed with 7 key attributes

Our scientists have specifically engineered our ADCs to address limitations of two critical components of an ADC: the payload and the linker. Therefore, our ADCs will most effectively deliver their cytotoxic payload inside cancer cells while limiting exposure of healthy tissues to the payload.

Characteristics of Payload

Mode of action payload
DXd, an investigational topoisomerase I inhibitor [2,3]

High potency of payload
~10x more potent topoisomerase I inhibition than SN-38, based on a cell-free assay evaluating HIC [3,6]

Bystander antitumour effect
Elimination of both target tumour cells and surrounding tumour cells enabled by the high cell-membrane permeability of the payload. [4,6]

Low systemic exposure
Short systemic half-life of payload [5]

Characteristics of Linker

Stable linker
In vitro plasma stability, designed to limit exposure of non-cancerous normal tissue to the payload [5]

Linker release
Cleaved (divided) by lysosomal enzymes [3]

High drug-to-antibody ratio (DAR)
High number of payload entities per antibody molecule with flexibility to vary DAR during drug design. Can achieve a DAR of up to 8. [3,5]

ADC: Antibody Drug Conjugate
DAR: Drug-to-antibody ratio
Deruxtecan: linker + payload
DXd: a topoisomerase I inhibitor (payload)
HIC: Hydrophobic Interaction Chromatography

1, Okajima D, et al. Poster presented at: AACR-NCI-EORTC International Conference; October 26-30, 2019; Boston, MA [abstract C026].
2, Beck A, et al. Nat Rev Drug Discov. 2017;16:315-337.
3, Ogitani Y, et al. Clin Cancer Res. 2016;22(20):5097-5108.
4, Ogitani Y, et al. Cancer Science. 2016;107(7):1039-1046.
5, Daiichi Sankyo Data on File.
6, ENHERTU® SmPC 03/2021, www.ema.europa.eu

Daiichi-Sankyo

Our new treatment options for patients with cancer

We have applied our ADC technology to different antibodies to create a series of novel medicines – each targeting a specific antigen on the surface of cancer cells

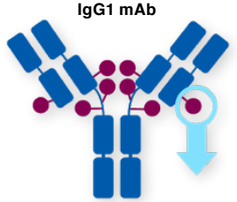
ADC: Antibody Drug Conjugate
DAR: Drug-to-antibody ratio
Dato-DXd: datopotamab deruxtecan
Deruxtecan: linker + payload
DXd: a topoisomerase I inhibitor (payload)
EGFR: Epidermal Growth Factor Receptor
ER+: Estrogene Receptor positive
HER3-DXd: patritumab deruxtecan
IgG1: Immunoglobulin G1
mAb: monoclonal Antibody
NSCLC: Non-Small Cell Lung Carcinoma
T-DXd: trastuzumab deruxtecan
TNBC: Triple-Negative Breast Cancer
anti-HER2: anti-Human Epidermal growth factor Receptor 2
anti-HER3: anti-Human Epidermal growth factor Receptor 3
anti-TROP2: anti-TROPhoblast cell-surface antigen 2

Our DXd ADC portfolio currently features 3 lead ADCs

Our ADC technology is designed to provide enhanced cancer cell elimination upon release of the cytotoxic payload inside the cell to reduce systemic exposure to the payload compared to conventional chemotherapy

T-DXd
DS-8201
Trastuzumab deruxtecan

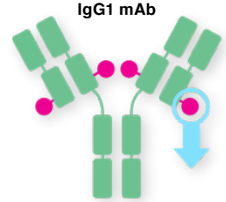
Humanized anti-HER2 IgG1 mAb



DAR ≈8

Dato-DXd
DS-1062
Datopotamab deruxtecan

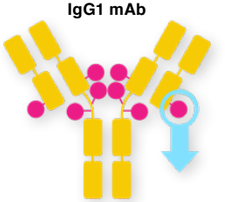
Humanized anti-TROP2 IgG1 mAb



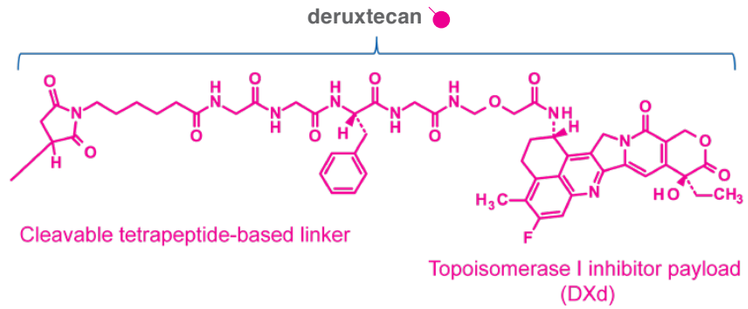
DAR ≈4

HER3-DXd
U3-1402
Patritumab deruxtecan

Human anti-HER3 IgG1 mAb



DAR ≈8



ADC	TARGET
DS-8201	**HER2** (human epidermal growth factor receptor 2) is a growth-promoting tyrosine kinase receptor expressed on the cell surface of many tumour types, including breast, gastric and lung cancer.[1,4]
DS-1062	**TROP2** (trophoblast cell-surface antigen 2) is a transmembrane glycoprotein that is highly expressed by several types of adenocarcinomas, including NSCLC and TNBC.[3,6]
U3-1402	**HER3** (human epidermal growth factor receptor 3) is a member of the EGFR family of receptor tyrosine kinases. HER3 is overexpressed in lung adenocarcinomas, ER+ breast cancer (among other cancers).[2,5]

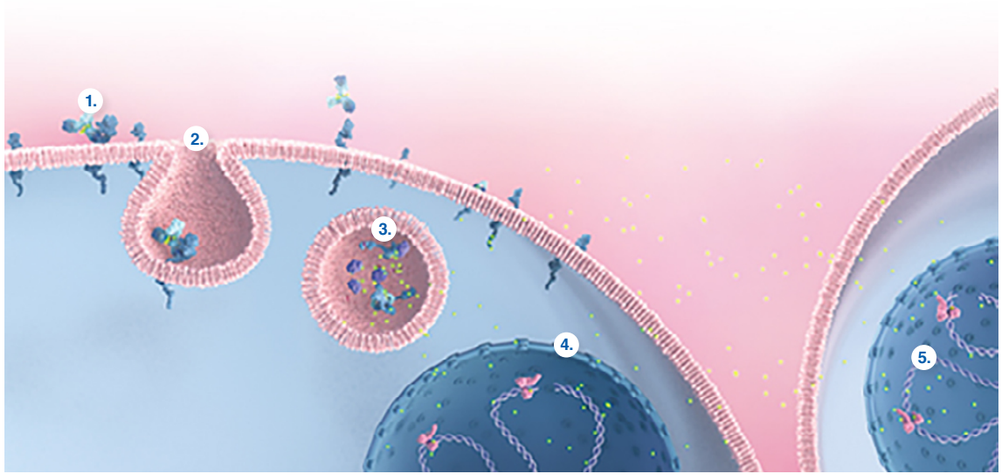
1, Iqbal N, et al. Mol Biol Int. 2014;852748.
2, Mishra R, et al. Oncol Rev. 2018;12(1):355.
3, Son S, et al. Int J Biol Macromol. 2018;110:406–415.
4, Hirsch FR, et al. Lancet 2017;389:299-311.
5, Kumagai T, et al. Thorac Cancer. 2018;9(4):466-471
6, Inamura K, et al. Oncotarget. 2017; 8(17):28725-28735

Daiichi-Sankyo

Mode of action of ADCs

Our ADCs have a cytotoxic effect on cancer cells that express a specific cell surface antigen and on adjacent cancer cells without that cell surface antigen

The proposed mode of action of our ADCs is depicted in detail below



After the ADC has been administered, the stability of the ADC complex in the circulation helps to minimise systemic release of the drug payload and promotes delivery of the intact ADC to the target cancer cells.

1. Once the ADC arrives at the target cancer cell, the monoclonal antibody moiety attaches with high specificity to its target antigen (receptor) on the cell surface.

2. The ADC-receptor complex is subsequently internalised by the cancer cell by means of endocytosis.

3. After internalisation, the linker is cleaved by lysosomal enzymes, breaking down the ADC complex and releasing free payload inside the cancer cell.

4. Free payload then travels to the cell nucleus, causing irreparable DNA damage leading to cell death.

5. Owing to its high membrane permeability, free payload can diffuse into adjacent cancer cells and kill these cells as well, even if they do not have the target antigen. This effect is known as the bystander antitumour effect of the payload.

ADC: Antibody Drug Conjugate
DNA: Desoxyribonucleic acid

Interview with Prof. Fabrice André

Gustave Roussy Cancer Campus
Villejuif, France

Why are antibodies drug conjugates (ADCs) emerging so strongly in oncology?
The evolution of ADCs in recent years is quite incredible. It is offering our patients better chances. When we compare the different generations of ADCs, we realize that the new generations of ADCs provide new clinical options to patients who were resistant to the first generation of conjugated antibodies. And this is only the beginning!

What are the strengths of ADCs?
The strength of ADCs is based on three principles: less toxicity, better efficacy and the perspective of new therapeutic combinations. I believe that reducing the systemic toxicity of chemotherapy is a major step forward in patient management. Bioengineering has made it possible to create ADCs with an important DAR (Drug Antibody Ratio) and therefore higher efficacy. These antibodies carry and deliver many more chemotherapeutic molecules directly into the tumour cell, without damaging healthy tissues. The targeted action of these cytotoxic agents spares the patient's immune system, and this opens up wonderful prospects for new therapeutic combinations. Indeed, until today it was often difficult, if not impossible, to combine certain treatments because of the risk of causing too much toxicity. With ADCs, it will potentially be easier to use the synergy of certain therapies to treat patients whose treatment failed.

How does the payload influence the ADC's final mechanism of action?
The final mechanism of action depends on the chemotherapeutic payload conjugated with the monoclonal antibody. After administration of the ADC, the payload will be carried in a conjugated form to the target tumour cell. There, the ADC will bind to the target membrane receptor, followed by internalization in endosomes and, after that, transfer to lysosomes. Through the combined effect of pH and certain enzymes, such as proteases, the payload will be released and will regain its activity either by acting on microtubules or by blocking topoisomerase. The cytotoxic agent alone determines the mode of action at the cellular level. With ADCs, we find the same variety of mechanisms as with the so-called conventional chemotherapy.

What is the issue with the drug-antibody ratio (DAR)?
As you know, the payload-antibody ratio represents the amount of chemotherapy the monoclonal antibody can provide to the cell. Theoretically, the higher the DAR, the more effective the ADC will be, provided that the chemotherapeutic agent will be released well.

The issue, therefore, is to put a large number of chemotherapeutic molecules on a single antibody molecule. But one should be aware that there are factors that limit the release of chemotherapeutics. It is not known how this phenomenon occurs. For a high DAR to be a real asset, we will have to discover the precise mechanisms that promote the release of cytotoxic agents in the tumor cell and those that prevent their release. We hope that the fundamental research underway will soon provide these answers.

Is the bystander effect an asset?
The bystander effect is the effect that cells that have not absorbed an ADC will still be destroyed by a collateral effect. So that's an asset. But this raises a major question: what is the mechanism of action that is responsible for the bystander effect? The mechanism of action is assumed to be that the cell which internalized the ADC, releases its chemotherapeutic payload to neighboring cells even if the latter do not have the surface receptors corresponding to the ADC.

What is interesting about the bystander effect is that it could remove bias from the minimal number of receptors needed to internalize an ADC or facilitate the action of the ADC in the case of heterogeneous tumours.

What do you think the future of ADCs will be?
I think the ADCs will completely replace conventional chemotherapy. This will result in less systemic toxicity, less toxicity to the healthy tissues, and better efficacy. And it will be possible to make therapeutic combinations that are still impossible at this moment.

However, one key element remains to be defined: the minimum number of membrane receptors needed to internalize an ADC. When this data will be known, it will certainly open up new therapeutic perspectives.

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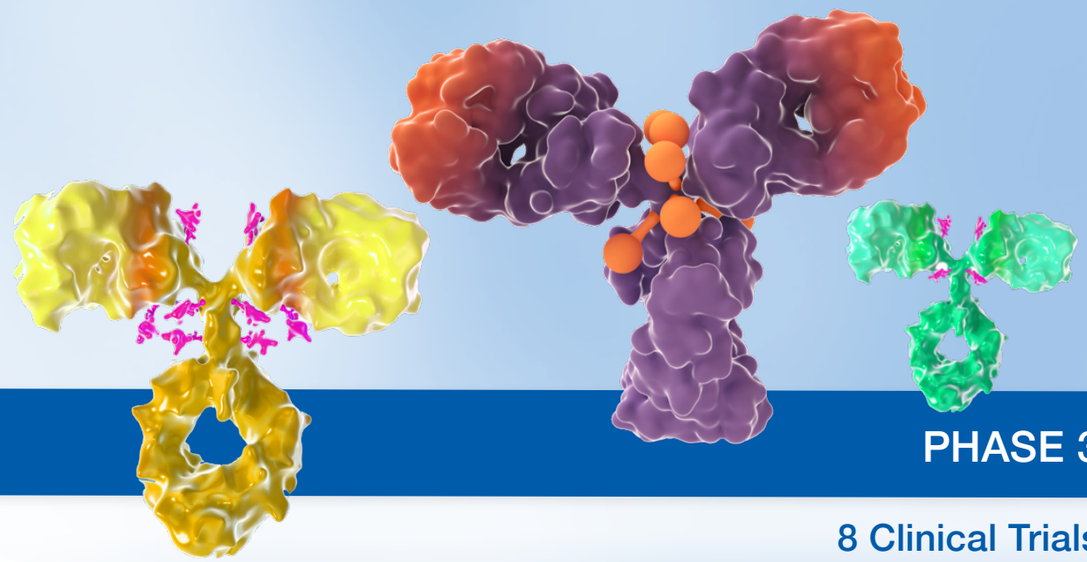
Clinical Development Program*

Phase 1-3 studies of our 3 lead ADCs



PHASE 3

8 Clinical Trials

DESTINY-Breast02, DESTINY-Breast03, DESTINY-Breast04, DESTINY-Breast05, DESTINY-Breast06, DESTINY-Breast09, DESTINY-Gastric04, TROPION-Lung01

PHASE 2

12 Clinical Trials

DESTINY-Lung01, DESTINY-Lung02, DESTINY-CRC01, DESTINY-CRC02, DESTINY-Gastric01, DESTINY-Gastric02, DESTINY-PanTumor01, DESTINY-PanTumor02, Hudson, HERTHENA-Lung01, U31402-A-U202, TROPION-Lung05

PHASE 1 & PHASE 1/2

13 Clinical Trials

TROP2

HER2

HER3

*The following pages reflect investigational compounds and/or investigational uses of approved products. The safety and efficacy of these investigational agents or investigational uses of approved products have not been established. Any approved products should be used in accordance with their product labeling (or Prescribing Information). This Oncology Pipeline Information is up to date as of April 2021. Molecules listed as compound (target/pathway). Regions are divided into 4 geographic areas: Europe, United States, Asia, and Japan. The study locations provided are not all inclusive. For a complete list, please refer to clinicaltrials.gov or clinicaltrials.jp and search for information on clinical trials sponsored by Daiichi Sankyo. Alternatively, you may also visit our pipeline chart at https://www.daiichisankyo.com/rd/pipeline. Enhertu as monotherapy is indicated for the treatment of adult patients with unresectable or metastatic HER2-positive breast cancer who have received two or more prior anti-HER2-based regimens In Europe, United States and Japan.

ADC: Antibody Drug Conjugate
HER2: Human Epidermal growth factor Receptor 2
HER3: Human Epidermal growth factor Receptor 3
TROP2: TROPhoblast cell-surface antigen 2

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Breast cancer*

Breast cancer cells may have different receptors (antigens) on their surface, such as HER2, HER3, TROP2

HER2 amplification or overexpression in breast cancer is estimated at

15-30%[1]

HER3 is mutated in up to

14%

of metastatic ER+ breast cancers[2]

TROP2 overexpression in triple-negative breast cancer is estimated at up to

80%[3]

- **Trastuzumab deruxtecan (T-DXd, DS-8201)*** (HER2)
  - Phase 1 (US, EU) — DS8201-A-U105
  - Phase 1 (US, EU) — DS8201-A-U106
  - Phase 1 (US, Asia) — DESTINY-Breast08
  - Phase 1/2 (US, EU, Asia) — BEGONIA
  - Phase 1b/2 (US, EU, Asia) — DESTINY-Breast07
  - Phase 2 (Asia) — DESTINY-Breast01
  - Phase 3 (US, EU, Japan, Asia) — DESTINY-Breast02
  - Phase 3 (US, EU, Japan, Asia) — DESTINY-Breast03
  - Phase 3 (US, EU, Japan, Asia) — DESTINY-Breast04
  - Phase 3 (US, EU, Japan, Asia) — DESTINY-Breast05
  - Phase 3 (US, EU, Asia) — DESTINY-Breast06
  - Phase 3 (EU, Asia) — DESTINY-Breast09
- **Datopotamab deruxtecan (Dato-DXd)** (TROP2)
  - Phase 1 (US, Japan) — TROPION-PanTumor01
  - Phase 1/2 (US, EU, Asia) — BEGONIA
- **Patritumab deruxtecan (HER3-DXd; U3-1402)** (HER3)
  - Phase 1/2 (US, Japan) — U31402-A-J101

*Enhertu as monotherapy is indicated for the treatment of adult patients with unresectable or metastatic HER2-positive breast cancer who have received two or more prior anti-HER2-based regimens In Europe, United States and Japan.

ER+: Estrogene Receptor positive
EU: Europe
HER2: Human Epidermal growth factor Receptor 2
HER3: Human Epidermal growth factor Receptor 3
TROP2: TROPhoblast cell-surface antigen 2
US: United States

Gastric cancer*

Gastric cancer cells may have different HER2 receptors (antigens) on their surface

12-20%

of gastric adenocarcimonas are **HER2** positive (by gene amplification or protein over-expressing or both)[5]

- **Trastuzumab deruxtecan (T-DXd, DS-8201)*** (HER2)
  - Phase 1b/2 (US, EU, Asia) — DESTINY-Gastric03
  - Phase 2 (Asia) — DESTINY-Gastric01
  - Phase 2 (US, EU) — DESTINY-Gastric02
  - Phase 3 — DESTINY-Gastric04

*Enhertu as monotherapy is indicated for the treatment of adult patients with unresectable or metastatic HER2-positive breast cancer who have received two or more prior anti-HER2-based regimens In Europe, United States and Japan.

1, Iqbal N, et al. Mol Biol Int. 2014;852748. 2, Mishra R, et al Oncol Rev. 2018;12(1):355. 3, Son S, et al. Int J Biol Macromol. 2018;110:406–415.
4., https://www.cancer.org/cancer/stomach-cancer/about/key-statistics.html. 5, Van Cutsem E, et al. Lancet. 2016;388:2654-2664.

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Lung cancer*

NSCLC cells may have different receptors (antigens) on their surface, such as HER2, HER3, TROP2

HER2 mutations are estimated at **2%**[1] in NSCLC

TROP2 is highly expressed in lung adenocarcinomas[2]

High **TROP2** expression is associated with poor prognosis[3]

HER3 is expressed in **> 80%** of lung adenocarcinomas[4]

- **Trastuzumab deruxtecan (T-DXd, DS-8201)*** (HER2)
  - Phase 1 (US, EU) — DS8201-A-U106
  - Phase 1 (US, EU, Asia) — DESTINY-Lung03
  - Phase 2 (US, EU, Asia) — HUDSON
  - Phase 2 (US, EU, Japan) — DESTINY-Lung01
  - Phase 2 (US, EU, Japan) — DESTINY-Lung02
- **Datopotamab deruxtecan (Dato-DXd)** (TROP2)
  - Phase 1 (US, Japan) — TROPION-PanTumor01
  - Phase 1 (US, Japan) — TROPION-Lung02
  - Phase 1 (US, Japan) — TROPION-Lung04
  - Phase 2 (US, EU, Japan, Asia) — TROPION-Lung05
  - Phase 3 (US, EU, Japan, Asia) — TROPION-Lung01
- **Patritumab deruxtecan (HER3-DXd; U3-1402)** (HER3)
  - Phase 1 (US, EU, Japan, Asia) — U31402-A-U102
  - Phase 1 (US, Japan) — U31402-A-U103
  - Phase 2 (US, EU, Japan, Asia) — HERTHENA-Lung01

*Enhertu as monotherapy is indicated for the treatment of adult patients with unresectable or metastatic HER2-positive breast cancer who have received two or more prior anti-HER2-based regimens In Europe, United States and Japan.

1, Hirsch FR, et al. Lancet 2017; 389:299-311. 2, Inamura K, et al. Oncotarget. 2017(8):28725-28735.
3, Li Z, et al. Biochem Biophys Res Commun. 2016;470:197-204. 4, Kumagai T, et al. Thoracic Cancer 2018; 9, 466–471

ASCO: American Society of Clinical Oncology
DAR: DAR: Drug-to-Antibody Ratio
DXd: a topoisomerase I inhibitor (payload)
EU: Europe
HER2: Human Epidermal growth factor Receptor 2
HER3: Human Epidermal growth factor Receptor 3
mAb: monoclonal Antibody
TROP2: TROPhoblast cell-surface antigen 2
US: United States
WCLC: World Conference on Lung Cancer

Interview with Prof. Egbert Smit

Antoni van Leeuwenhoek Cancer Institute
Amsterdam, The Netherlands

Which new biomarkers are emerging in lung cancer?
In NSCLC, three new biomarkers are currently of particular interest: HER2, HER3 and TROP2. HER2 is a well-known biomarker and drug target in e.g., breast cancer. It has emerged as an interesting biomarker in lung cancer as well. In NSCLC, HER2 overexpression, gene mutation and gene amplification may occur. HER3 is a biomarker that is frequently overexpressed in EGFR mutation-positive NSCLC patients who have developed resistance to first- and second-generation EGFR inhibitors. TROP2 is highly expressed in many solid tumours, including NSCLC.

What is the pathophysiological significance of these three biomarkers in lung cancer?
All three biomarkers are associated with a poorer prognosis. We need to distinguish between overexpression of the biomarker protein and mutations of the biomarker gene, as diagnosed by immunohistochemistry and next-generation sequencing, respectively. Apart from being prognostic biomarkers, HER2, HER3 and TROP2 may also serve as cell surface targets for antibody drug conjugates (ADCs) whose antibody moieties are directed against these antigens.

What about biomarker overexpression and mutation?
For HER2, both gene mutation and overexpression have been documented in lung cancer, while for HER3 and TROP2 overexpression is the predominant phenomenon. HER2 mutations in lung cancer are quite relevant for the sensitivity to ADCs. In case of overexpression of a biomarker, the biomarker on the cell surface may transport the ADC with its chemotherapeutic payload into the cell.
In the case of HER2, mutations seem more important for the sensitivity to ADCs than overexpression or gene amplification. HER2-mutated tumour cells are addicted to HER2 signaling, so here the antibody component of the ADC can also block the oncogenic signaling. HER2 mutations are present in 2-4% of patients with NSCLC.

For HER3 and TROP2 overexpression of the biomarker is the only determinant of sensitivity to ADCs. The activity of the HER3-directed ADC U3-1402 in NSCLC seems to be independent from tumour resistance status.

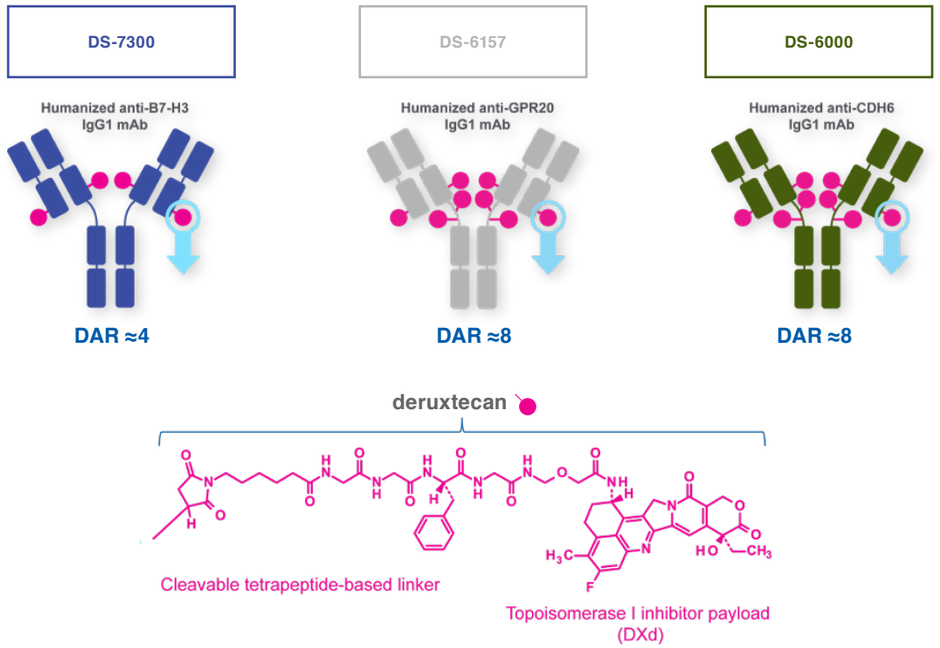
What is the development status of antibody-drug conjugates directed at HER2, HER3, or TROP2?
Three novel ADCs, targeting HER2, HER3 and TROP2, respectively, are being evaluated clinically in third-line treatment of NSCLC. Promising interim results from a phase 2 trial in HER2-mutated and HER2-overexpressing NSCLC with trastuzumab deruxtecan, a HER2-directed ADC, have been presented by Smit et al. at ASCO 2020 (J Clin Oncol. 38, no. 15_suppl (May 20, 2020) 9504-9504) and WCLC 2020 (abstract MA11.03) and by Nakagawa et al. at WCLC 2020 (abstract OA04.05). Clinically meaningful and durable responses were reported in both cohorts, however with a higher response rate in the HER2-mutated cohort. A phase 1 study of the TROP2-directed ADC datopotamab deruxtecan has just been completed. Promising clinical activity of this ADC in advanced NSCLC has also been reported at WCLC 2020 (Spira A, et al. abstract OA03.03). Phase 2 and/or 3 studies with all three ADCs have been initiated.

Overall, what are your expectations regarding HER2, HER3 and TROP2 in lung cancer?
I expect that the novel ADCs targeting these biomarkers will become valuable additions to our treatments for NSCLC. I see these ADCs as a kind of ‘targeted chemotherapy’ with far less toxicity than conventional chemotherapy. An important feature of these medications is that their topoisomerase I inhibitor payload is tightly linked to the antibody protein via a tetrapeptide-based linker, resulting in little release of the cytotoxic payload in plasma, which may contribute to the low level of off-target toxicity and the acceptable general toxicity profile.

Daiichi-Sankyo

Looking beyond 2025

Our future DXd ADC portfolio features 3 additional ADCs directed at new promising molecular targets



ADC	TARGET
DS-7300	**B7-H3** (a transmembrane protein, member of the B7 ligand family) is frequently overexpressed in various tumour types and has been associated with disease progression and poor prognosis in many tumour types.[1]
DS-6157	**GPR20** (G protein- coupled receptor 20) is a seven-pass transmembrane protein, which is selectively and abundantly expressed in GIST. GPR20 expression is detected in more than 80% of 139 GIST tumour samples irrespective of the number of prior lines of TKI treatments received.[2]
DS-6000	**CDH6** (a cadherin-family protein) is overexpressed by several cancers, in particular kidney, ovarian cancer and thyroid cancers. CDH6 overexpression has been correlated with poor prognosis in renal cell carcinoma.[3,4]

*Enhertu as monotherapy is indicated for the treatment of adult patients with unresectable or metastatic HER2-positive breast cancer who have received two or more prior anti-HER2-based regimens In Europe, United States and Japan.

1, Kontos F, et al. Clinical Cancer Research 2021; 27: 1227-35. 2, Lida K, et al. Cancer Discov 2021 Published Online DOI: 10.1158/2159-8290.CD-20-1434 3, Bialucha CU, et al. Cancer Discov 2017;(9): 1–16. 4, Stassar MJJG, et al. British Journal of Cancer 2001; 85(9), 1372–1382.

ADC: Antibody Drug Conjugate
B7-H3: Human B7 homolog 3
CDH6: Cadherin 6
GPR20: G protein- coupled receptor 20
HER2: Human Epidermal growth factor Receptor 2
HER3: Human Epidermal growth factor Receptor 3
IgG1: Immunoglobulin G1
NSCLC: Non Small Cell Lung Cancer
TKI: Tyrosine Kinase Inhibitor
TROP2: TROPhoblast cell-surface antigen 2

Enhanced capabilities through collaboration

Some of our strategic research partnerships

AstraZeneca

2020 A clinical trial collaboration
to evaluate the combination of patritumab deruxtecan (U3-1402), a HER3-directed DXd ADC, and osimertinib, an epidermal growth factor receptor EGFR-TKI, in patients with EGFR-mutated advanced or metastatic non-small cell lung cancer.

2020 A global development and commercialisation collaboration
for Daiichi Sankyo's DS-1062, a TROP2-directed DXd ADC in clinical development for NSCLC and triple-negative breast cancer, except in Japan where Daiichi Sankyo maintains exclusive rights.

2019 A global development and commercialisation collaboration
for Daiichi Sankyo's HER2-targeting ADC trastuzumab deruxtecan (DS-8201), except in Japan where Daiichi Sankyo maintains exclusive rights.

GUSTAVE ROUSSY CANCER CAMPUS GRAND PARIS

2020 A multi-year, multi-study research collaboration
for the development of two of Daiichi Sankyo's lead DXd ADCs: DS-1062, a TROP2-directed DXd ADC, in patients with advanced NSCLC, and patritumab deruxtecan (U3-1402), a HER3-directed DXd ADC, in patients with metastatic breast cancer.

MERCK INVENTING FOR LIFE

2020 A clinical trial collaboration agreement
with MSD, a subsidiary of Merck & Co., Inc., to evaluate the combination of DS-1062, a TROP2-directed DXd ADC, and pembrolizumab in patients with previously treated advanced or metastatic NSCLC without actionable genomic alterations.

SARAH CANNON Fighting Cancer Together.

2020 A strategic oncology development collaboration
currently advancing two investigational ADC: DS-6157, a GPR20-directed ADC, in patients with advanced gastrointestinal stromal tumours and DS-7300, a B7-H3-directed ADC, in patients with metastatic solid tumours.

Daiichi-Sankyo

Daiichi-Sankyo

At Daiichi Sankyo we know that for people living with cancer every moment matters.
That's why we focus our efforts on addressing areas of unmet medical need.
We do this with passion for true innovation and by putting the patient at the heart of everything we do.
Our purpose is to contribute to the enrichment of quality of life and to improve standards of care across the world.

TOGETHER FOR OUR PATIENTS. TOGETHER FOR THOSE WHO STAND BESIDE THEM.

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