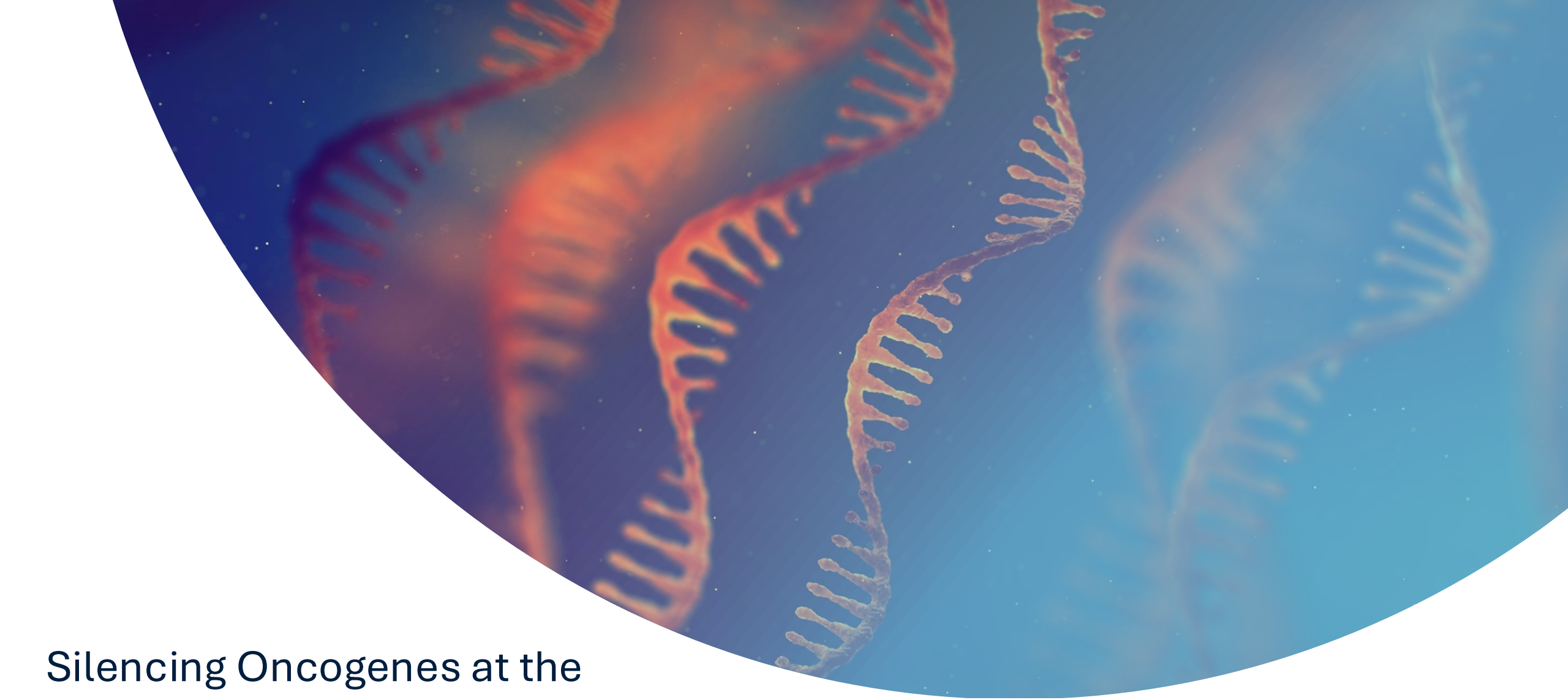


Silencing Oncogenes at the Level of Gene Expression

Corporate Presentation August 2025

Nasdaq: SLXN

Silexion Therapeutics

Forward-Looking Statements

The statements contained in this presentation that are not purely historical are forward-looking statements. Our forward-looking statements include, but are not limited to, statements regarding our or our management team's expectations, hopes, beliefs, intentions or strategies regarding the future. In addition, any statements that refer to projections, forecasts or other characterizations of future events or circumstances, including any underlying assumptions, are forward-looking statements. The words "anticipate," "believe," "continue," "could," "estimate," "expect," "intends," "may," "might," "plan," "possible," "potential," "predict," "project," "should," "would" and similar expressions may identify forward-looking statements, but the absence of these words does not mean that a statement is not forward-looking. Forward-looking statements in this presentation may include, for example, statements about:

- the future performance of the Company, including Silexion's projected timeline for regulatory approvals of its product candidates; and
- the Company's future plans and opportunities.

The forward-looking statements contained in this presentation are based on our current expectations and beliefs concerning future developments and their potential effects on us. There can be no assurance that future developments affecting us will be those that we have anticipated. These forward-looking statements involve a number of risks, uncertainties (some of which are beyond our control) or other assumptions that may cause actual results or performance to be materially different from those expressed or implied by these forward-looking statements. These risks and uncertainties include, but are not limited to, the items in the following list:

- Silexion is a development-stage company and has a limited operating history on which to assess its business;
- Silexion has never generated any revenue from product sales and may never be profitable;
- The approach Silexion is taking to discover and develop novel RNAi therapeutics is unproven for oncology and may never lead to marketable products;
- Silexion does not have experience producing its product candidates at commercial levels, currently has no marketing and sales organization, has an uncertain market receptiveness to its product candidates, and is uncertain as to whether there will be insurance coverage and reimbursement for its potential products;
- Silexion may be unable to attract, develop and/or retain its key personnel or additional employees required for its development and future success;
- Silexion may not succeed at maintaining its listing on Nasdaq; and
- Additional factors relating to the business, operations and financial performance of Silexion.

Should one or more of these risks or uncertainties materialize, or should any of our assumptions prove incorrect, actual results may vary in material respects from those projected in these forward-looking statements. We undertake no obligation to update or revise any forward-looking statements, whether as a result of new information, future events or otherwise, except as may be required under applicable securities laws.

Silexion Therapeutics

Silexion: Silencing Cancer-Driving Proteins Before They Are Produced

KRAS-Focused RNA Interference Platform

Mutated KRAS proteins are the most prevalent cancer drivers:
92% pancreatic cancer
49% colorectal cancer
35% non-small-cell-non-squamous lung cancer

They are well-validated targets, as KRAS inhibitor products represent a multi-billion dollar market opportunity, despite limitations in side effects, resistance and efficacy

Instead of targeting mutated KRAS proteins after they are formed (the approach of our competitors), Silexion's siRNA technology silences their expression at the outset, offering a potentially safer and more durable approach

Silexion Therapeutics

Silexion's Phase 2 Proof-of-Concept (POC) Paves the Way for Late-Stage Clinical Development

Promising Clinical Data in KRAS-Driven Locally Advanced Pancreatic Cancer

Compelling Preclinical Results for Second Generation Product

First generation, KRAS mutation G12D/G12V "Loder" silencing RNA (siG12DLoder siRNA)

- Completed POC- Phase 2 clinical trial in locally advanced pancreatic cancer (LAPC), shows strong trend for 9.3 months improvement in overall survival with siG12DLoder + SoC chemo vs. SoC chemo alone

Second generation, pan-KRAS mutation silencing siRNA "SIL204"

- **Significantly improved product stability and delivery while providing a broader gene silencing activity**
- Successful preclinical results support dual-route strategy to administer SIL204 both intratumorally targeting primary tumor and subcutaneously targeting metastases
- Key preclinical studies demonstrate SIL204's pan-KRAS anti-tumor activity in pancreatic, colorectal and lung cancers
- Oncogene silencing expected to **overcome tumoral resistance observed with KRAS inhibitors**
- Phase 2/3 clinical trial, planned Q2, 2026, using integrated approach administering systemic (subcutaneous) and intratumorally
- Strong IP protection expected until 2043 plus extension up to ~2048

Silexion Therapeutics

Silexion is Positioned to Develop the Most Advanced Oncogene Silencing Therapy

Late-Stage Company with Advanced Stable siRNA Asset

- NASDAQ listing – ticker SLXN
- Management team experienced with late-stage development
- Validated cancer target in mutated KRAS; KRAS-driven cancers represent large potential and need for oncogene silencing therapy in pancreatic, GI and lung cancers
- Late-stage pan-KRAS silencing therapy, GMP-grade SIL204 manufactured with FDA/EU-acceptable partner, Q1, 2026 ready for Ph2/3 clinical trial

Silexion Therapeutics

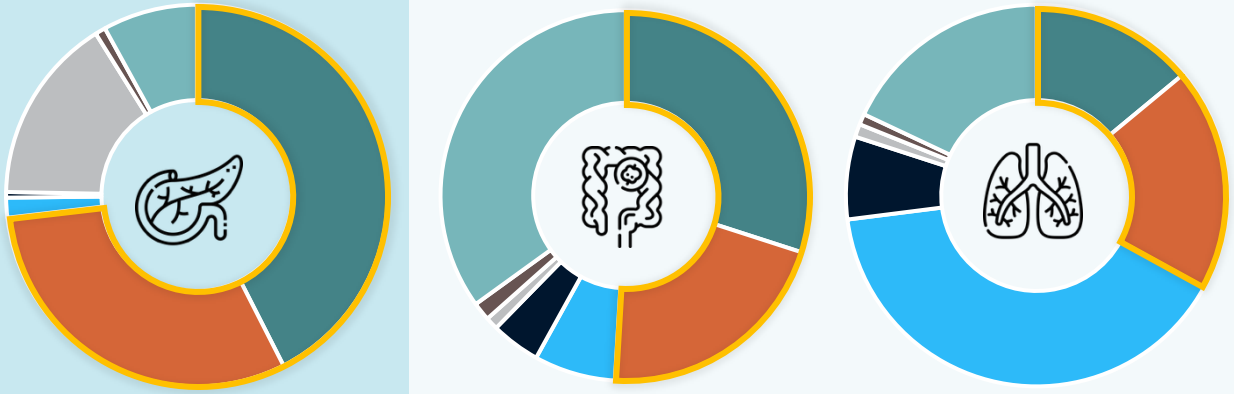
Targeting Mutated KRAS by Silencing RNA

Silexion Therapeutics

KRAS Oncogene is a Validated Target for Numerous Cancers

Most Common Types of KRAS Mutations Across Cancers

	Pancreatic Ductal Adenocarcinoma (PDAC)	Colorectal Cancer (CRC)	Non-squamous Non-Small-Cell Lung Cancer (Non-sq NSCLC)
% KRAS mutations	92%	49%	35%
Incidence USA+EU	~200K	~500K	~400K



KRAS is the most common oncogenic gene driver in human cancers with gastrointestinal cancers having high percentages of KRAS G12D/V mutations

Lee, J.K. et al. NPJ Precis Oncol. 2022;6(1):91.

Silexion Therapeutics

Pancreatic Cancer Among Highest Mortality Rates of Any Major Cancers

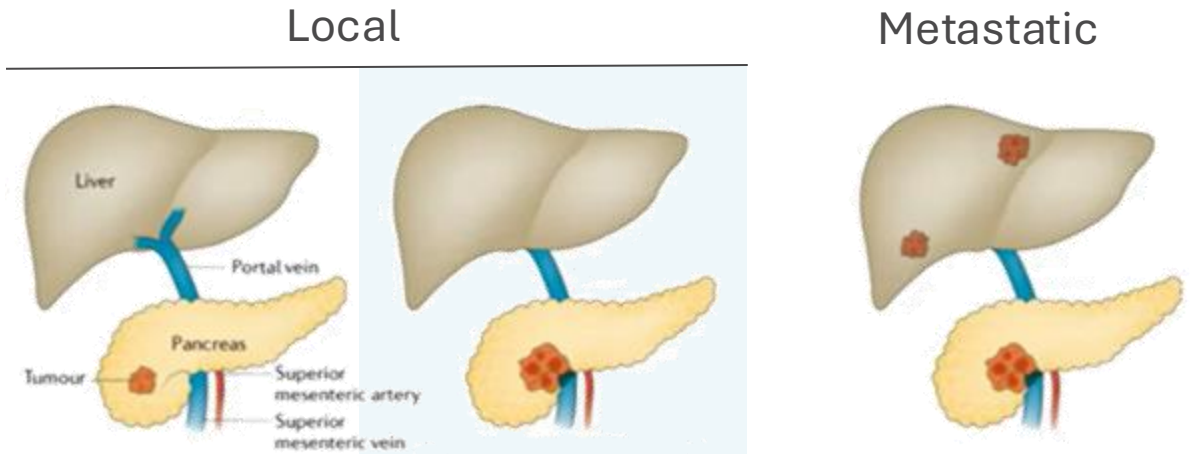
3rd leading cause today in the U.S.[2]

2nd leading cause by 2030[2]

12.8% 5-year relative survival (2014-2020) is one of the poorest in the U.S.[3]

Median overall survival for non-resectable PC populations is 14-17 months[4]

Types and Prevalence of Pancreatic Cancer[4,5]



At diagnosis:	**10-20% resectable**	**30-40% LAPC + BRPC**	**50-60% metastatic**

There are no effective treatment options for our intended indication LAPC

BRPC=borderline resectable pancreatic cancer; LAPC = locally advanced pancreatic cancer.

1. Bray F, et al. *CA Cancer J Clin.* 2024;74(3):229-263. 2. Hirshberg Foundation for Pancreatic Cancer Research. Pancreatic cancer Facts. https://pancreatic.org/pancreatic-cancer/pancreatic-cancer-facts. 3. National Cancer Institute. Cancer Stat Facts: Pancreatic Cancer. https://seer.cancer.gov/statfacts/html/pancreas.html. 4. Gemenetzis G, et al. *Ann Surg.* 2019;270(2):340-347. 5. Kleeff J, et al. *Nat Rev Dis Primers.* 2016;2:16022.

Silexion Therapeutics

Focused Pipeline to Address KRAS-driven Solid Tumor Localized Cancers

Program	Indication	Setting	Discovery	Preclinical	Phase 1	Phase 2	Phase 3	Status/ Anticipated Milestone
LODER siG12D + KRAS amplify with extended release PLGA delivery system	Locally advanced pancreatic cancer	Adjunct to chemotherapy	Phase 2 Completed					Phase 2 completed: observed 9.3 months improvement with LODER over SOC. Continue development of SIL204.
Current Focus: Second generation SIL204								
SIL204 (Integrated treatment regimen) KRAS G12D/V + KRAS amplify	Locally advanced pancreatic cancer	Adjunct to chemotherapy						Q1 2026: CTA submission in E.U. for Phase 2/3 1H 2026: Initiate Phase 2/3
	Colorectal cancer	Adjunct to chemotherapy						Preclinical ongoing
SIL204 adjunct to CPIs	KRAS-driven cancers	Adjunct to CPI+ chemotherapy						Preclinical ongoing

CTA=clinical trial application; SOC=standard of care.

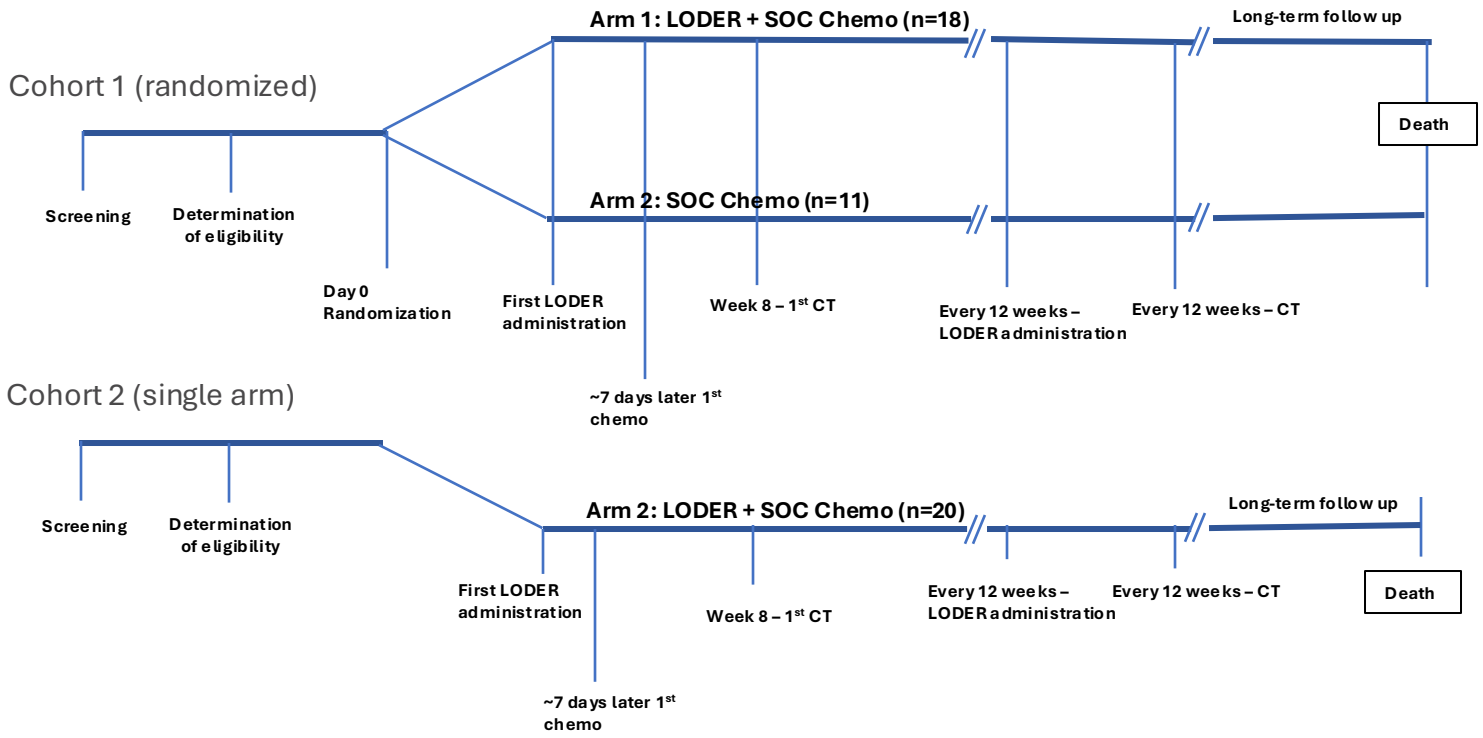
LODER (First generation siRNA)

Phase 2 Clinical Trial Data

Silexion Therapeutics

Phase 2 Proof of Concept Trial of Loder Completed in 2023

Two-part, open label, study of LODER + SoC chemotherapy vs SoC chemotherapy alone across the U.S. and Israel in patients with non-resectable pancreatic cancer



Key inclusion criteria

- Non-resectable without signs of metastasis
- ECOG Status ≤ 1
- Enrollment independent of KRAS mutation status

Treatment

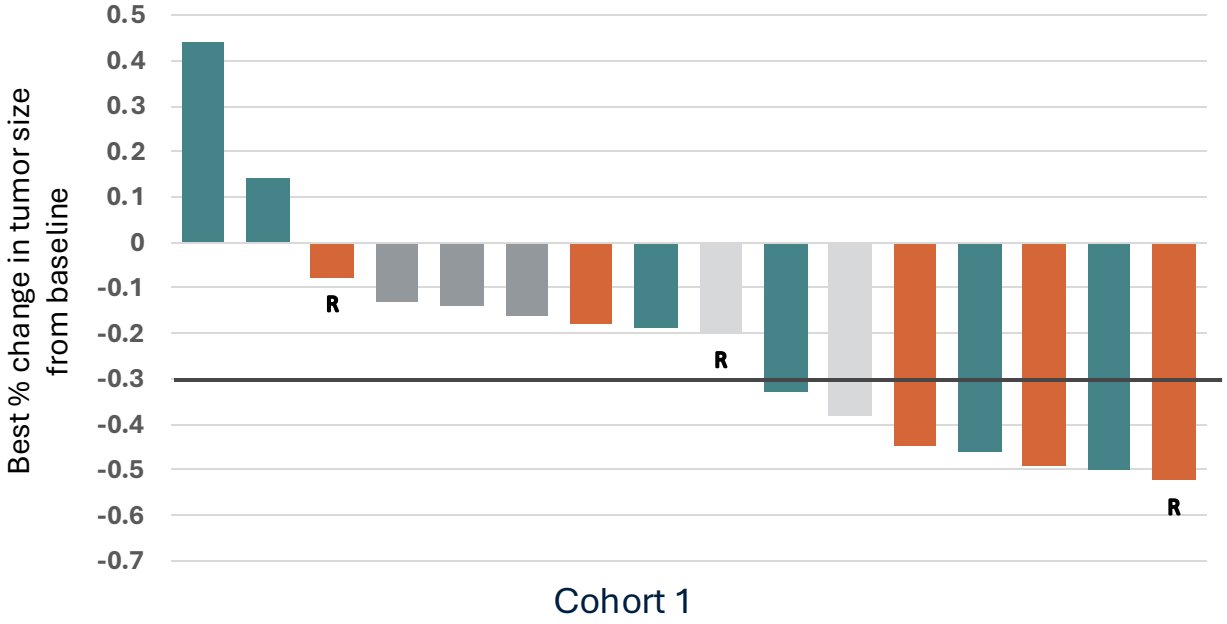
- Range of treatment cycles 1 to 5
- Safety from ~370 Loder injection cycles

Endpoints

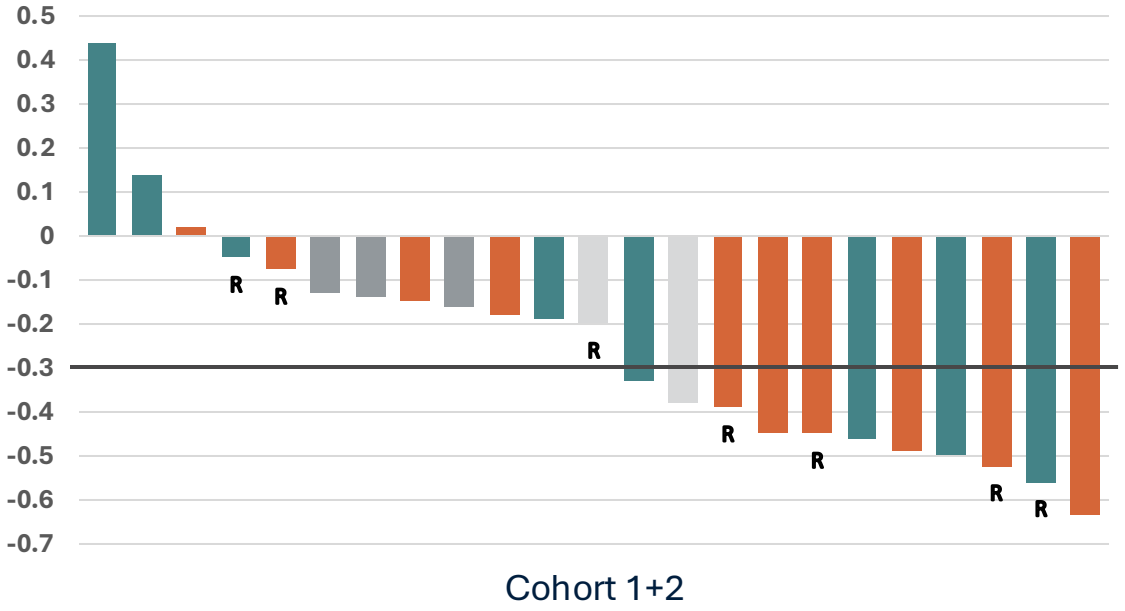
- Overall survival (OS)
- Response rate (RR, RECIST v1.1)
- Safety, Tolerability

SoC=standard of care.

Loder Treatment Led to Robust Objective Response Rate in Patients with LAPC Harboring G12D/V Mutations*



Cohort 1



Cohort 1+2

G12D G12V

Chemo:

Loder:

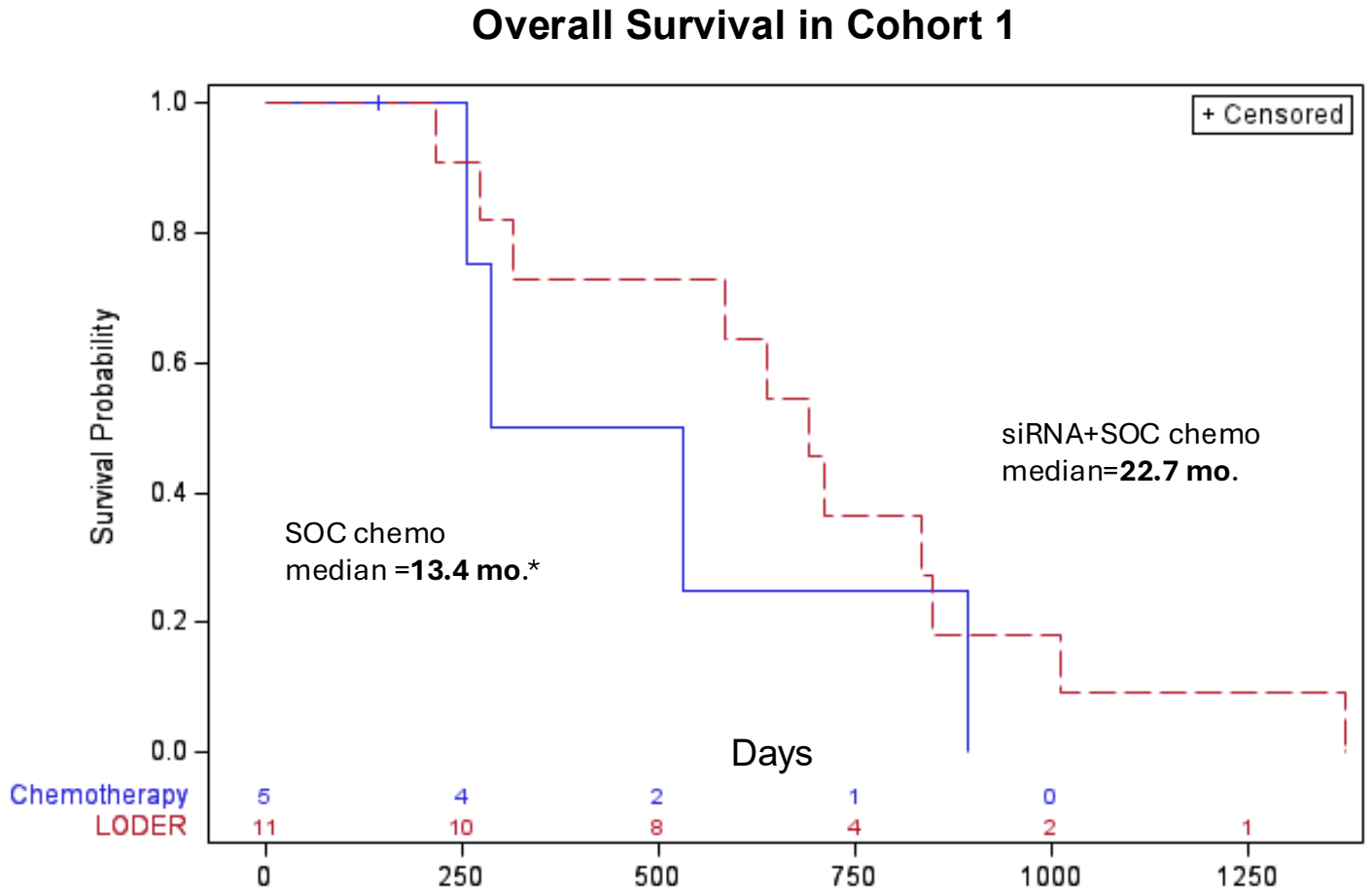
R = Non-resectable tumor becomes resectable

	Cohort 1 LODER+Chemo	Cohort 1+2 LODER+Chemo	Chemo
% Response	55 (6/11)	56 (10/18)	20 (1/5)
% Response + becoming resectable	64	67	40

LAPC=locally advanced pancreatic cancer.
*Overall response rate was confirmed by RECIST 1.1 of the target tumor, as analyzed by sites.
Bar curves below the solid black line starting at y-axis -0.3 indicates criteria for positive RECIST response.

Silexion Therapeutics

Loder + SOC Chemotherapy Extends Median Survival by 9 Months in KRAS G12D/V Mutations in POC Study



Hazard ratio (HR)=0.59, (95% CI, 0.18, 1.96, p=0.39)

41% lower risk of death at any time compared to SOC chemotherapy

Implications

- **69% improvement in overall survival**
- **Patients live longer with Loder+SOC vs. SOC**
- **Positive proof of concept demonstrating improved survival with siRNA silencing KRAS mutation**

* SoC (Control) OS consistent with recent trials for LAPC (Gemenetzis G, et al. *Ann Surg*. 2019;270(2):340-347).

LODER Was Overall Well Tolerated with an Acceptable Safety Profile

- Combination siG12D-LODER and SoC chemotherapy generally well-tolerated
- All serious TEAEs resolved: 9 (pyrexia x2, abdominal pain, hyperbilirubinemia, pancreas infection x2, sepsis, procedural hemorrhage/presyncope, gastric hemorrhage)
- Two Grade 5 events reported in the siG12D-LODER population, both assessed unrelated to treatment: acute intestinal ischemia and gram negative sepsis.
- No Treatment Emergent Adverse Events (TEAEs) leading to study discontinuation related to LODER treatment
- No meaningful observations in any vital sign parameter nor any physical examination findings in the study
- Independent Drug Safety Monitoring Board (DSMB) Reviews had no safety concerns nor safety restrictions
- In a subset analysis, no measurable amount of siG12D siRNA was detected (<BLQ, 0.25ng/mL) in any plasma samples suggesting low systemic levels

Silexion Therapeutics

SIL204 (Second Generation siRNA)

Silexion Therapeutics

Silexion Innovative Approach Expected to Improve Clinical Outcomes

Overcoming the Limitations of Currently Approved and Investigational Small Molecule KRAS Inhibitors:

- Treatment resistance
- Low tolerability with adverse events such GI side effects (e.g. diarrhea) and rashes and that require special monitoring
- Unmet need in survival and QoL remains

Silexion's Approach

2nd generation siRNA SIL204 optimized for enhanced stability and broader pan-KRAS activity

Pan-KRAS silencing inhibits expression of oncogenic KRAS before it is active

SIL204 designed for better efficacy and safety profiles with less resistance

Integrated Treatment: Intratumoral delivery for the primary tumor and systemic (s.c.) administration for micrometastases

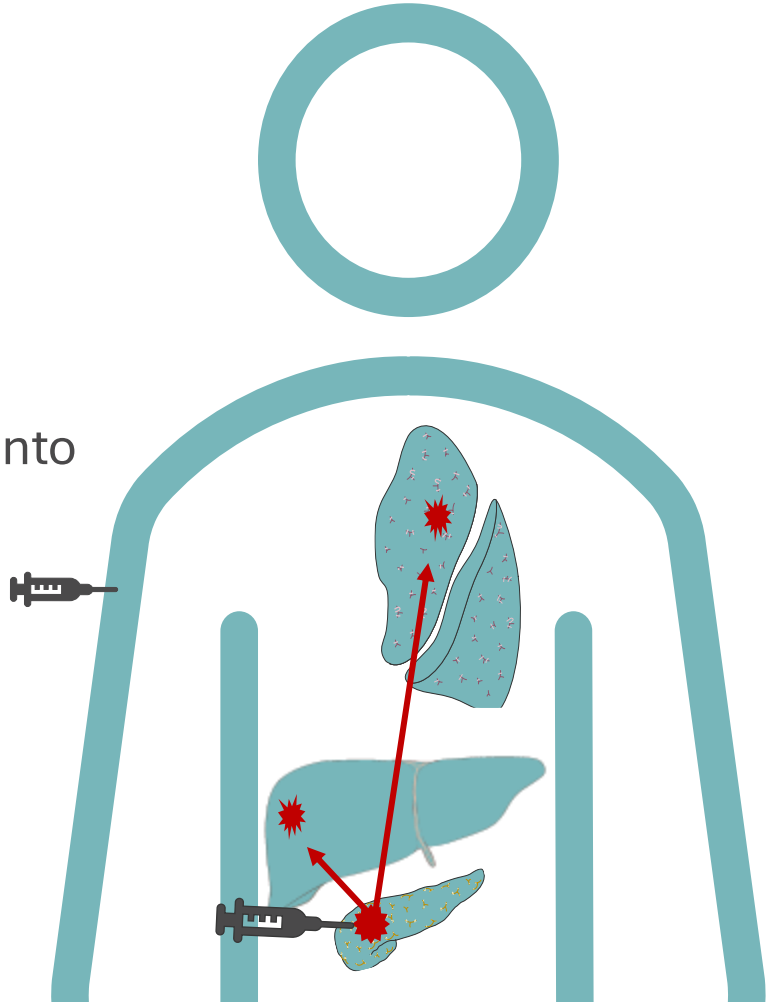
s.c. = subcutaneous

Dual Administration of SIL204 Designed to Eradicate Primary Tumor and Metastases

Integrating systemic (s.c.) with intra-tumoral administration complement the effect of SIL204 on the primary pancreatic tumor with eradication of migrating metastatic cells

Metastatic invasions into liver, lung targeted by **systemic SIL204**

Primary pancreatic tumor targeted by **intratumoral SIL204**

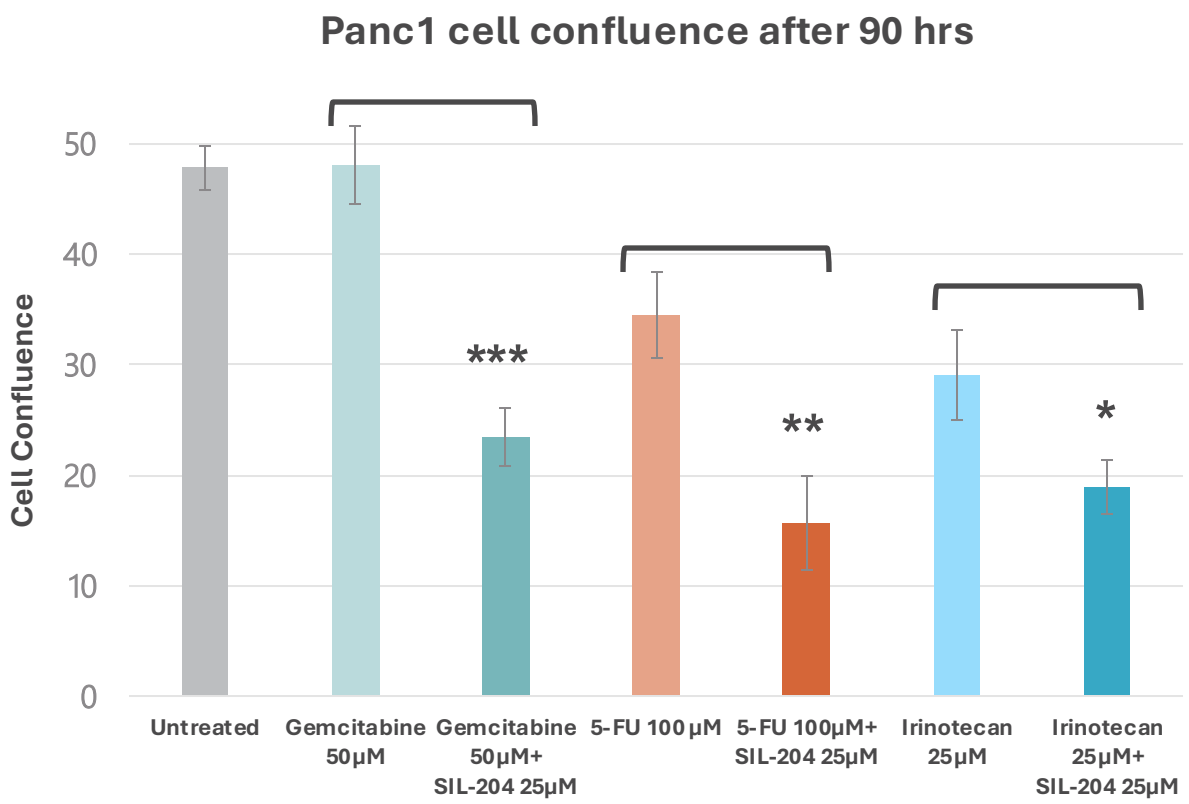


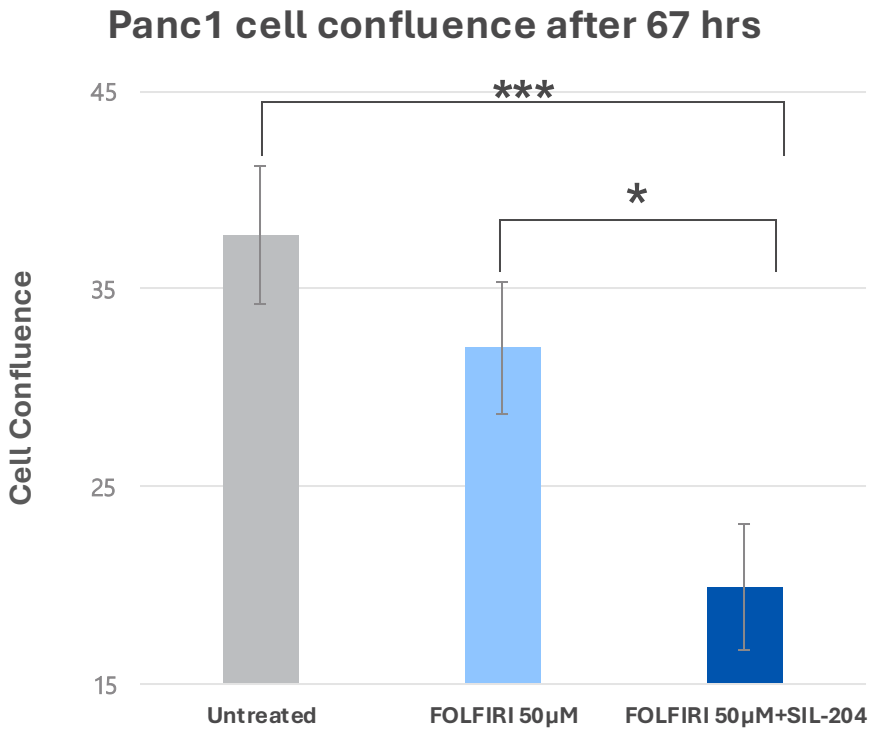
- Pancreatic cancer mortality driven by early and rapid metastases
- Systemic (SC) administration of SIL204 aims to silence secondary tumors as they are cleared from primary tumor (micrometastases) and those in the pre-metastatic niche before their stromal “barrier” is fully formed
- Intratumoral administration of SIL204 aims to silence primary tumor and prevent metastatic “shedding”; overcoming the tumor barriers

Silexion Therapeutics

SIL204's Anti-Tumor Activity Synergistic with Pancreatic Cancer SOC Chemotherapy

Preclinical study measuring confluence of the human pancreatic cell line Panc-1 containing a G12D KRAS Mutation





*p<0.05; ** p<0.005; ***p<0.0005

Silexion Therapeutics

SIL204 In Silico Analysis: Highly Specific for KRAS vs Other RAS or Off-Target Sites

- **Approach**
  - SIL204 must bind the KRAS mutation mRNA for activity
  - Negative values indicate binding.
  - Negative Gibb's free energy changes (ΔG) of -20s and 30s indicates strong, favorable binding
- **KRAS ^{G12V} -31.8 kcal/mole**
- **No Off-Target Active Anti-sense Binding**
- **Low risk for side effects.**
- **No effect on regulatory RNAs**

Conclusions

- **SIL-204 will silence the intended target KRAS mutations, but low risk for any effect with other proteins besides KRAS, with implications for better safety**
- **HRAS and NRAS very unlikely affected, continuous endogenous RAS activity**

Analysis conducted by Dr. James McSwiggen, McSwiggen Biotech Consulting LLC

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SIL204 Highly Effective with Broad Inhibition Across Human KRAS Mutations at Sub-nanomolar Concentrations

SIL204 maintains and expands the silencing activity of the siG12DLoder

Model is a co-transfection setup where human KRAS is transfected in mouse Hepa1-6 cells with Dual-Glo reporter plasmids.

Mutation	Negative siRNA Control	WT KRAS	KRAS G12D	KRAS G12V	KRAS G12C	KRAS G12R	KRAS Q61H*	KRAS G13D*
IC_{50} (nM)		0.16	0.19	0.44	0.47	0.59	0.24	0.37
MAX Inhibition (%)	0-7	91	90	80	73	71	88	88

IC_{50}=half-maximal inhibitory concentration.
*G13D and Q61H tested in separate studies from the G12 mutations and wild type (non-mutated).
Negative siRNA control collected over various studies

Silexion Therapeutics

SIL204 Inhibits Human Tumor Cell Line Growth at Concentrations Achievable with the Planned Clinical Trial Human Equivalent Doses

- SIL204 efficacy in tumor cells from 4 different human cancer cell lines: pancreatic; colon; lung and cholangiocarcinoma can be shown in a cell proliferation (CellTiter-Glo (GTC)) assay

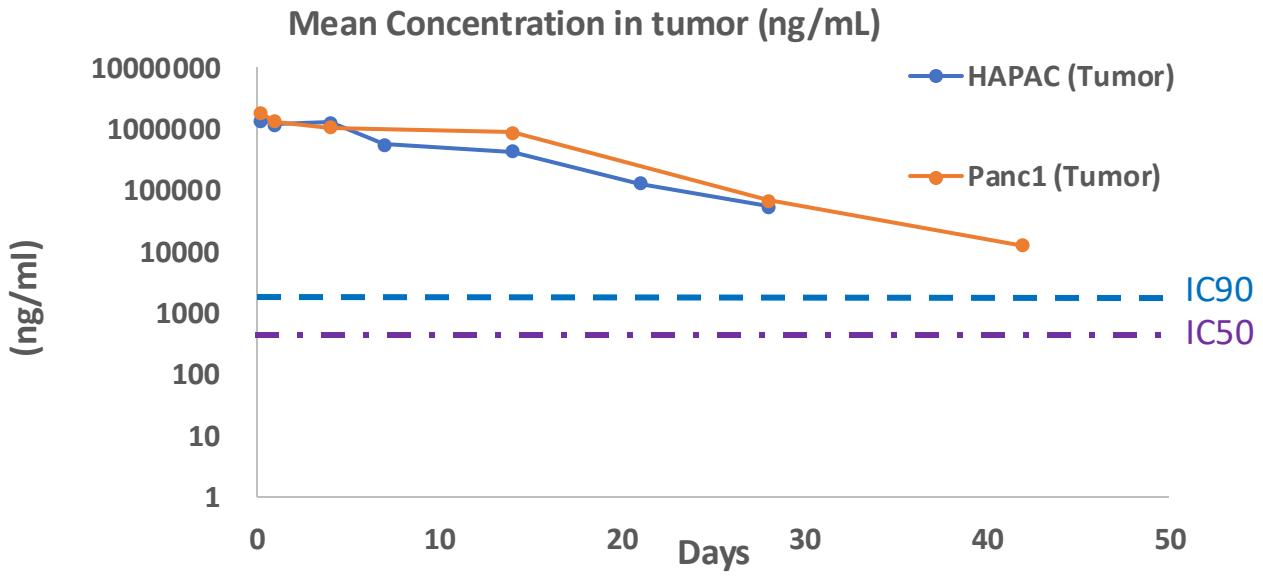
Cell line	IC_{90} (nM)	IC90 (ng/mL)
A427 (Lung) G12D	**70**	**1,079**
PK59 (Panc) G12D	**163**	**2,496**
GP2D (Colon) G12D	**56**	**852**
HS766T (Panc) Q61H	**124**	**1,907**
AVG	**103**	**1,583**

- Results support the pan-KRAS silencing activity observed in transfected hepa cells
- IC90 is the concentration for 90% inhibition of tumor cell growth, IC50 is the concentration to achieve 50% inhibition
- SIL204 growth inhibition effect for all tumor lines: IC50 = 613 ng/mL, IC90 = 1,583 ng/mL

Intratumor PK Data Confirm Durable SIL204 Exposure Above GTC 100% Inhibition Target (IC90)

Human pancreatic tumors in mice (2 separate lines) administered by intratumoral injection 3mg SIL204/tumor (approximate human equivalent dose (HED) planned in the clinical trial) showed that the intratumoral concentrations are much higher than IC90 for a prolonged period

- Tumor levels from 3mg SIL204/tumor were over 1,000,000 ng/mL after injection (600 folds higher than IC90) and declined slowly with half life of about 5 days
- Effective intratumoral concentrations of SIL204 over planned dosing period should be maintained
  (- - -) represents the IC90 growth inhibition concentration

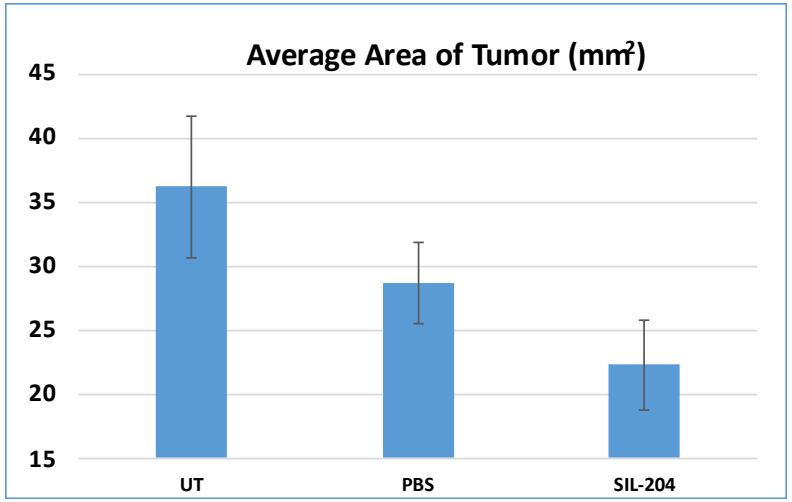


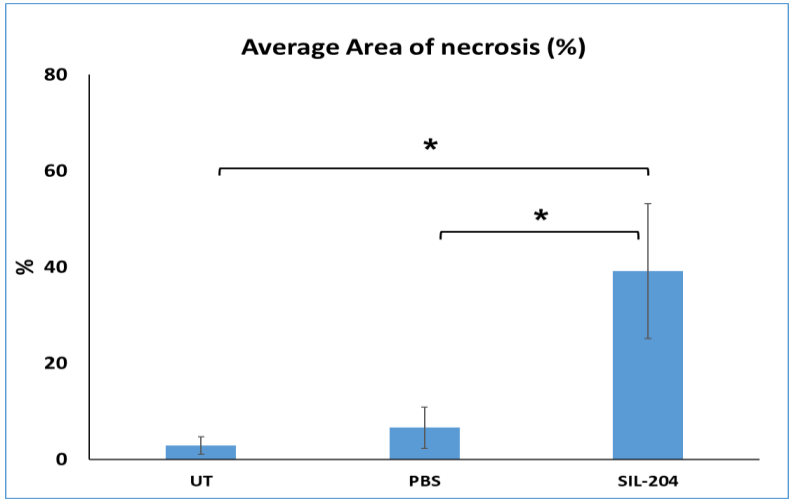
PK demonstrates durable and effective intratumor exposure supporting planned Phase 2/3 dosing

Silexion Therapeutics

Intratumoral HED SIL204 Shrinks Implanted KRAS G12V tumors in Mice

- Intratumoral SIL204 (3mg/tumor, planned P2/3 ~HED) reduced tumor size in nude mice bearing KRAS G12V xenograft
- Histological examination revealed a higher degree of tumor necrosis than in the untreated control group
- Results further support the dose to be used to effectively treat the primary tumor in LAPC patients

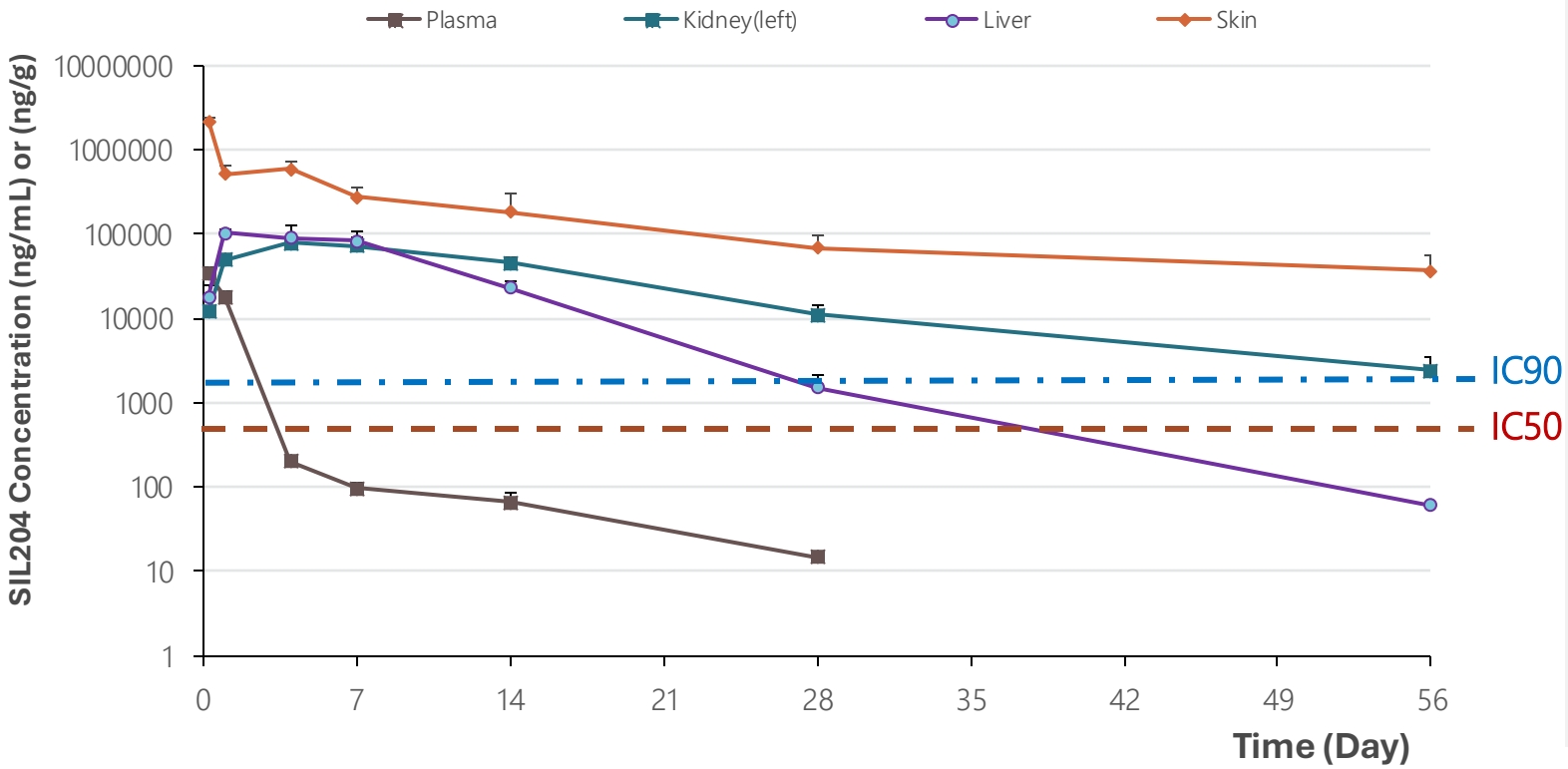




- * p<0.05
- PBS = Control tumors inoculated with PBS buffer; S.C. = sub cutaneous; UT = untreated control.

S.C. SIL204 PK Demonstrates Persistent Effective Exposure at Major Metastatic Site

Potential for durable efficacy of siRNA for treating micrometastases with monthly s.c. injections

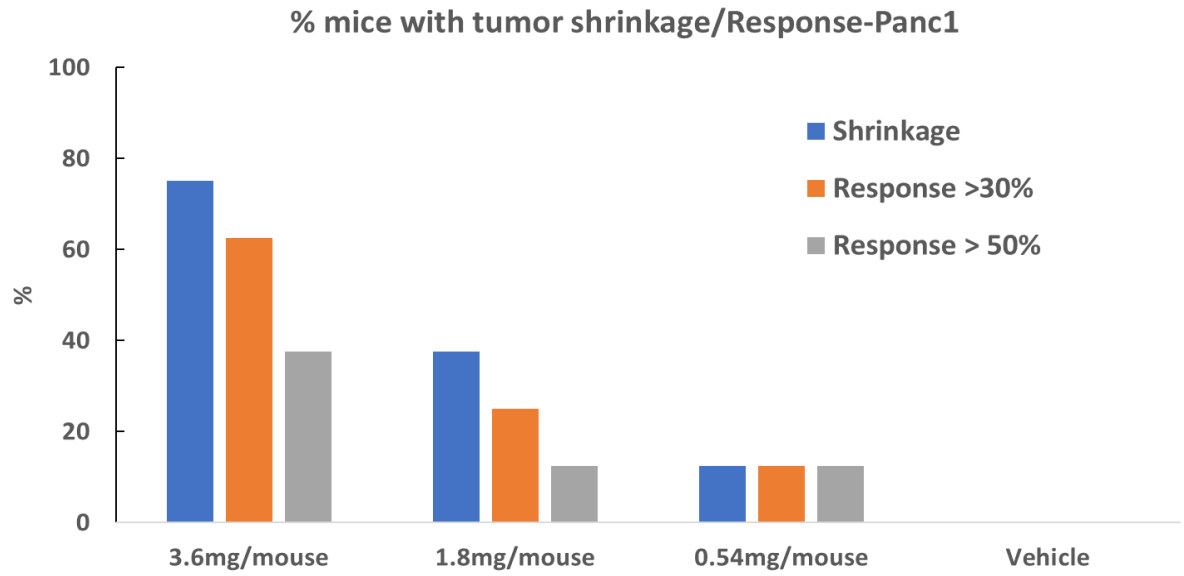


- PK Studies in rats 10 mg/rat (~HED 400mg, the planned clinical s.c. dose) showed that s.c. injection slowly releases SIL204
- SIL204 slowly distributes to other tissues, primarily to the liver (main site of metastasis, purple) and kidney (final excretion organ)
- SIL204 levels in the liver are maintained above IC90 (1583 ng/mL) through the proposed dosing period
- Minimal plasma exposure post administration

S.C. SIL204 Demonstrates Positive Dose Response in Tumor Volume

- Nude mice with orthotopic PANC-1 (KRAS G12D, luciferase) tumors showed dose-dependent tumor inhibition after subcutaneous (SC) SIL204
- Three different definitions of a positive response were evaluated, all three showed a similar dose response
- A human equivalent dose of SIL204 (0.54mg/mouse) used in this experiment is the approximate middle dose planned in the clinical trial. The clinical trial high dose is 3x the mid-dose

Responder analysis to SIL204 following SC injections in an orthotopic model.

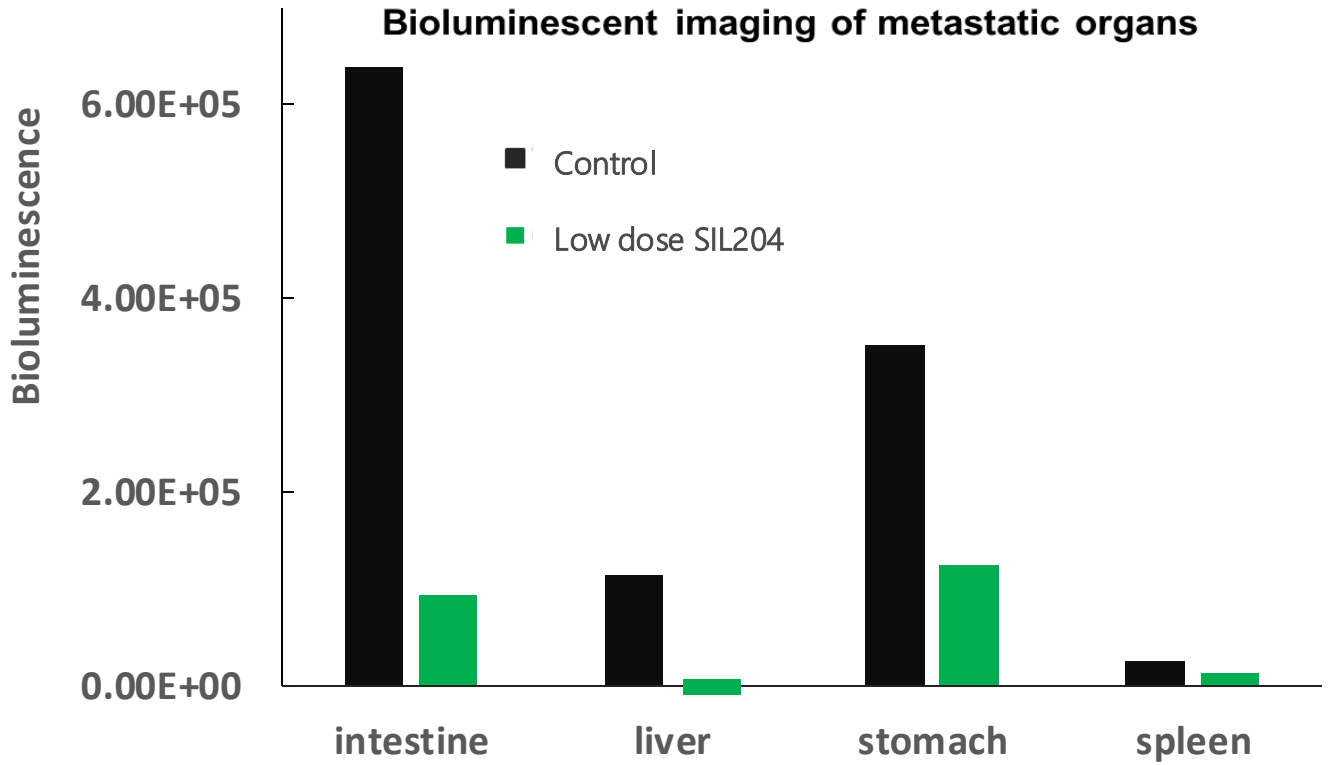


30% response is the standard rate used in solid tumor clinical trials (RECIST v1.1) to determine Objective Response Rates criteria

Subcutaneous SIL204 Shrinks Distal Metastases in Animal Model

Subcutaneous SIL204 Proved Effective in Mouse Metastatic Pancreatic Orthotopic Models
Animal model represents human equivalent dose of SIL-204

- Reduction in bioluminescence indicates decreased viable tumor burden, consistent with SIL204 treatment effect (green bars)
- The human equivalent of the mouse dose used (0.54mg/mouse, low dose) is a planned clinically relevant dose
- considerable decrease in the marker for the tumor cells (green bars) in organs where metastasis occurs clinically (e.g. liver and intestine)



* Human tumor cell line Panc-1 harboring KRAS G12D mutation

Late-Stage Clinical Development Roadmap for SIL204 in Pancreatic Cancer

2023	2024	H1 2025	H2 2025	H1 2026	H2 2026
✓ Phase 2 Clinical proof of concept for prototype siRNA (Loder) in LAPC with trend survival, ORR	✓ Initial Scientific Advice, German regulatory authorities	✓ Scientific Advice, German regulatory authorities. Received “buy-in” for integrated regimen (s.c./IT), and plans to proceed to Phase 2/3 trial ✓ Clinical Advisory Board buy-in to trial design	• German regulatory scientific advice • Submission Israel Health authorities ✓ • Entered national phase of SIL204 patent submissions	Submit CTA in E.U. for Phase 2/3	• Complete Segment 1 of Phase 2/3 (selection 2 best doses) • Expand to Segment 2, randomized, s.c. doses blinded • Pre-IND meeting • Submit IND for expansion Phase 2/3 to USA/additional EU, etc
	✓ Optimization of siRNA to improve efficacy and stability	✓ Toxicology: 2 species to initiate clinical trial, in-life completed; Histology ongoing ✓ GMP production API (SIL204) for Segment 1, Phase 2/3	✓ • s.c. formulation stability (initial) • GMP production s.c. formulation • Toxicology to initiate trial	Initiate Phase 2/3, LAPC Germany/Israel	

✓ Indicates completed activity. Unmarked activities to be performed or completed

Strong Intellectual Property Protection

Submissions	Filing – Term-claims
Patents supporting SIL204 • Compositions for Inhibition of Kras Expression and Methods of Use Thereof. Main patent protection for SIL204 Includes composition of matter claims and method of use.	• National phase submissions pending in: USA; EU; Japan; China; Brazil; India; Australia; Canada; Korea; Israel from PCT (PCT/ IL2023/051276). Priority from December 14, 2023 and expected protection 2043 plus estimated extension up to 2048) USPTO used as PCT search with positive review
• Compositions for inhibition of KRAS expression and treatment regiments therewith. Patent submission for additional methods of use and combination therapies for SIL204 e.g. CPIs	• Provisional US63/745/358 filed December 2024 (expected protection 2046 plus extension)
• siRNA against KRAS G12x for regional perineural invasion or pain associated with a solid tumor. Broader siRNA claims which can include SIL204 in mechanism important for pancreatic cancer metastases	• US17/611927 ; EU20731233.1 priority date May 17, 2020 Pending USA/EU, expected term till 2040 plus extension
Patents supporting siG12DLoder	• Granted patents: 6 in USA and 2 EU. Protection until ~2030 plus extension

Exclusivity can be extended under country-specific regulatory-based extension rules.

Silexion Therapeutics

Silexion Positioned to Develop the Most Advanced Oncogene Silencing Therapy

KRAS-Focused RNA Interference Platform

Instead of targeting mutated KRAS proteins after they are formed (as competing technologies do) Silexion's siRNA technology silences their expression at the outset, offering a potentially safer and more durable approach

Promising Clinical Data in KRAS-Driven Locally Advanced Pancreatic Cancer

Compelling Preclinical Results for Second Generation Product

First generation siG12DLoder siRNA Completed POC- Phase 2 clinical trial in LAPC
Demonstrated improvement in overall survival. Expected that siRNA silencing mechanism of the oncogene will **overcome the tumoral resistance mechanism observed with KRAS inhibitors**

Second generation, silencing siRNA "SIL204," significantly improved product stability and delivery while providing a broader gene silencing activity.
Preclinical results demonstrate SIL204's pan-KRAS anti-tumor activity in pancreatic, colorectal and lung; supports dual-route strategy to administer SIL204 both intratumorally and s.c. to target primary tumor and metastases.
Phase 2/3 clinical trial planned Q2, 2026; using integrated administration approach, s.c. plus intratumor

Late-Stage Company with a Stable Advanced siRNA Asset

NASDAQ listed with experienced management team
Validated cancer target in mutated KRAS
Late-stage pan-KRAS silencing therapy. SIL204 API already manufactured by under GMP and ready for Phase 2/3 clinical trial

Silexion Therapeutics

World-Renowned Expert Scientific Advisory Board

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Milind Javle, MD
The University of Texas & MD Anderson Cancer Center, Houston, TX
Professor, Department of Gastrointestinal (GI) Medical Oncology, Division of Cancer Medicine

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Talia Golan, MD
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Silexion Therapeutics

Highly Experienced Leadership Team

Ilan Hadar, MBA Chairman and Chief Executive Officer

Over 25 years of multinational executive managerial and corporate experience with pharmaceutical and high-tech companies. CEO PainReform (Nasdaq: "PRFX"), CFO and Country Manager Foamix Pharmaceuticals Inc. (Currently Nasdaq: "VYNE")

Mitchell Shirvan, PhD, MBA Chief Scientific and Development Officer

Over 30 years of experience in R&D, innovation and discovery in biotech companies. CEO Macrocure Ltd., Sr. V.P. R&D Foamix Pharmaceuticals Inc. Currently Nasdaq "VYNE"), Sr. Director Strategic Business Planning Teva Pharmaceuticals Industries Inc.

Mirit Horenshtein Hadar, CPA Chief Financial Officer

Over 15 years of corporate finance experience in senior financial positions of public companies and privately held companies, in the pharmaceutical and high-tech industries. CFO Gouzy Israel (Nasdaq: "GAUZ"). V.P. Finance Foamix Pharmaceuticals Inc. (currently Nasdaq"VYNE")

Thank You

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Silexion Therapeutics

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