R&D Day/ Investor Day

5 Feb. 2024

SCANDION ONCOLOGY

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Company overview

SCANDION ONCOLOGY

Company Overview

SCANDION ONCOLOGY

- **Headquarters:** Copenhagen, Denmark
- **Year Founded:** 2017
- **Employees: approx. 10** FTE
- **Ticker:** SCOL
- **Exchange:** Nasdaq First North Stockholm

Shareholder Information

Share Information

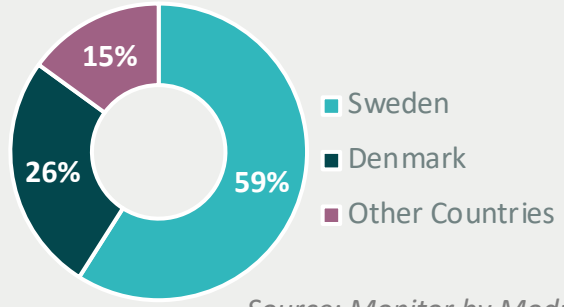
- Shares of Scandion Oncology A/S are listed on the Nasdaq First Growth Market Sweden
- Scandion's share capital amounts to 2,992 TDKK divided into 40,706,972 shares of nominal value 0.0735 DKK each
- There is only one class of shares, and each share represents one vote
- As of September 30, 2023, the number of shares was 40,706,972

Listing	First North Growth Market Sweden
Number of Shares	40,706,972
Ticker	SCOL
ISIN	DK0061031895

Shareholders

- There are no individual shareholders that own 5% or more of the shares in Scandion Oncology as of September 30, 2023
- According to the shareholder register maintained by Euroclear Sweden AB, Scandion Oncology had 7,643 shareholders as of September 30, 2023
- At the Annual General meeting on April 27, 2022 a new warrant program was approved, authorizing the Board of Directors to issue up to 4,177,620 new warrants which carry the right to subscribe for an equal number of shares in Scandion Oncology A/S

Shareholders by country, September 30, 2023



Source: Monitor by Modular Finance AB

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The Team – Track Record of Successfully Developing Biotech Companies

Francois Martelet, MD

Chief Executive Officer

- Doctorate in Medicine
- Master's Degree in Business
- Advanced Management Program, INSEAD
- Executive education finance & management programs, Harvard Business School

- +30 years experience in the global life science industry
- Leadership positions in several pharmaceutical Cos and CEO and chairman of a number of US and European biotech Cos

Johnny Stilou, MSc

Chief Financial Officer

- MSc in Business Economics and Auditing
- Executive Management Program, INSEAD

- +20 years' experience within biotech and the pharmaceutical industry where he has held numerous positions as CFO

Lars Damstrup, MD, PhD

Chief Medical Officer

- Medical Doctor,
- Ph.D. with specialization in Oncology

- +20 years' experience in clinical development
- 6 years of lung cancer research, 15 years in academia
- Leadership positions in several pharmaceutical and Biotech companies

Jan Stenvang, PhD

Chief Scientific Officer & Co-Founder

- Ph.D. in Molecular and Cellular Biology

- +20 years of experience in cancer research
- Specialized in translational cancer research particularly focusing on drug resistance and biomarker identification

Board of Directors

Martin Møller
Chairman of the Board

Jørgen Bardenfleth
Deputy-Chairman of the Board

Alejandra Mørk
Board member

Keld Flintholm Jørgensen
Board Member

Martine J. van Vugt
Board Member

SCANDION ONCOLOGY

Worldwide renowned Clinical Advisors

Richard L. Schilsky
MD, FACP, FSCT, FASCO

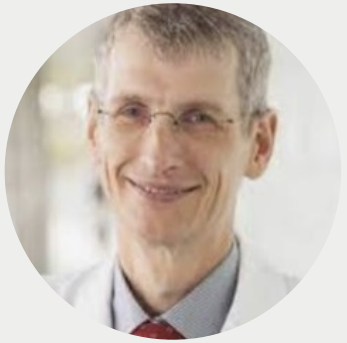
Former Executive Vice President and Chief Medical Officer of American Society of Clinical Oncology (ASCO) and past President of ASCO (2008-2009). Former Board member of Conquer Cancer, the ASCO Foundation

Josep Tabernero
MD, PhD

Member of the Executive Board of the European Society for Medical Oncology (ESMO) and past ESMO President (2018 – 2019). Appointed as member of several Educational and Scientific Committees of ESMO, ASCO, AACR, AACR/NCI/EORTC, ASCO Gastro-intestinal, and ESMO-GI/WCGIC meetings

Eric Van Cutsem
MD, PhD

President of the Belgian Foundation against Cancer. He co-founded ESMO GI/World Congress on Gastrointestinal Cancer, and is Chair of the meeting in Barcelona, Spain. He serves/served on the board or key committee of ESMO, ASCO, EORTC, ENET, ECCO, ESDO, and many others



Thomas Seufferlein
MD

President of the German Cancer Society (DKG) and chairman of the committee for cancer prevention of the German Cancer Aid (DKH). Editor in Chief of the German Journal of Gastroenterology

Scandion Oncology has a strong pipeline of innovative assets

Program	Compound	Indication	Discovery/Pre-Clinical	Phase I	Phase II	Phase III
CORIST	SCO-101	Colorectal Cancer	*SCO-101 + FOLFIRI*		*Topline Part 3 data in January 2024*	
PANTAX	SCO-101	Pancreatic Cancer	*SCO-101 + nab-paclitaxel and gemcitabine*		*Final data in H1 2024*	
Gastric	SCO-101	Gastric		*Data published December 2023*		
201	SCO-201	Solid tumors		*Currently on hold*		

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SCO-101 – Summary of Work Performed To-Date

Preclinical Profiling
- Pharmacology
- ADME
- Toxicology

CMC
- Drug Product
- Drug Substance

Regulatory Interactions
- DKMA
- FDA

Identification of Chemical Analogs

Physical, Chemical, and Pharmaceutical Properties & Formulation

Robust IP Portfolio

Phase 1 Completion (CORIST)

Phase 2 Initiation (CORIST)

Phase 1 Completion (PANTAX)

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Cancer Drug Resistance

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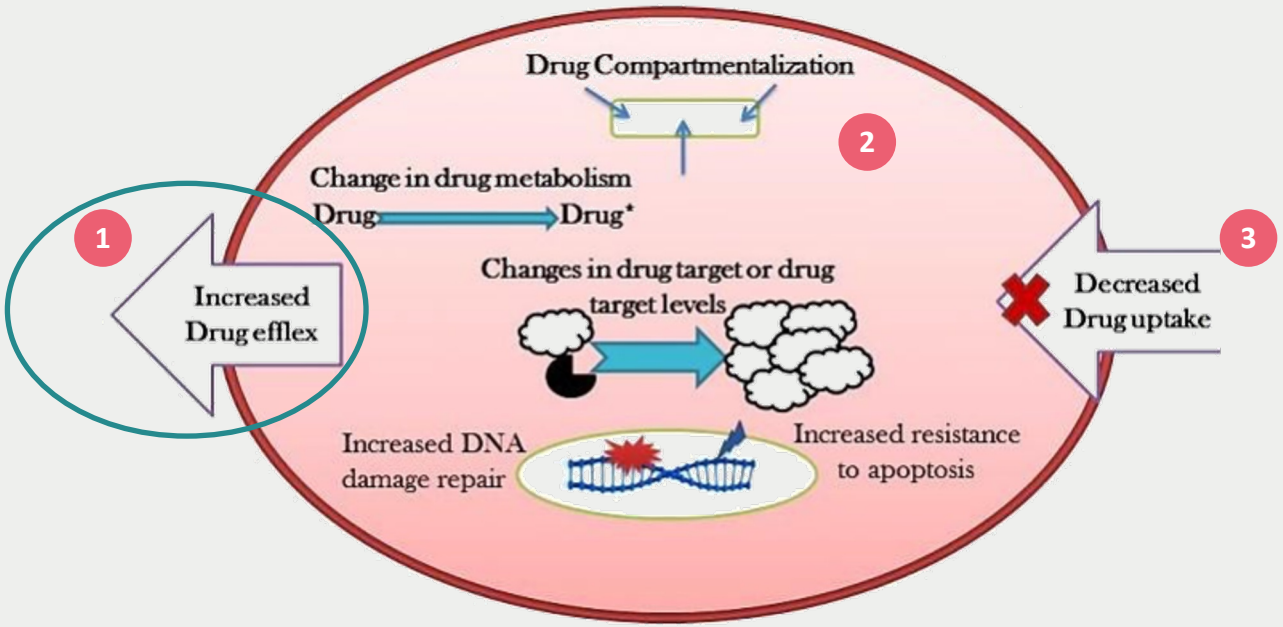
Cancer Drug Resistance

The Problem

Cancer Drug resistance continues to be one of the major limiting factors for achieving tumor reduction and cancer remission in patients

- Cancer drug resistance predominately arises through acquired and intrinsic resistance
- The overexpression of drug efflux pumps (such as ABCG2) is a common mechanisms for both acquired and intrinsic chemotherapy resistance

Cancer drug resistance is a multi-factorial challenge



Mechanisms of Cancer Drug Resistance

There are several mechanisms by which cancer cells can gain resistant properties to anti-cancer agents such as chemotherapy

1. **Increasing the release of drugs outside of the cell:**
   - A well-studied family of ATP-dependent transporters, called ABC transporters, act as a pump for removing chemotherapy agents
   - Reduces concentration of drug substrate intracellularly, thereby limiting the therapeutic effects of anti-cancer drugs
2. **Intracellular mechanisms and signaling:**
   - Several mechanisms such as the blocking of apoptosis, changing of drug metabolism, changes in drug target, and others play a key role in drug resistance
3. **Reducing the absorption of drugs into the cell:**
   - Reduction of binding affinity to transporters and a reduction in the number of transporters can inhibit drug absorption
   - Mutations in key transporters inhibit the ability for drug absorption into the tumor cell

90% of cancer deaths are due to resistance against current treatment options and there are no drugs currently available to counteract drug resistance by cancer cells

Sources: Mansoori et. al. (2017); Bukowski et al. (2020)

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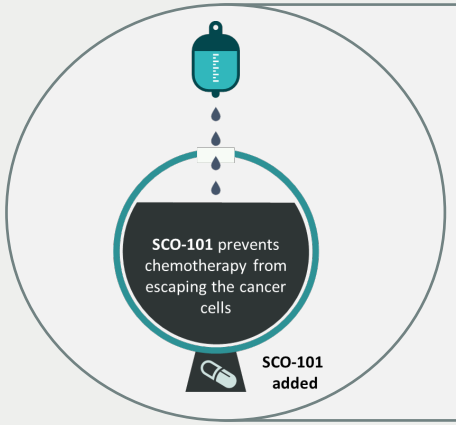
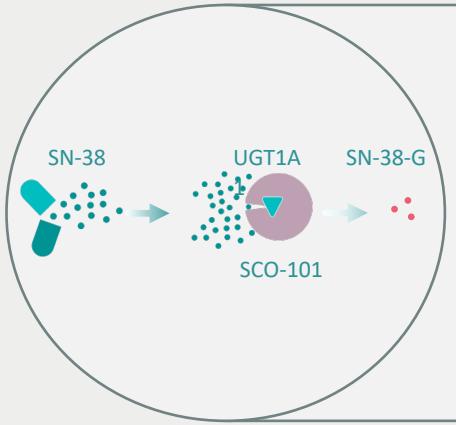
SCO-101 is a dual-acting molecule targeting ABCG2 and UGT1A1



Tumor Effect

Inhibition of ABCG2 prevents the drug efflux pump from removing the chemotherapy from the cell, thereby increasing the chemotherapy concentration in the tumor



Plasma Effect

Inhibition of UGT1A1 prevents the conversion of the active chemotherapeutic metabolite (SN-38) into its inactive form (SN-38-G), resulting in an increased concentration of the chemotherapy in the plasma

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SCO-101 targeting of ABCG2

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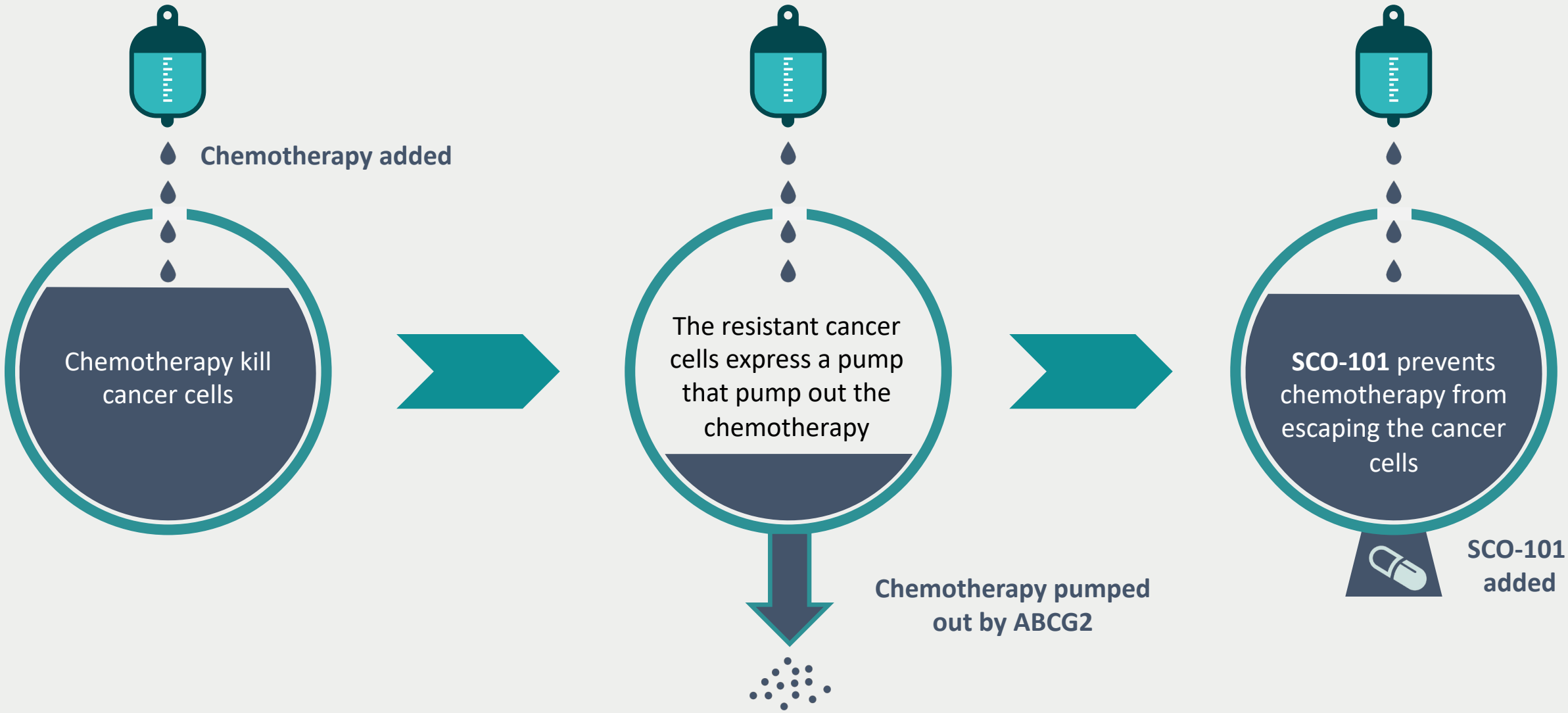
SCO-101 inhibition of ABCG2 efflux pump restores chemotherapy sensitivity



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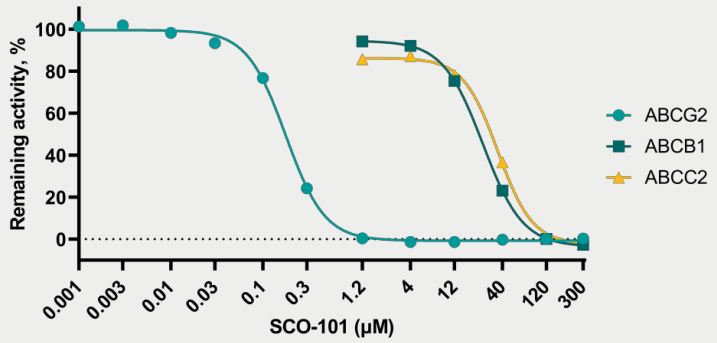
SCO-101 is a selective and potent ABCG2 inhibitor

Selective inhibition of ABCG2

SCO-101 is a selective and potent inhibitor of ABCG2

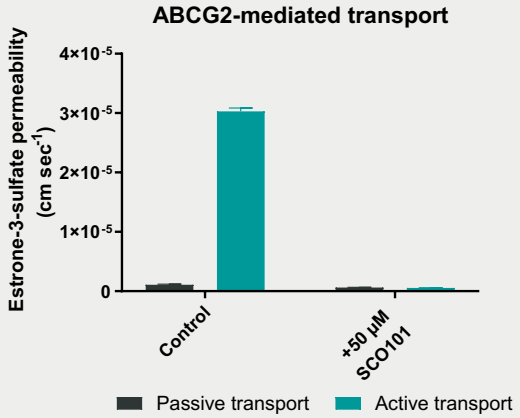
- Activity of the three major ABC transporters was measured using vesicular uptake upon titration with SCO-101 and their respective substrates
- ABCG2 was >100 fold more sensitive than ABCB1 or ABCC2
- IC_{50} for ABCG2 inhibition is 0.15 µM



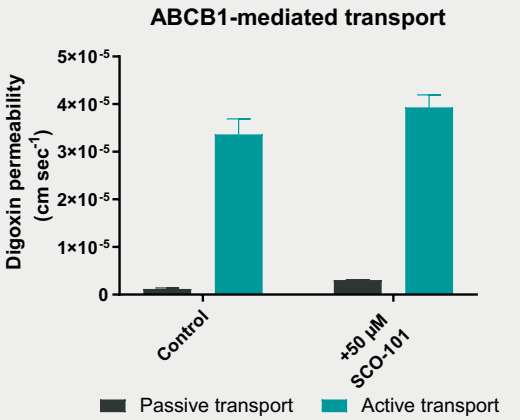
Transporter	ABCG2	ABCB1	ABCC2
IC50 (µM)	**0.15**	**16.1**	**24.1**

Selective inhibition of ABCG2

SCO-101 blocks ABCG2 mediated transport but not ABCG1 transports



Bi-directional transport of the ABCG2 substrate Estrone-3-sulfate in Caco-2 cells, in the absense and presence of SCO-101



Bi-directional transports of the ABCB1 substrate Digoxin in Caco-2 cells, in the absence and presence of SCO-101

**Passive transport: Apical to basolateral; Active transport: basolateral to apical*

*Stenvang et al. SCO-101 mediates re-sensitization of irinotecan (SN38) resistant colorectal cancer cells [abstract].
In: Proceedings of the AACR-NCI-EORTC Virtual International Conference on Molecular Targets and Cancer Therapeutics; 2021 Oct 7-10. Philadelphia (PA): AACR; Mol Cancer Ther 2021;20(12 Suppl):Abstract nr P077.

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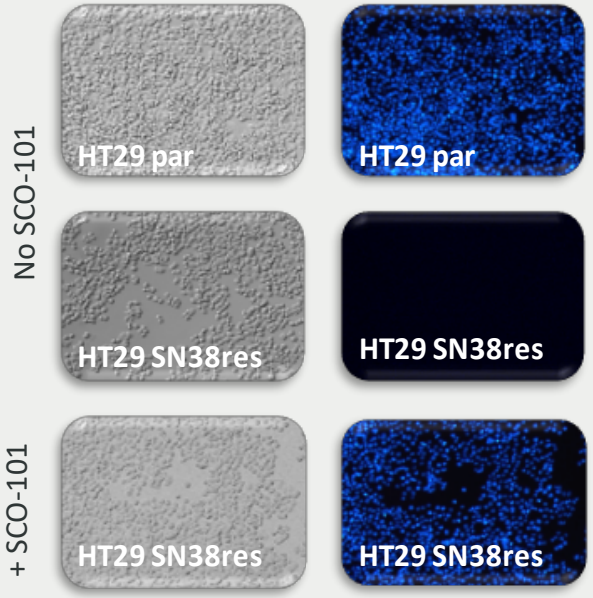
SCO-101 cause accumulation of active chemotherapy inside cancer cells

SCO-101 mediated dye accumulation

SCO-101 induces accumulation of ABCG2 substrate in SN38 resistant HT29 cells

SCO-101 prevents the ABCG2 substrate Hoechst33342 from being effluxed from the SN38 resistant HT29 cells

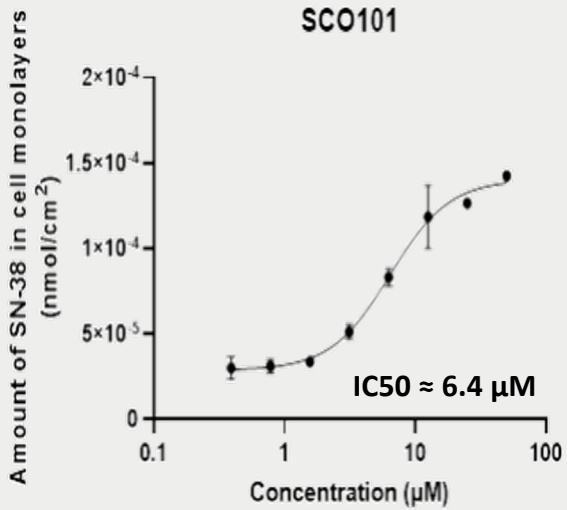


Fluorescence microscopy images of Hoechst33342 stained HT29 cells

Cytotoxic Accumulation

SCO-101 causes ^{3}H-SN38 accumulation in cancer cells

- Effect of SCO-101 on ^{3}H-SN38 accumulation in ABCG2 positive HT29-SN38resistant colon cancer cells
- IC_{50} measurement with SCO-101 and ^{3}H-SN38 found an IC_{50} of 6.4 µM



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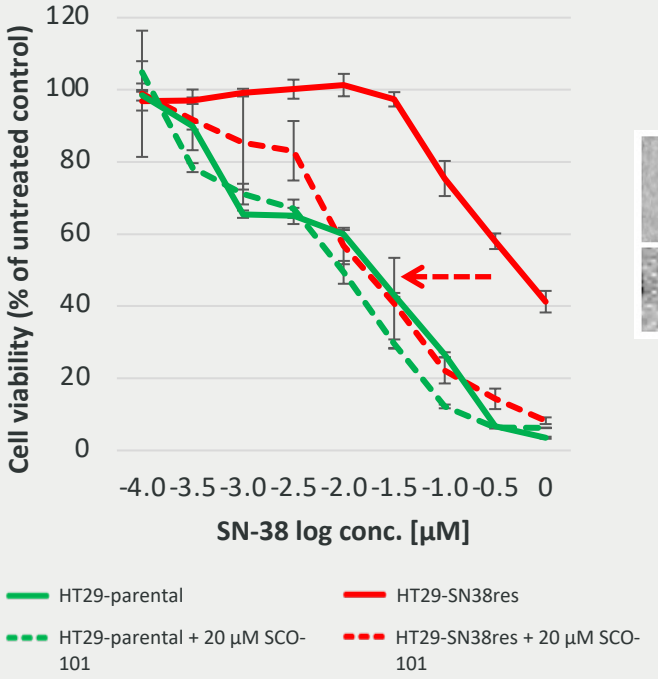
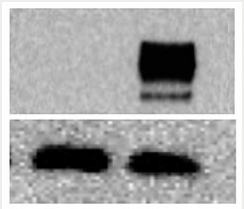
SCO-101 reduce the clonogenic potential and re-sensitizes ABCG2 positive SN-38 resistant colorectal cancer

Re-sensitization

SCO-101 re-sensitizes ABCG2 positive Irinotecan/SN-38 resistant colorectal cancer cells in *in vitro* pre-clinical models

- This effect is mediated primarily through the inhibition of the efflux pump ABCG2, leading to an increased intracellular exposure and prolonged retention of SN-38 inside cancer cells*

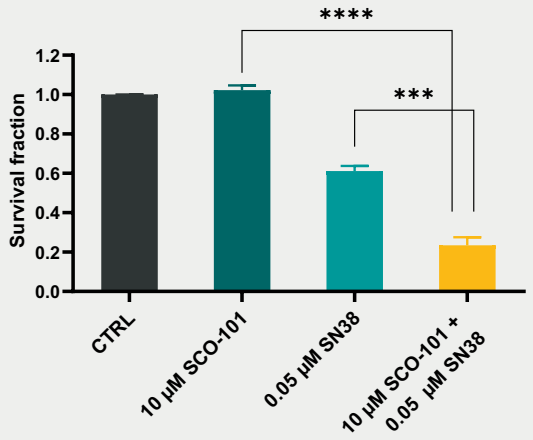




Cancer Killing Potential

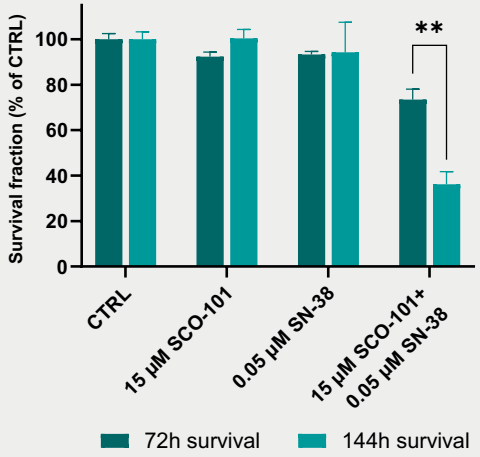
SCO-101 & SN-38 reduce clonogenic potential and short-term exposure is efficient

SCO-101 + SN38 reduces the clonogenic potential of HT29-SN38 resistant cells



Colony formation assay (6 days) on HT29-SN38 resistant cells treated with SN38, SCO-101 or the combination

Short exposure to SCO-101 and SN38 causes strong effects



HT29-SN38 resistant cells were treated with single drugs or combination for 24h, followed by a washout period of 48h or 120h; MTT viability assays at 72h or 144h

*Stenvang et al. SCO-101 mediates re-sensitization of irinotecan (SN38) resistant colorectal cancer cells [abstract]. In: Proceedings of the AACR-NCI-EORTC Virtual International Conference on Molecular Targets and Cancer Therapeutics; 2021 Oct 7-10. Philadelphia (PA): AACR; Mol Cancer Ther 2021;20(12 Suppl):Abstract nr P077.

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SCO-101 targeting of UGT1A1

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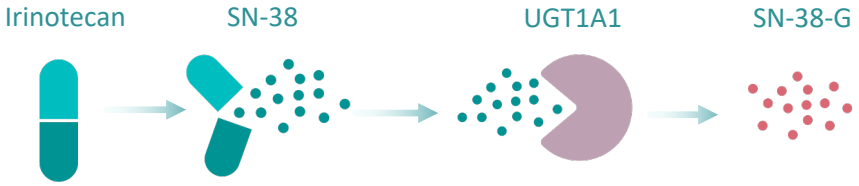
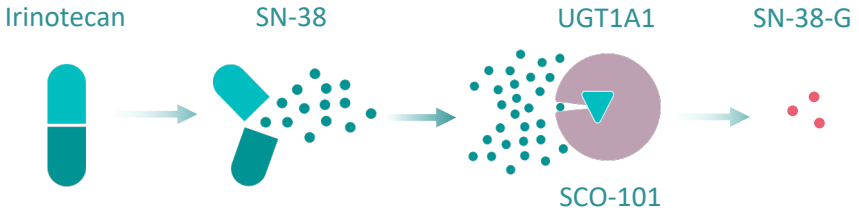
SCO-101 inhibition of UGT1A1 increase chemotherapy (SN-38)

No SCO-101



- In the liver, Irinotecan is converted to the active form of the chemotherapy (SN-38)
- SN-38 is converted by UGT1A1 to an inactive form (SN-38-G)

Plus SCO-101



- SCO-101 inhibition of UGT1A1 increase the level of active chemotherapy (SN-38)
- Increased levels of SN-38 will reach the tumor and cause stronger anti-tumor effects

SN-38 (active chemotherapy)

SN-38-G (inactive chemotherapy)

SCO-101

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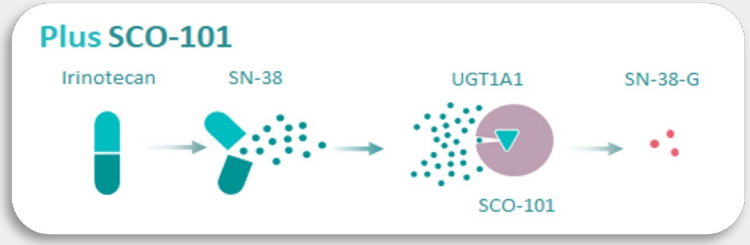
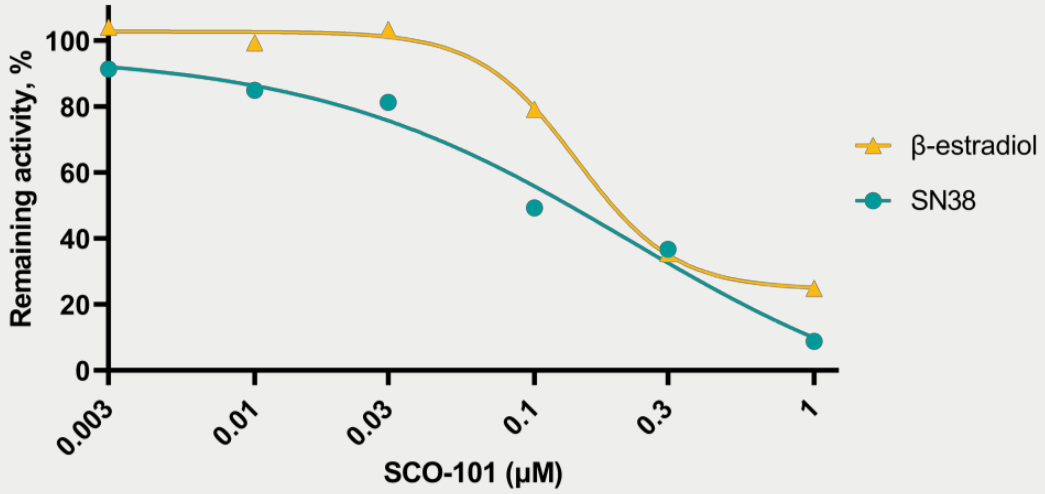
SCO-101 selectively and potently inhibits UGT1A1

UGT1A1 inhibition





Activity determination for UGT1A1 upon titration with SCO-101 showed potent inhibition with two UGT1A1 substrates.

Selective UGT1A1 inhibition

SCO-101 is a selective and potent inhibitor of UGT1A1

- Activity of seven UGTs was measured using human recombinant enzymes as a function of SCO-101 concentration.
- UGT1A1 was selectively and potently inhibited
- IC_{50} for UGT1A1 inhibition is 0.1 µM for SN-38

Enzyme	Substrate	IC50 (µM)
UGT1A1	SN-38	0.1
UGT1A1	B-estradiol	0.3
UGT1A3	CDCA	14
UGT1A4	Trifluoperazine	9.0
UGT1A6	4-MU	5.7
UGT1A9	4-MU	1.5
UGT2B7	Naloxone	5.7
UGT2B15	4-MU	9.4

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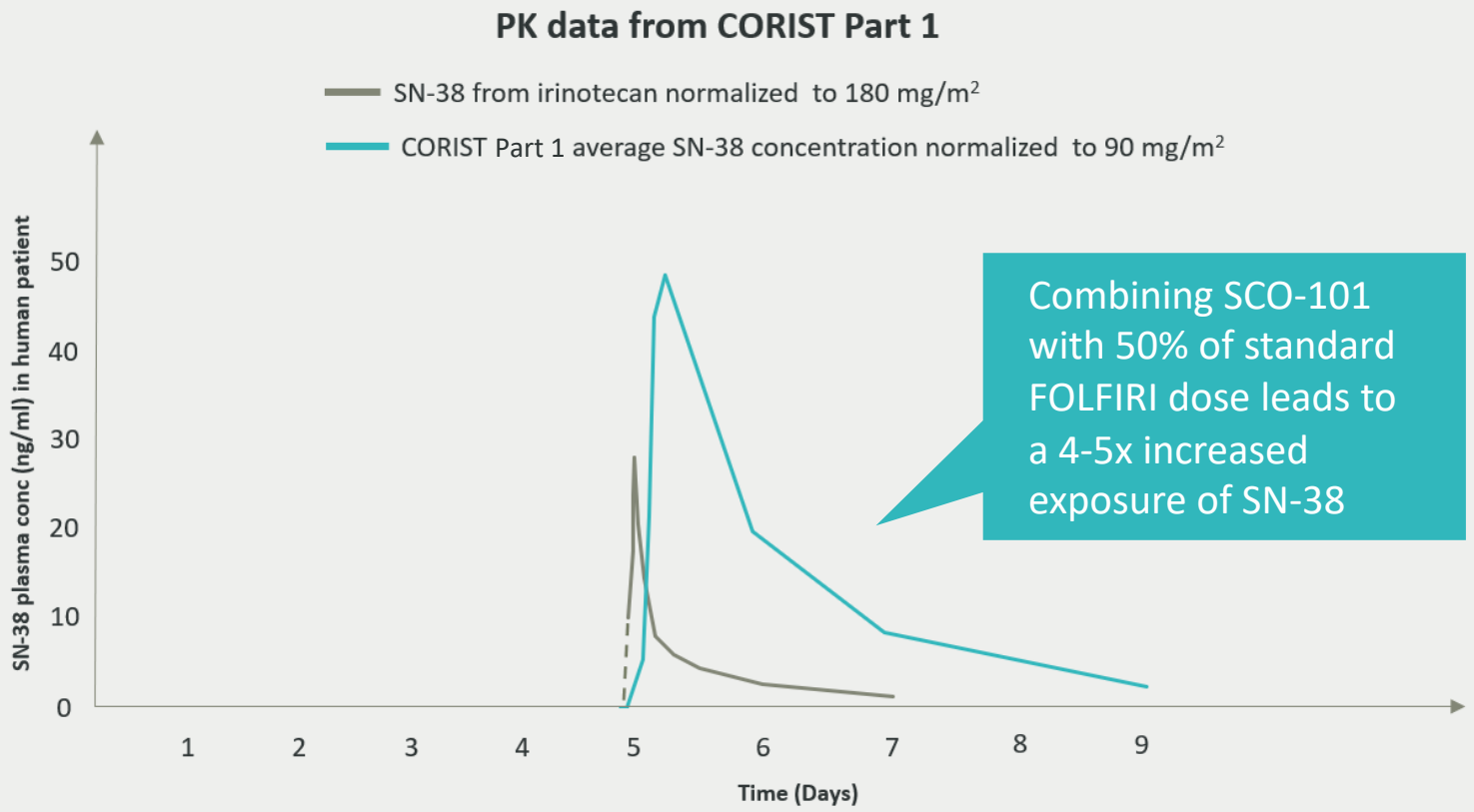
In patients SCO-101 caused increased exposure of active chemotherapy



Key Findings

SCO-101 notably potentiated the biological effect of FOLFIRI in patients; enabling increased therapeutic affects on malignant tissues without drastically changing the safety/tolerability profile

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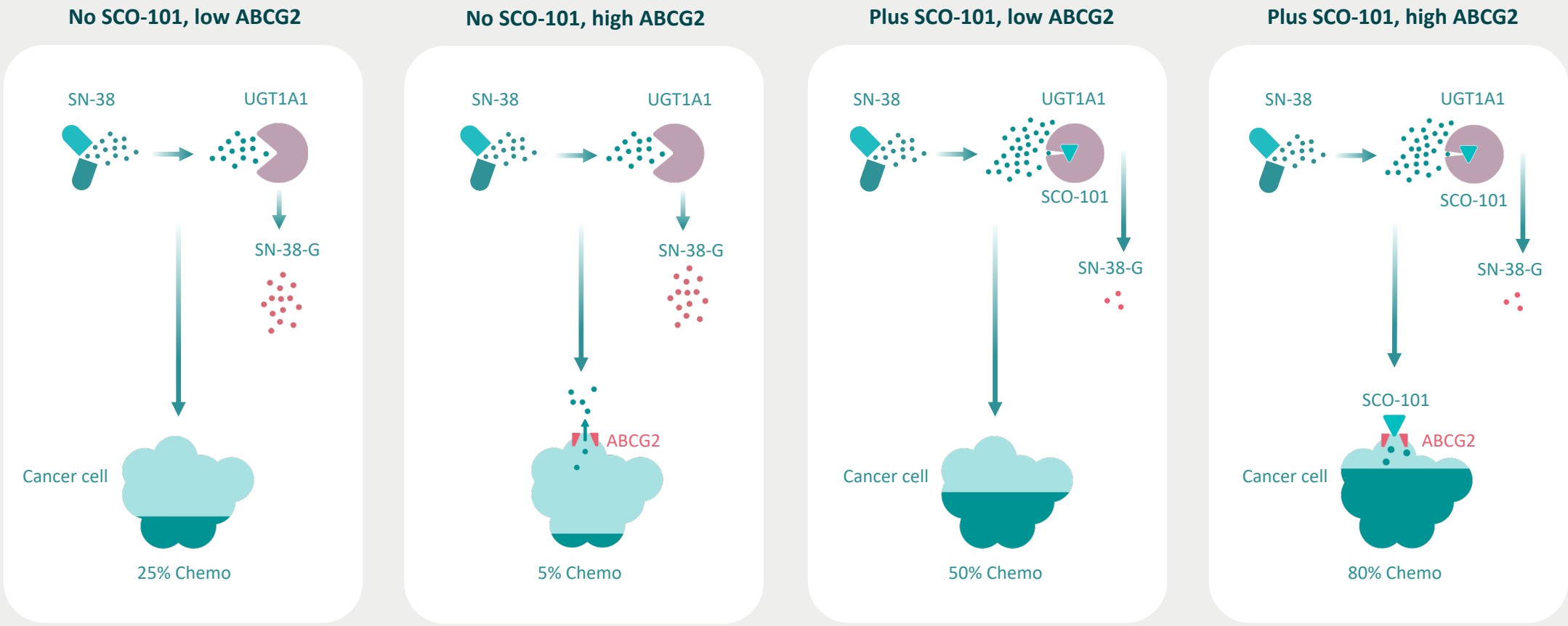
Effect of SCO-101 on chemotherapy tumor impact



SN-38 (active chemotherapy)

SN-38-G (inactive chemotherapy)

SCO-101

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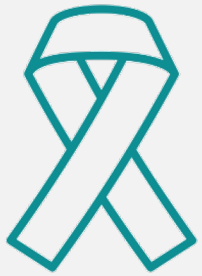
SCO-201 is a small molecule drug that has demonstrated activity as both an antiviral and in treating multi-drug resistant cancer



Cancer Potential*

- SCO-201 is a highly promising drug candidate for drug-resistant cancer by specifically inhibiting ABCG2 (IC50 0.027 µM), while also being a selective inhibitor of UGT1A1 (IC50 0.7 µM)
- Studies showed that SCO-201 reverses resistance to SN-38 in both *in vitro* cell viability assays in SN-38 resistant colon cancer cells and in non-cancer cells with ectopic expression of ABCG2
- Re-sensitization to various anti-cancer drugs has been demonstrated in cell line models
- Impressive safety profile – no cytotoxic effects up to 100 µM in human hepatocytes
- ABCG2 specificity could provide benefits to the safety and tolerability of co-medication compared to the application of broad-spectrum inhibitors

Antiviral Potential#

- SCO-201 targets Picornaviridae, especially Rhino and Enterovirus by integrating into the hydrophobic pocket formed by viral capsid protein 1
- Binding to the viral capsid leads to the prevention of virus adsorption to their host cells and/or uncoating thus blocking viral replication at an early stage of the cycle
- Antiviral activity has been shown for 8 of 14 clinical isolates of human enterovirus (HEV), especially Coxsackie virus B3 (CVB3), and 44 of 46 serotypes of the human rhinovirus (HRV)
- Mean antiviral activity was observed with an IC50 and 26 µM for HRV, 5 µM for HEV, and 0.14 µM for CVB3 (*in vitro* PFU inhibition)
- Highest activities were found against CVB3, Nancy, RV5, RV42, RV44, RV48, and RV69, all of them containing amino acid substitutions conferring high-level pleconaril resistance

*Ambjørner et al. The Pyrazolo[3,4-d]pyrimidine Derivative, SCO-201, Reverses Multidrug Resistance Mediated by ABCG2/BCRP. Cells, 2020, Mar 4;9(3):613.
#Makarov et al. Pyrazolopyrimidines: Potent Inhibitors Targeting the Capsid of Rhino- and Enteroviruses. ChemMedChem. 2015 Oct;10(10):1629-34.

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Despite the number of ART therapies available for HIV, increased use has been accompanied by drug resistance

ABCG2 Role in HIV

Disease progression in HIV is associated with increased activation of adaptive and innate immune systems – it is hypothesized that ABC transporters play an indirect role in sustaining persistent HIV infection by increasing proinflammatory cytokine release

- Despite effective antiretroviral therapy (ART), there is evidence that most HIV patients will have residual inflammation and increased immune activation
- The expression of several drug efflux transporters, including ABCG2, has been correlated with T-cell activation
- Zhang et al. demonstrated that HIV patients (both treated and untreated) have higher expression of ABCG2 in CD4+ and CD8+ T-cells
  - This could suggest that activated CD4+ T-cells, which is the preferred target for HIV infection and replication, may also express higher levels of efflux pump transporters

Additionally, there is evidence that ABC drug efflux transporters and drug metabolic enzymes may reduce antiretroviral concentrations in HIV target cells

- Cell lines have demonstrated that the efflux transporters P-gp, MRP, and BCRP/ABCG2 limit accumulation of antiretrovirals

Drug resistance remains a significant unmet need

1.2M	HIV patients in the US
32k	New cases in 2021 in the US
13%	Are undiagnosed, resulting in lack of early care
29.8M	Patients receiving ART worldwide
20-30%	Of patients not achieving viral load suppression
50-90%	Of patients failing ART is due to resistance
50%	Of infants born to mothers with HIV has HIV drug resistance

ABC = ATP-binding cassette

Sources: WHO; HIV.gov; Whyte-Allman et al., *Front. Pharmacol.* (2021);12; Whytte-Allman et al., *AIDS* (2020);34(10):1439-1449; Janneh et al., *J Antimicrob Chemother.* (2007);60(5):987-93; Janneh et al., *AIDS* (2005);19(18):2097-102

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CORIST is a Phase IIa study of SCO-101 in combination with FOLFIRI to treat patients with FOLFIRI resistant metastatic colorectal cancer (mCRC)

Design

- A four-part multi-center, open label, Phase IIa study of SCO-101 in combination with FOLFIRI

Part 1: Completed ✓
Dose Finding
18 Patients

→

Part 2: Completed ✓
Efficacy Assessment
25 Patients

→

Part 3: Ongoing
Optimized Dose Finding
25 Patients

→

Next Steps:
Additional Efficacy Assessment

Patient Population & Inclusion Criteria

- Patients with metastatic colorectal cancer with acquired resistance to FOLFIRI (up to N=103)
- >18 years old, maximum reduction of 33% in prior dose of FOLFIRI, performance status of ECOG≤1, recovered to Grade 1 or less from prior treatment(s)*
- Adequate conditions: ANC ≥ 1.5 x 109/L, hemoglobin ≥ 6.0 mmol/L, platelets ≥ 100 x 109/L, ALT ≤ 2.5 x AST ≤ 2.5 x ULN, total serum bilirubin ≤ 1.0 ULN, alkaline phosphatase ≤ 2.5 x ULN, creatinine ≤ 1.5 ULN, eGFR within normal limits, adequate blood clotting function INR ≤ 1.2
- Life expectancy ≥ 3 months

Dosing Schedule

- Updated treatment cycle schedule expected to improve efficacy and tolerability due to prolonged duration of exposure to SN-38:

6-Day Schedule	4-Day Schedule**
+ SCO-101 once daily on day 1 to day 6	+ SCO-101 once daily on day 2 to day 5
+ FOLFIRI on day 2-4	+ FOLFIRI on day 2-4

*Prior surgery or acute toxicities of prior radiotherapy or cytotoxic or biologic agents
** 4-Day Schedule will be used if 2 DLTs occur in any dose level with the 6-day regimen

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CORIST Overview

	CORIST Part 1			CORIST Part 2	CORIST Part 3			
Primary endpoint	MTD			Objective response	MTD			
Patients (N)	18 patients			25 patients (gCSF mandated)	25 patients (gCSF recommended)			
Population (mCRC)	All-comers			K-Ras wild type	All-comers			
SCO-101 (mg) and Patients (N)	150mg (4)	150mg (8)	100mg (6)	150mg (25)	150mg (7)	200mg (4)	200mg (7)	250mg (7)
Dose IRI (%)	80%	65%	50%	50%	50%			
Dose FOL and 5-FU (%)	80%	65%	50%	50%	100%			
Schedule	SCO-101: Days 1-6 FOLFIRI: Days 5-7			SCO-101: Days 1-6 FOLFIRI: Days 5-7	SCO-101: Days 1-6 FOLFIRI: Days 2-4		SCO-101: Days 2-5 FOLFIRI: Days 2-4	
Main outcome	RP2D used in part 2 decided by the DSMB			Impressive OS Potential biomarker 5 patients with tumor reduction	MTD established for 6 day schedule MAD determined for 4 day schedule Biomarker also positive for CORIST part 3 Follow-up ongoing			

MTD: Maximal Tolerated dose - RP2D: Recommend Phase 2 Dose
gCSF: granulocyte Colony Stimulating Factor – mCRC: metastatic Colo-Rectal Cancer
FOLFIRI: Folinic acid Leucovorin, 5-Fluoracil and Irinotecan – DSMB: Data Safety Monitor Board

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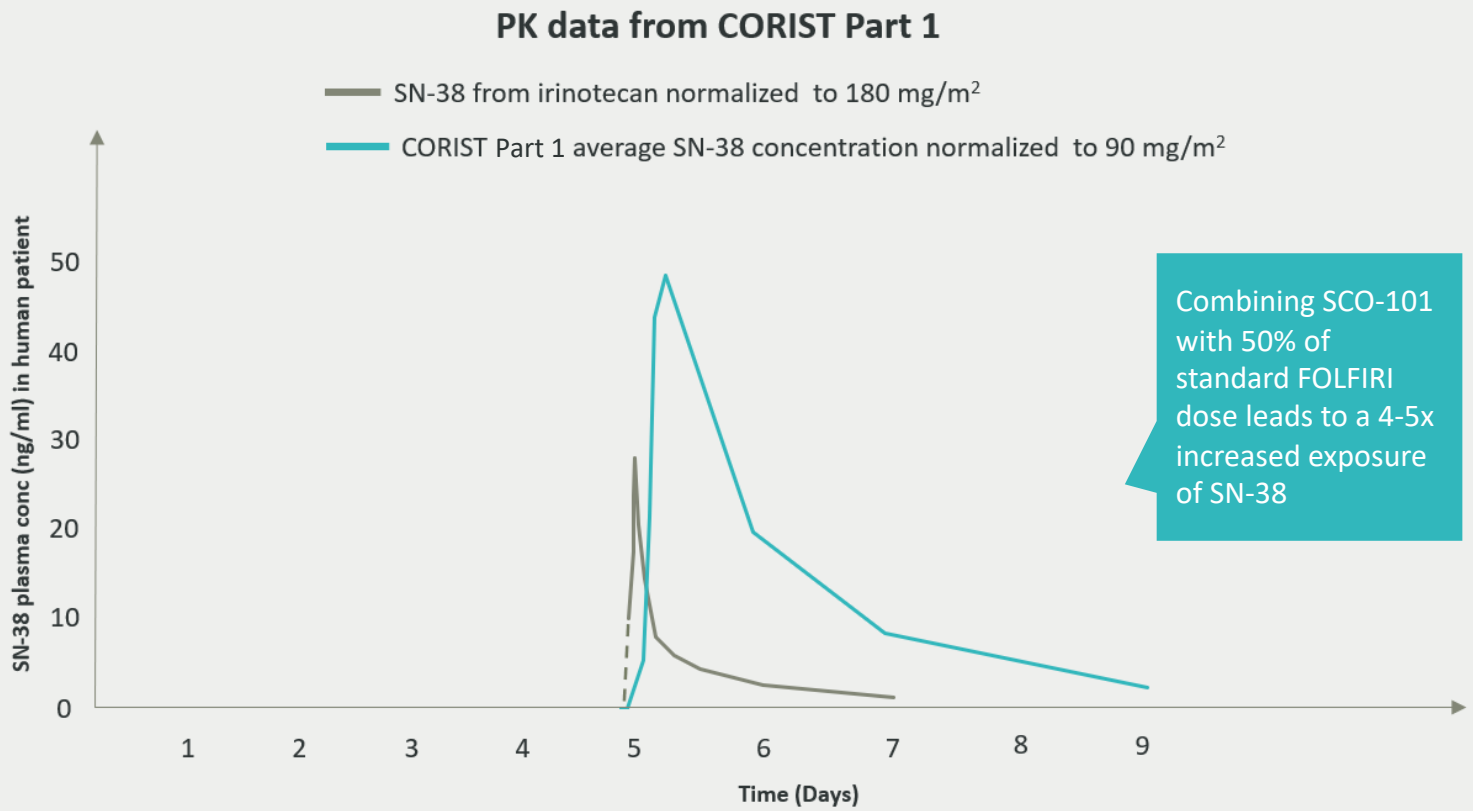
CORIST Part 1: SCO-101 was found to be safe and its maximum tolerated dose (MTD) was identified



Key Findings

- Established a maximum tolerated dose (MTD) of SCO-101 in combination with FOLFIRI
  - + MTD was 100mg for the RAS-mutated patients and 150mg for the RAS-wildtype patients, with RP2D to be 150mg
- SCO-101 notably potentiated the biological effect of FOLFIRI in patients; enabling increased therapeutic affects on malignant tissues without drastically changing the safety/tolerability profile
- SCO-101 was found to safe and well-tolerated, with no treatment-related grade 3 or 4 events observed at the MTD dose

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CORIST Part 2: Key Findings

Impressive median Overall Survival (OS) of 10.4 months*

- OS was evaluated as a secondary endpoint, with a median OS of 10.4 months in the 25 patients studied
- Historical median OS data for the same patient population treated with placebo or best supportive care have been reported in the range of 5-7 months**

Promising median Progression Free Survival (PFS) of 2 months

- An additional secondary endpoint was PFS, which was observed to be a median 2.0 months
- Historical data is reported in the range of 1.7-1.8 months**

Exciting Clinical Benefit Rate (CBR) of 20%

- CBR assessed after 8 weeks was found to be 42%
- Historical controls where CBR was evaluated after 6 weeks have been reported to be 11-16%

Successful tumor reduction

- Tumor reduction was observed in 5 out of the 25 patients
- However, it was below the -30% threshold defined as the trial's primary endpoint

Identification of unconjugated bilirubin as a potential biomarker

- Subset of patients (17 out of 25) that had a transient increase in unconjugated bilirubin had a median OS of 13.4 months
- The remaining patients (8 out of 25) that had a more persistent increase in unconjugated bilirubin had a median OS of 8.0 months

*Final Data from the Phase IIa Open-Label CORIST Part 2 Trial Shows Impressive Median Overall Survival of 10.4 months;**Xu et.al., 2018, J Clin Oncol., Van Cutsem et. al., 2018, Eur J Cancer., Mayer et. al., 2015, N Engl J Med, Grothey et. al., 2013, Lancet, Li et. al., 2015, Lancet Oncol, Yoshino et. al., 2012, Lancet Oncol

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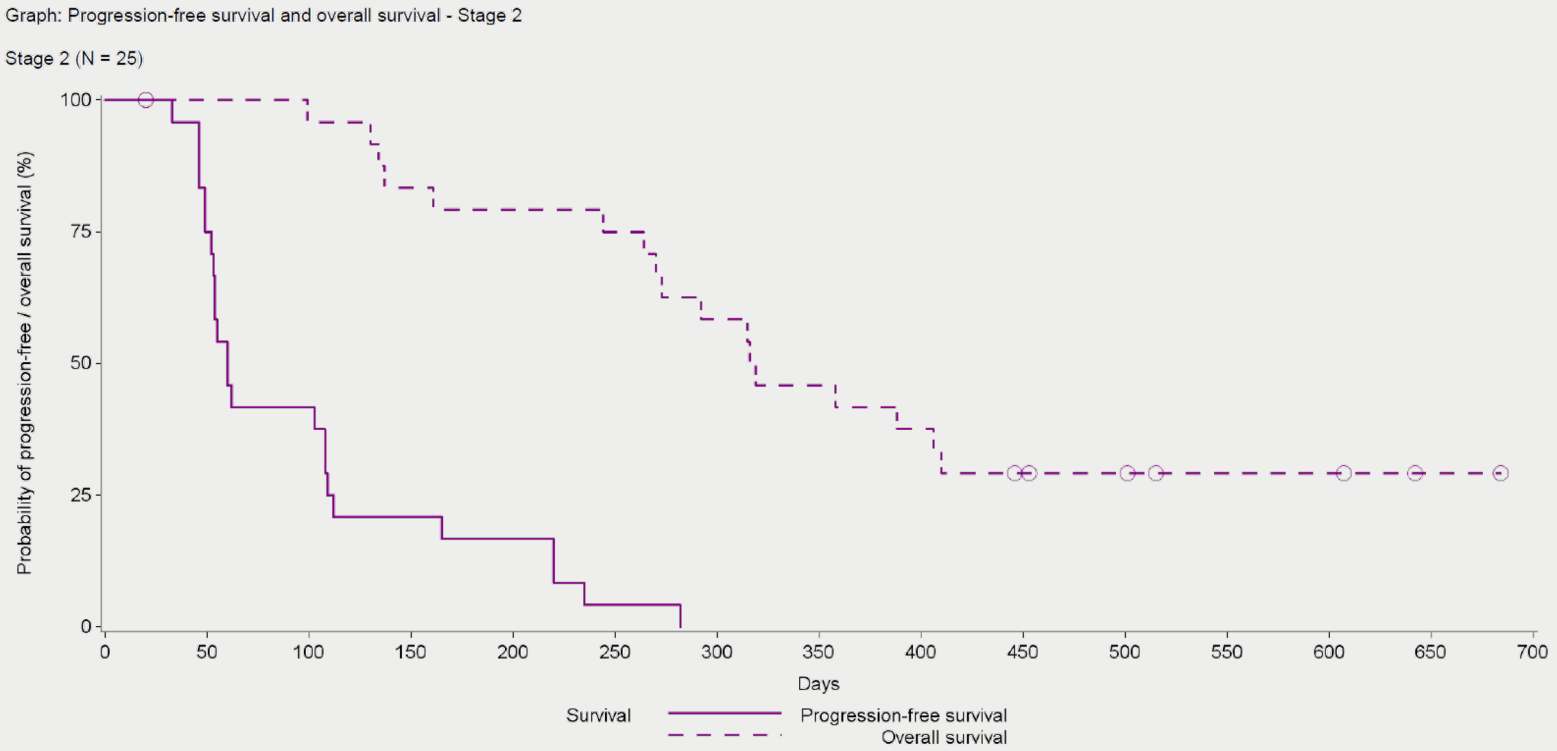
CORIST Part 2: SCO-101 showed promising median OS and PFS data when used to treat mCRC in combination with FOLFIRI

Progression Free Survival (PFS) and Overall survival (OS) in CORIST Part 2



Censoring is displayed with circles. Patient 001-028 is censored on Day 20.

Key Findings

Overall Survival (OS)

- Median OS of 10.4 months
- Historical median OS data for the same patient population treated with placebo or best supportive care 5-7 months
- Seven patients are still alive more than 15 months after start of treatment

Progression Free Survival data

- Median PFS was 2.0 months
- Indicating that SCO-101 + FOLFIRI was solely responsible for delaying disease progression by more than 0.2-0.3 months compared to historical data

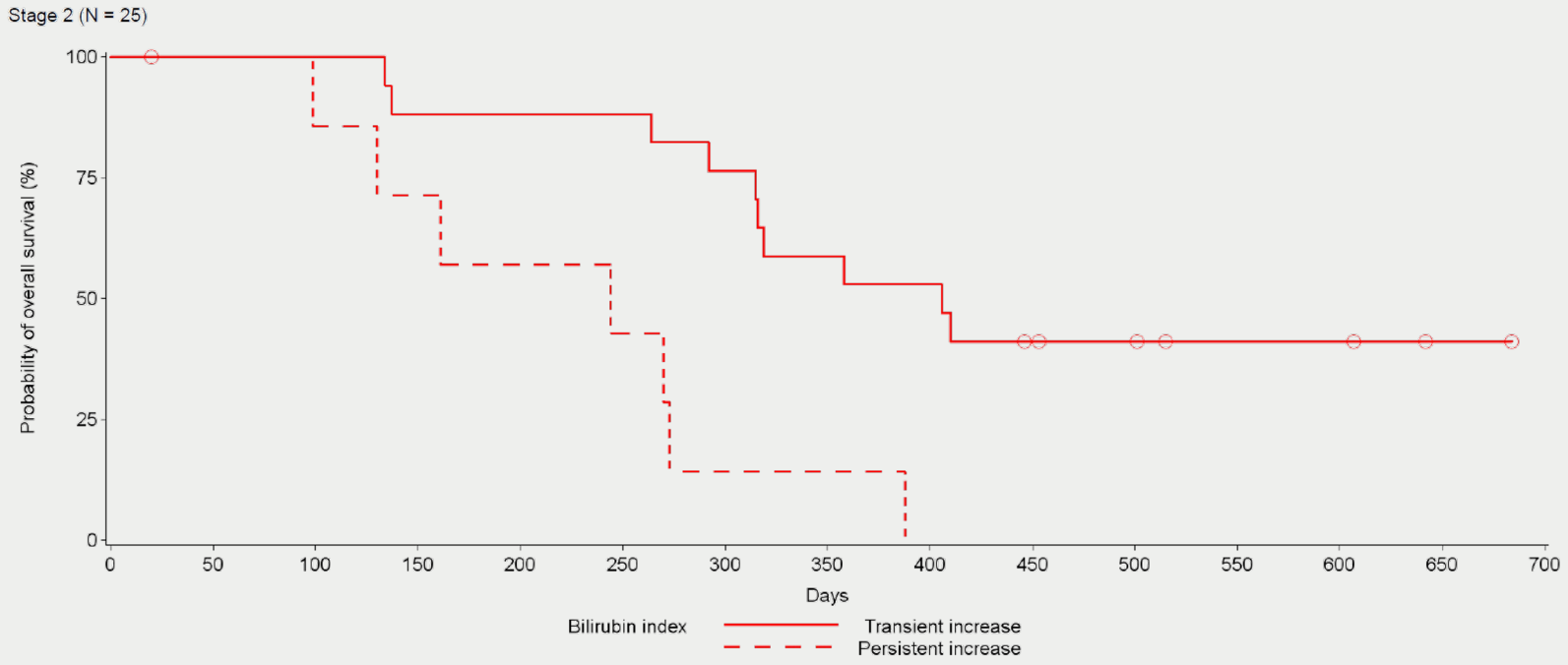
High Clinical Benefit Rate (CBR)

- CBR assessed after 8 weeks was found to be 42%
- Historical controls where CBR was evaluated after 6 weeks have been reported to be 11-16%

SCANDION ONCOLOGY

CORIST Part 2: Unconjugated bilirubin was identified as a potential biomarker

Overall survival (OS) in CORSIT Part 2 (N=25); Patients separated on the Bilirubin Index



P-value of Log-rank test *
0.0011

Censoring is displayed with circles. Patient 001-028 is censored on Day 20.
* The Log-rank test compares the survival distributions between patients with transient and persistent increase in bilirubin.

Key Findings

Bilirubin as a biomarker

- Unconjugated bilirubin was identified as a potential blood-based biomarker for patients most likely to respond to SCO-101
  - + SCO-101 targets UGT1A1, which is a liver enzyme that converts unconjugated bilirubin to conjugated bilirubin
  - + It has been observed in multiple studies that plasma levels of unconjugated bilirubin transiently increase after exposure to SCO-101
  - + In some patients, they are able to normalize the level of unconjugated bilirubin, resulting in a low bilirubin index (BI) with 0.4 utilized as the threshold
- 17 out of 25 patients had a low bilirubin index whereas the other eight patients had a higher bilirubin index
- Comparing OS, the low bilirubin index group had a significantly higher median of 13.4 months vs. 8.0 months in the high bilirubin group (P=0.0011)
- Notably, the seven patients (7/17 = 41%) still alive are all in the low bilirubin index group

SCANDION ONCOLOGY

CORIST Part 3: Refined dosing schedule was designed to optimize the activity of SCO-101 in combination with chemotherapy

Approach

- A dose escalation study in patients with mCRC irrespective of k-RAS status using two different dosing regimes
- Irinotecan given at 50%, 5-FU and leucovorin at 100% of SOC
- MTD established at 150 mg of SCO-101 given in a 6-day schedule. MAD of 250 mg SC0-101 in the 4 day schedule

Dosing Regime #1

- SCO-101 given days 1-6
- 2 dose limiting toxicities (DLTs) in 4 patients; no further dose escalation possible

Cohort 1 (N = 4)
150 mg of SCO-101

→ *Cohort 2 (N = 4)*
200 mg of SCO-101

→ *Cohort 3 (N = 3)*
150 mg of SCO-101

Dosing Regime #2

- SCO-101 given days 1-4
- No DLTs observed

Cohort 4 (N = 7)
200 mg of SCO-101

→ *Cohort 5 (N = 7)*
250 mg of SCO-101

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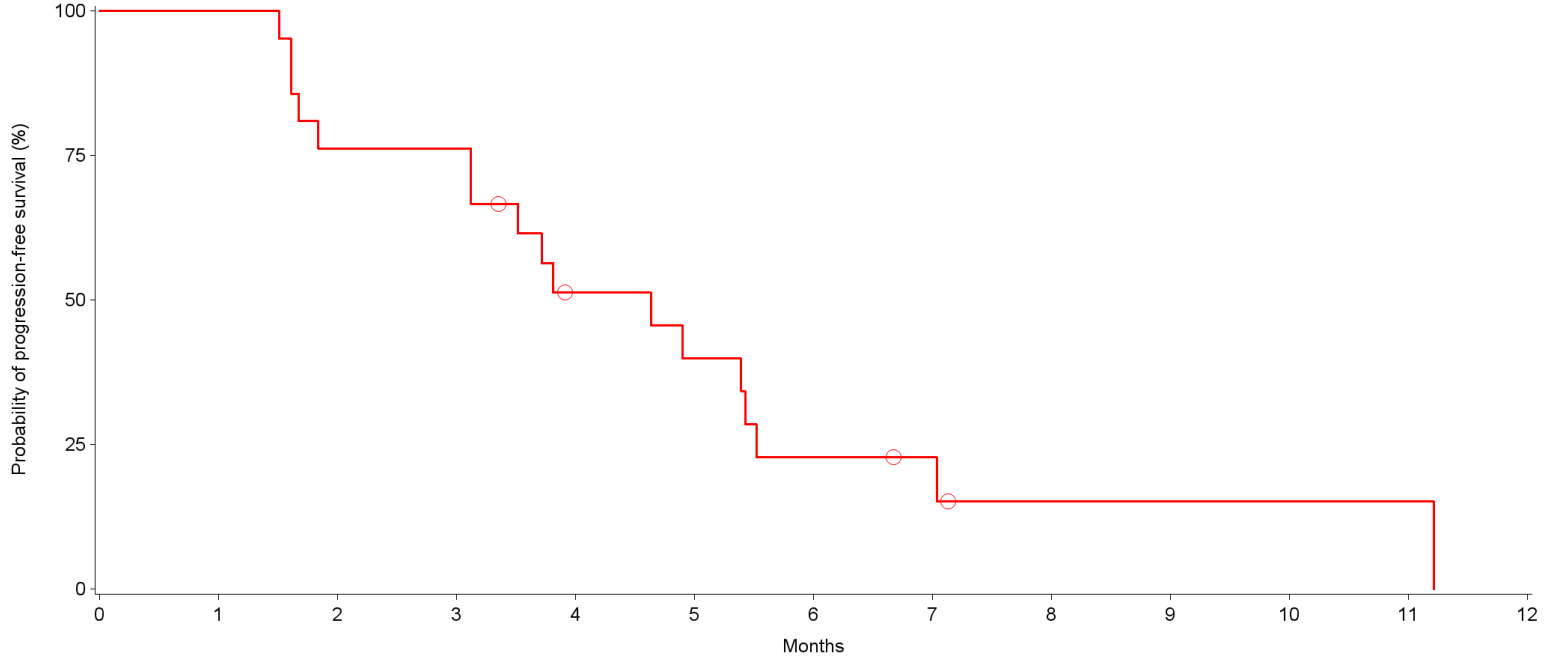
CORIST Part 3: SCO-101 showed promising median PFS data when used to treat mCRC in combination with FOLFIRI

Progression Free Survival (PFS) in CORIST Part 3



Median PFS (mPFS) is 4.6 months.
3 patients still ongoing and censoring is displayed with circles.

Key Findings

Progression Free Survival (PFS) data

- Median PFS is 4.6 months
- Historical median PFS data for the same patient population treated with placebo or best supportive care 1.7 – 1.8 months
- In CORIST Part 2 the mPFS was 2.0 months
- Three patients are still under treatment
- Indicating that SCO-101 + FOLFIRI was solely responsible for delaying disease progression by more than 2.8/2.9 months compared to historical data

High Clinical Benefit Rate (CBR)

- CBR assessed after 8 weeks was found to be 76%
- Historical controls where CBR was evaluated after 6 weeks have been reported to be 11-16%
- The CBR in CORIST Part 2 was 46% at week 8

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CORIST Part 3: Key Findings

Promising median Progression Free Survival (PFS) of 4.6 months

- One of the secondary endpoint was PFS, which was observed to have a median of 4.6 months
- Historical data is reported in the range of 1.7-1.8 months** In CORIST part 2 we recently reported PFS to be 2 months*

Exciting Clinical Benefit Rate (CBR) of 76%

- CBR assessed after 8 weeks was found to be 76%. CBR at later time points are not available yet
- Historical controls where CBR was evaluated after 6 weeks have been reported to be 11-16% In CORIST part 2 we recently reported CBR at week 8 to be 46%*

Successful tumor reduction

- Tumor reduction with more than 30% reduction was observed in 1 patient
- Tumor reduction with less than 30% reduction was observed in 15 patients

Identification of unconjugated bilirubin as a potential biomarker

- Our initial data confirms that bilirubin could be used as a potential biomarker

*Final Data from the Phase IIa Open-Label CORIST Part 2 Trial Shows Impressive Median Overall Survival of 10.4 months;**Xu et.al., 2018, J Clin Oncol., Van Cutsem et. al., 2018, Eur J Cancer., Mayer et. al., 2015, N Engl J Med, Grothey et. al., 2013, Lancet, Li et. al., 2015, Lancet Oncol, Yoshino et. al., 2012, Lancet Oncol

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CORIST: Lessons learned and Next Steps

Impressive median Overall Survival (OS) of 10.4 months in CORIST part 2

PFS in CORIST part 2 was 2 months

PFS in CORIST part 3 is 4.6 months

Several dosing schedules have been explored in CORIST part 1 and 3 with an intermediate dose in CORIST part 2

Maximal tolerated dose has been established in CORIST 1 and for the 6-day schedule in CORIST part 3

Maximal administrated dose has been reported for CORIST part 3 in the 4-day schedule

Exciting Clinical Benefit Rate (CBR) at week 8

In CORIST part 2 the CBR was 42%

In CORIST part 3 the CBR was 76%

Successful tumor reduction

In CORIST part 2 we saw several patients with a decrease in tumor burden, but no patient had a 30% or more reduction (threshold for a partial remission)

In CORIST part 3 we saw 1 patients with a PR and another patient who after 24 weeks continue to have tumor reduction but not yet at 30%

Next steps: Enabling study to optimize the dose of irinotecan in combination with SCO-101. *Goal is to Initiate a randomize proof-of-concept phase IIb study*

In CORIST part 3 have one or more additional cohorts in the 4-day schedule, where the dose of irinotecan will be increased to optimize the combination in efficacy but not toxicity

Continue the evaluation to address the observation that bilirubin could be used in the future as a potential biomarker

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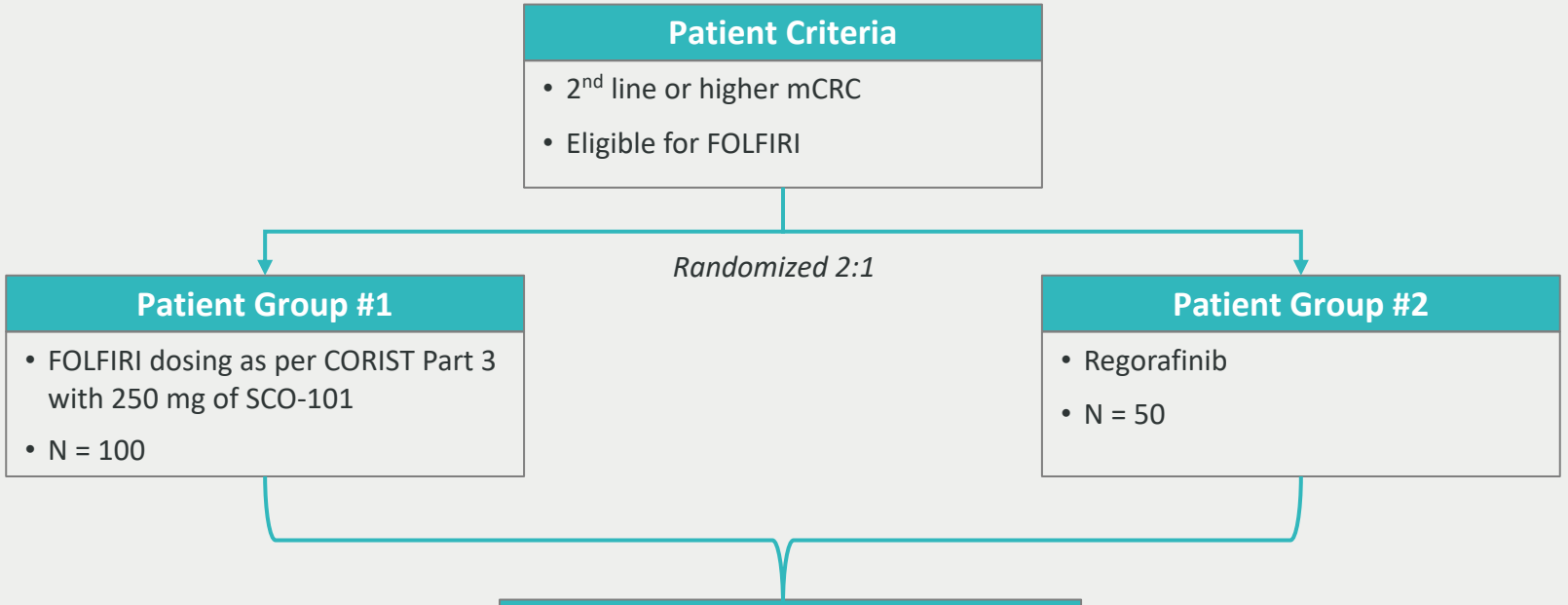
Beyond CORIST: SCO-101 in a randomized proof of concept Phase IIb study



Design, endpoints and comparator discussed and endorsed at an advisory board meeting with 6 EU KOLs

Primary endpoint: OS
Secondary endpoint: PFS, RR, CBR, DOR, evaluation of bilirubin, PRO, QoL etc

Primary endpoint: OS. Numbers needed to be treated has been confirmed based on the primary endpoint

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PANTAX is a Phase Ib dose escalation study of SCO-101 with gemcitabine and nab paclitaxel in pancreatic cancer

Design & Dosing Regimen
- A European, open-label multi-center prospective dose escalating single arm Phase 1b
- 28-Day treatment cycle of: SCO-101 - Days 1-6, Gemcitabine - weekly (at 80%), and Nab-paclitaxel: Q3W (at 80%)

Patient Population & Inclusion Criteria
- Inoperable localized, locally advanced or metastatic pancreatic cancer, not amenable for curatively intended treatment, in patients who are to be treated with gemcitabine and nab-paclitaxel (N=18)
- >18 years old, Performance status of ECOG ≤2, Recovered from Grade 1 or less from prior treatment(s)*, adequate conditions**, life expectancy > 3 months

Primary Endpoints
- To determine the safety and tolerability of SCO-101 in combination with gemcitabine and nab-paclitaxel
- To determine the Maximum Tolerated Dose (MTD) of SCO-101 in combination with gemcitabine and nab-paclitaxel as evaluated by Common Terminology Criteria for Adverse Events (CTCAE)

Secondary Endpoints
- Find the objective response rate (CR and PR), clinical benefit rate, PFS, OS, and pK profile
- Gain insight into novel predictive biomarker feasibility

Interim Findings***
- MTD of 200 mg of SCO-101 has been successfully established
- Final analysis to be presented in H1 2024

*Prior surgery or acute toxicities of prior radiotherapy or cytotoxic or biologic agents; **ANC ≥ 1.5 x 109/L, hemoglobin ≥ 6.0 mmol/L, platelets ≥ 100 x 109/L, ALT ≤ 2.5 x AST ≤ 2.5 x ULN, total serum bilirubin ≤ 1.0 ULN, alkaline phosphatase ≤ 2.5 x ULN, creatinine ≤ 1.5 ULN, eGFR within normal limits, adequate blood clotting function INR ≤ 1.2

*** Phase Ib PANTAX Trial is Successfully Completed and Establishes the Maximal Tolerated Dose with Positive Safety Profile and Pharmacokinetic Data

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Feedback from KOLs

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KOLs saw the merit in SCO-101's dual mechanism-of-action along with its positioning in treating colorectal and pancreatic cancer

Consideration	*Key Takeaways*	
Dual-MoA	**KOLs intrinsically understood the dual-mechanism of action of inhibiting ABCG2 and UGT1A1 to overcome drug resistance** • KOLs agreed that ABCG2 and UGT1A1 can have significant implications to the concentration of drug metabolites both intracellularly and extracellularly • There is a strong rationale for inhibiting ABCG2 due to its role in drug efflux and resistance in CRC and pancreatic cancer • Also, the targeting of UGTs was thought to be very relevant, especially in pancreatic cancer due to having more advanced drug resistance mechanisms and tumor fibrosis resulting in greater unmet need	*"The inhibition of both ABCG2 and UGTs will certainly increase the concentration of drug metabolites"* *– US KOL*
Positioning in Colorectal Cancer	**KOLs agreed that SCO-101 should be studied in the 2L setting after patients demonstrate resistance to 1L therapy** • ~90% of patients with CRC progress to 2L treatment, where options are limited with patients typically receiving a different therapy from 1L; additionally, 60%-70% advance to 3L treatment where there are even more limited therapeutic options • Due to the dual-mechanism of SCO-101, KOLs are interested to see how it potentiates cancer cells to chemotherapeutic agents in conjunction with FOLFIRI or FOLFOX in the 2L	*"The therapeutic options here are scarce. We don't really have any great treatments that can either prolong or reverse cancer growth" – US KOL*
Positioning in Pancreatic Cancer	**Given the high unmet need in pancreatic cancer, there was significant rationale for the use of SCO-101 in the 2L** • KOLs emphasized the significant unmet need in pancreatic cancer, especially as many patients are diagnosed at later stages due to lack of symptoms in earlier disease • Only ~80% of patients pursue 1L therapy, and a large proportion forego additional lines of therapy due to the severity and aggressiveness of the disease (only 40-60% of patients progress to 2L)	*"UGT inhibitors would be very effective against pancreatic cancer compared to other malignancies"* *– US KOL*

KOLs were impressed with SCO-101's PFS data and saw value in a biomarker to identify the most receptive patient populations to SCO-101

Consideration	*Key Takeaways*
PFS vs. OS	**Overall, KOLs were impressed with SCO-101's initial efficacy data, highlighting that OS was meaningfully higher than what is currently seen in the advanced patient population** • As many patients will try multiple lines of therapy, physicians are interested in PFS as a metric of efficacy, since OS may be impacted by the lines of therapy after investigational treatment • KOLs stressed the limited options and high unmet need in both cancer types, stating that any improvement to either PSF or OS (~15%-20% improvement) would lead to clinical utilization
Potential Biomarker	**KOLs saw value in using a potential biomarker to enrich the patient population in SCO-101 clinical trials to further improve safety and efficacy** • KOLs agreed that an improvement of median OS by ~1 month in patient segments with the optimal biomarker and perceived ability to tolerate SCO-101 can potentially be interesting to further investigate • It was noted that screening for mutations are already something that is standard practice and a UGT1A1 screen could potentially enrich the patient population as well, due to its function in metabolizing chemotherapy

"PFS gives us a better look at whether that drug is working the way it is supposed to"

– US KOL

"Identifying a biomarker makes a lot of sense, there are certainly patients with UGT1A1 mutations that I would not give this [SCO-101] to"

– US KOL

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SCO-101 has strong patent protection until at least 2042

	Patent family name	Expiry	Coverage	Territorial scope
P105	SCO-101 combination treatment	2037	SCO-101 and selected anti-cancer agents	**Granted**: AU, EP (37 countries), HK, JP, US *Pending*: BR, CA, CN, EP (divisional), US (divisional)
P106	SCO-101 kinase inhibition	2039	SCO-101 and selected anti-cancer agents for treatment of subjects with increased SRPK1 level/activity	*Pending*: EP, US
P109	SCO-101 treatment of RAS mut/wt. populations	2042	SCO-101 and selected anti-cancer agents for treatment of RAS mut/wt. subpopulation	*Pending*: International phase (PCT)
P126	SCO-101 PK optimization	2042	SCO-101 for increasing AUC of anti-cancer agents	*Pending*: International phase (PCT)
P134	SCO-101 MAB combination treatment	2042	SCO-101, PD1/PD-L1, and selected anti-cancer agents	*Pending*: International phase (PCT)
P168	SCO-101 Bilirubin index	2042	Diagnostic tool for optimizing treatment regimens of UGT1A1 substrates using SCO-101	*Pending*: International phase (PCT)
P179	SCO-101 Pevo combination treatment	2043	SCO-101 and pevonedistat for treatment of cancer	*Pending*: International phase (PCT)
P108	SCO-101 crystal forms and amorph	2042	Commercial and other crystal forms of SCO-101	**Granted**: EP (countries not decided yet) *Pending*: International phase (PCT)
P124	SCO-101 salts	2043	SCO-101 salts and crystal forms	*Pending*: International phase (PCT)

Scandion is working to bolster its IP estate regarding SCO-201

Family	Patent number	Main comments
I	WO 2007/147401	Broad general structure Therapeutic use: anti-viral
II	WO 2013/053942	Broad general structure - Exact structure of a handful number of compounds is described Therapeutic use: anti-viral
III	WO 2017/198700	General structure covers a smaller chemical space than the previous patents Therapeutic use: oncology (activity on BCRP)

Strategies to reinforce the IP around SCO-201:

- Identification of a few derivative (or even one derivative) with better properties covered by the family III:
  - Superiority can be on any relevant aspect (AGCG2 inhibition, UGT1A1 selectivity, solubility etc.) – the more parameters the better
- Identification of a small class of close derivatives covered by the family I and II but not by the family III with application in oncology (via ABCG2 inhibition):
  - Note: even if the structure is not directly claimed in III it can still be considered obvious, in this case superiority would still be proved

Commercial Opportunity

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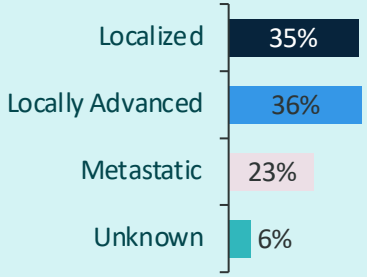
Colorectal Cancer Market Opportunity / Positioning

LARGE UNMET CLINICAL NEED

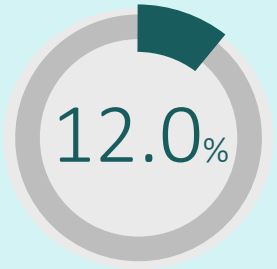
US incident annual cases

153,000

EU: 488,000/year

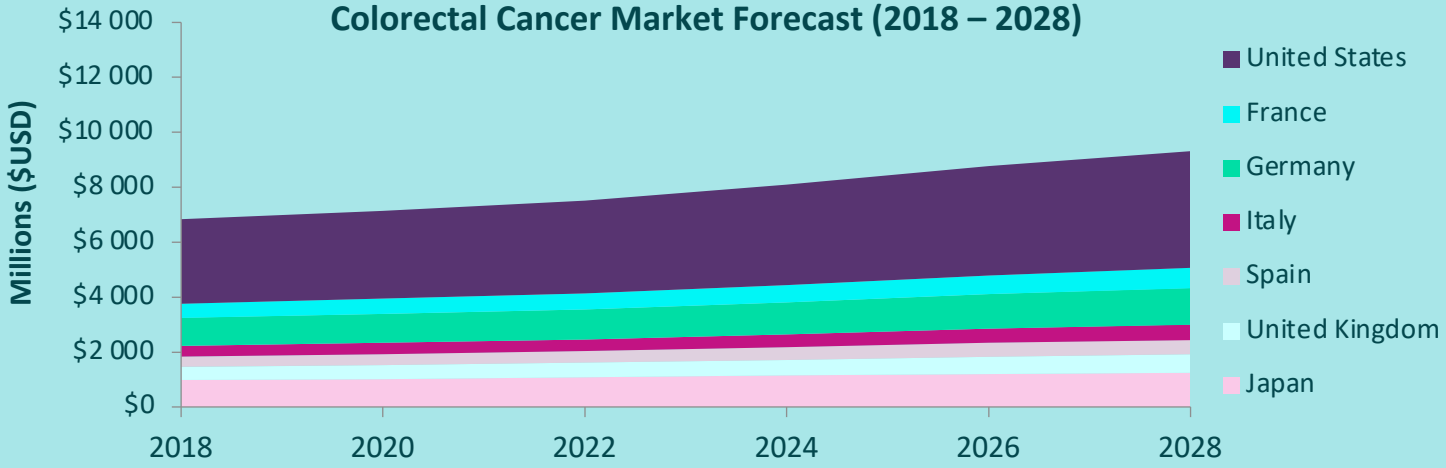


Stage at Diagnosis

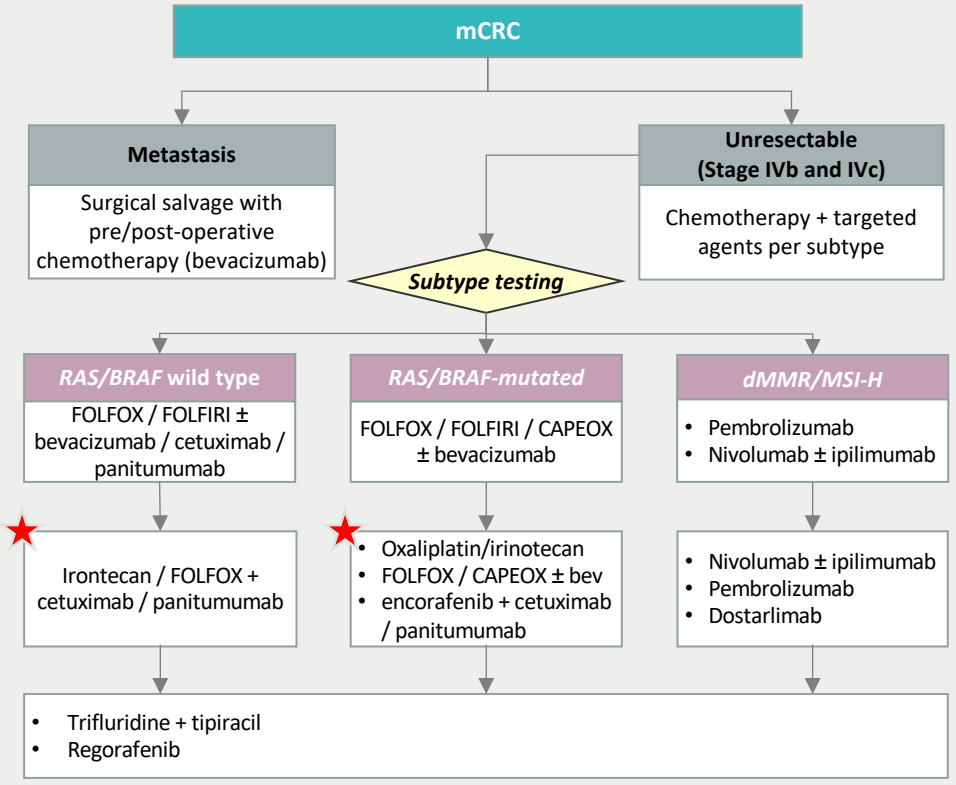


5-year survival rate, stage IV CRC

Colorectal Cancer Market Forecast (2018 – 2028)



SCO-101 Positioning



★ **Initial Positioning for SCO-101**

Sources: Datamonitor Healthcare: Colorectal Cancer, SEER, Rawla et, al. (2019), De Rosa et, al. (2015), Back Bay Analysis

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Metastatic Colorectal Cancer: SCO-101 Commercial Opportunity

# of new colorectal cancer cases per year:
US: 153k, EU: 488k

95% of cases are colorectal adenocarcinoma

# of pancreatic adenocarcinoma cases:
US: 145k, EU: 464k

40% of patients are metastatic

# of late stage pts:
US: 58k, EU: 185k

90% of late stage patients are treated

# of pts treated:
US: 52k, EU: 167k

70% of patients require 2L treatment

# of pts receive 2L treatment:
US: 37k, EU: 117k

Assume 60% market share due to high unmet need

# of pts prescribed SCO-101:
US: 22k, EU: 70k

Assume per tx price: $60k in US, $42k in EU

Peak revenue:
US: $1.3B, EU: $2.9B

**Price analogs include approved branded CRC agents, including Avastin, Erbitux, and Vectibix*

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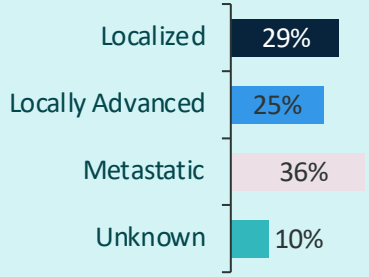
SCO-101 is also uniquely positioned to treat gastric cancer

LARGE UNMET CLINICAL NEED

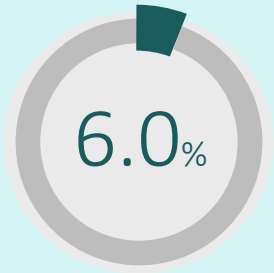
US incident annual cases

26,500

EU: 133,000/year



Stage at Diagnosis



5-year survival rate, stage IV disease

ABCG2 Expression and Relevance in Gastric Cancer

- ABCG2 is expressed in gastric cancer tissues & overexpressed compared to adjacent non-malignant tissue
- ABCG2 is correlated to lower OS or poor clinical outcome
- Inhibition of ABCG2 can positively modulate gastric cancer cells to irinotecan and mitoxantrone

SCO-101's Opportunity in Gastric Cancer

Gastric Cancer Opportunity

- Gastric cancer's large unmet need, promising commercial opportunity, and high expression of ABCG2 makes it an appealing indication for Scandion to target as its next solid tumor candidate

Next Steps

- Scandion is currently conducting two research processes to generate pre-clinical data in gastric cancer by utilizing CROs:
  1. Identify gastric cancer cells (cell lines, PDX cells or others) with high/low ABCG2 expression
  2. Conduct proof-of-concept synergy experiments with SCO-101 and SN38

Sources: Wang et al, (2017); Wang et al, (2010); Zhang et al, (2013), Priebsch et al (2006), Woehlecke et al, (2003), Kowalski et al, (2002), Bar-Zeev et al, (2018), Chang et al, (2016), Company Website, Datamonitor Healthcare, Back Bay Analysis, SEER

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Well established Drug substance and drug product manufacturing process and Phase II compliant

Drug Substance (DS) Manufacturing

- Well established manufacturing process and Phase II compliant
- Several GMP manufacturing campaigns successfully completed in >15 kg scale
- Understanding of solid-state properties – salt/polymorph screen & crystallization process
- Specifications for DS established & analytical methods validated
- DS has excellent stability at ambient temperature

Vendor:

Cambrex

Drug Product (DP) Manufacturing

- Well established manufacturing process and Phase II compliant
- Established process for manufacturing of tablets
- Several GMP batches manufactured
- Tablet formulation investigated: Compatibility, tablet prototype screening, scale-up & demo-batch manufacturing, test of significance of agglomerates
- Specifications for DP established & analytical methods validated to Phase II level
- DP has excellent stability at ambient temperature
- Thorough evaluation of commercial suppliers of DP performed and a preferred supplier has been identified

Vendor:

Solural Pharma

Regulatory CMC Considerations

- DS/DP specifications are compliant with EU & US regulations (confirmed by EU regulatory approval and FDA pre-IND feedback)
- DS/DP consistently resulted in batches of quality according to established specifications
- Multiple DS/DP tested with minimal degradation product; low risk associated to impurity or degradation, Nitrosamine impurity pending (required for Phase III)
- SCO-101 is used in ongoing Phase II prg.; approved across multiple states in EU (DK, DE, ES)
- Pre-IND with FDA confirmed the general acceptance of DS/DP specifications

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In 2023 Scandion reported positive preliminary data for both CORIST and PANTAX, with a number of milestones expected in 2024

H1 2023

PANTAX
Topline data from phase Ib ✓

H2 2023

CORIST
Final analysis of part 2 ✓

IP
New CoM patent ✓

Pre-clinical
Gastric cancer ✓

H1 2024

CORIST
Part 3 Topline data ✓

CORIST
Part 3 PK and Safety data

CORIST
Part 3 Final data

PANTAX
Phase 1b final results

Financing secured into **2025**

SCANDION ONCOLOGY