
MEDIVIR
INNOVATION IN AREAS OF HIGH UNMET MEDICAL NEED
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A pipeline of first-in-class programs targeting patient populations without approved treatment options

PROJECT	PARTNER	DISEASE AREA	PRE-CLINICAL	PH 1	PH 2	PH 3	ON MARKET	FINANCIALS	PROJECT STATUS
IN-HOUSE PROGRAMS									
Fostroxacitabine bralpamide (Fostrox)	In-house development	HCC (mono) HCC (combo)						100% Medivir	▪ Phase 1b/2a combo study completed 2025 ▪ 80 patient randomized phase 2 combo study start near-term
MIV-711	In-house development	Osteogenesis Imperfecta						100% Medivir	▪ Phase 2 clinical PoC study in development
PARTNERED PROGRAMS – NO FURTHER INVESTMENT REQUIRED BY MEDIVIR									
Xerclear	GSK, SYB	Herpes						Royalties	▪ Registration in China
Remetinostat	Biossil	CTCL, BCC, SCC						Royalties & up to $60m in milestones	▪ Out-licensed in Q4 2025 ▪ Phase 2/3 study start
MIV-701 / VBX-1000	Vetbiolix	Periodontal disease in dogs						Royalties & revenue share agreement on Vetbiolix partnering	▪ Randomized phase 2 results during Q4 2026
MET-X	Infex Therapeutics	Critical MBL Infections						Revenue Share Agreement	▪ Phase 1 study start in 2026

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Transformational progress

SEK 45 million directed issue to Carl Bennet AB, enabling MIV-711 clinical development in Osteogenesis Imperfecta, with market opportunity comparable to fostrox in HCC, while strengthening company financial position

FLEX-HCC in advanced primary liver cancer, study preparations with Korean Cancer Study Group continues to progress, all sites selected, including the three largest hospitals

VBX-1000 (MIV-701) initiation of randomized, placebo-controlled study to confirm disease-modifying benefit & unlock blockbuster potential, results expected Q4 2026

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Medivir; Strong track-record in out-licensing

Partnerships to effectively building shareholder value

- 2 approved drugs (Xerclear & Olysio) developed & out-licensed
- 3 first-in-class R&D programs out-licensed
- 2 in-house programs funded through phase 2, data read-outs in 27/28, for additional partnering potential

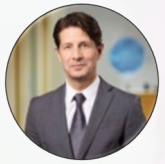
Leadership & board with extensive early & late stage drug development experience

- **CE0 – Jens Lindberg**
  - > 25 years in pharma with focus in Oncology, late-stage development & commercialization
  - Other experience includes interim CEO for Sedana Medical AB and Director Investor Relations at AstraZeneca.

- **CMO – Pia Baumann, MD PhD**
  - Medical & Radiation Oncologist
  - >10 yrs in clinic & academia followed by >15 yrs in global pharma/biotech roles

- **CFO – Patrik Norgren**
  - >20 years experience from CFO and financial management roles across multiple sectors in listed and private companies.

- **CSO – Fredrik Öberg, PhD**
  - >25 yrs experience in cancer research with >50 scientific articles and holds several patents.
  - During the last 10 years focused on industrial drug discovery and development projects in oncology.



- **Chairman of the Board – Anders Hallberg**
  - Master's degree in economics from Lund University
  - >25 years of experience in healthcare investments, including majority owner of HealthInvest Partners AB

- **Dr. Uli Hacksell, PhD, Organic Chemistry**
  - Over 30 years pharma & biotech experience, including 10 years' experience as CEO of publicly owned companies
  - Has served as CEO and as Chairman of the Board at Medivir

- **Dr. Angelica Loskog, PhD, Clin. Immunology**
  - CEO Lokon Pharma & scientific advisor at VC Nexttobe
  - More than 25 year's academic drug development experience within immune oncology

- **Dr. Anna Törner, PhD, Statistics**
  - Broad experience from drug development
  - Founder consulting company SDS Life Science within drug development, regulatory affairs and statistics.

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IN-HOUSE PROGRAMS

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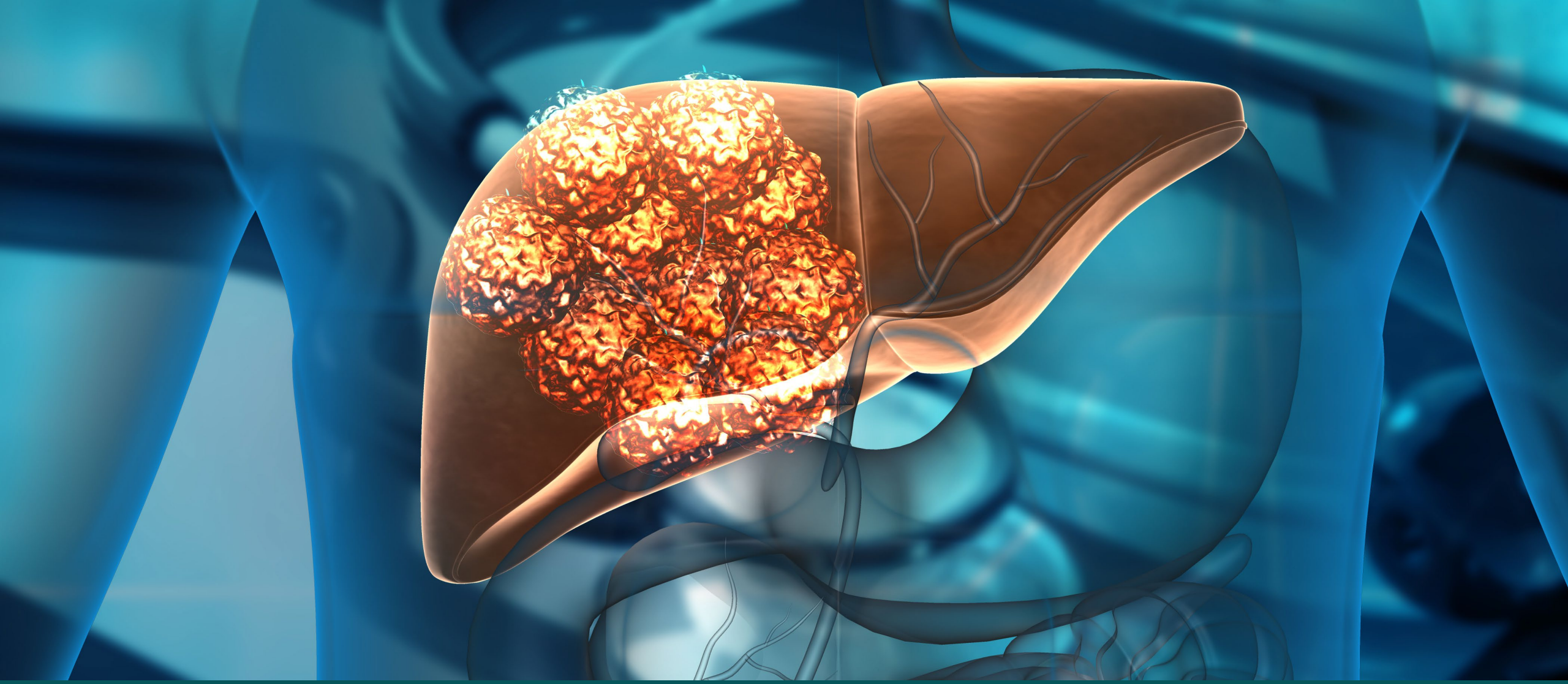

Improving life for advanced liver cancer (HCC) patients

Fostrox – The first oral, liver-targeted treatment for advanced HCC

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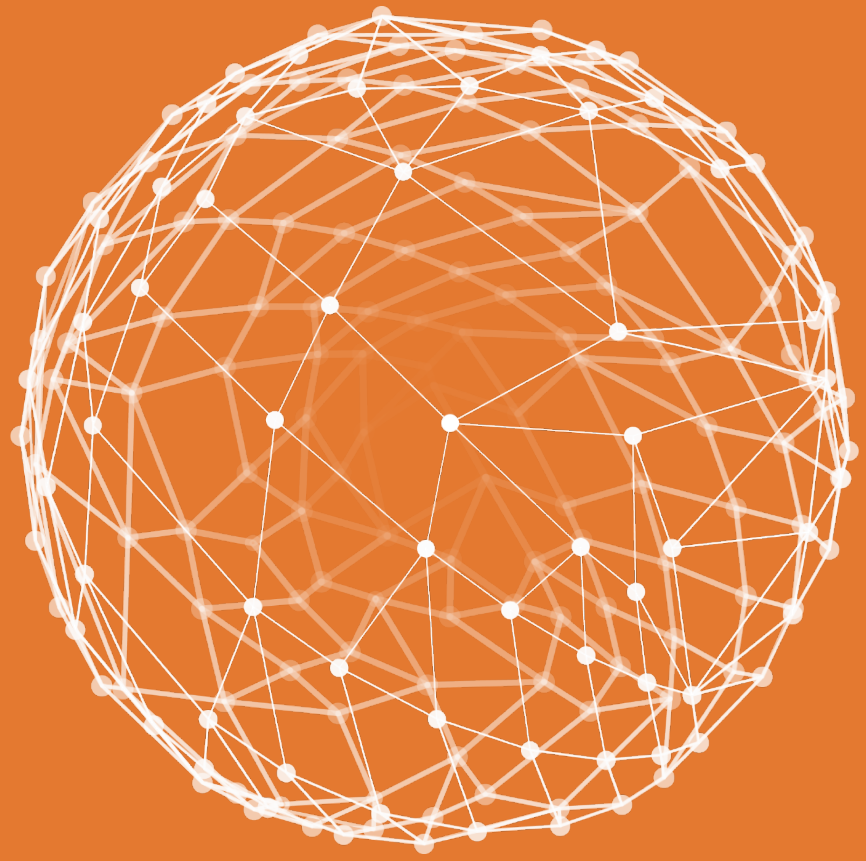
First-to-market opportunity in 2nd line HCC market valued >$2.5bn



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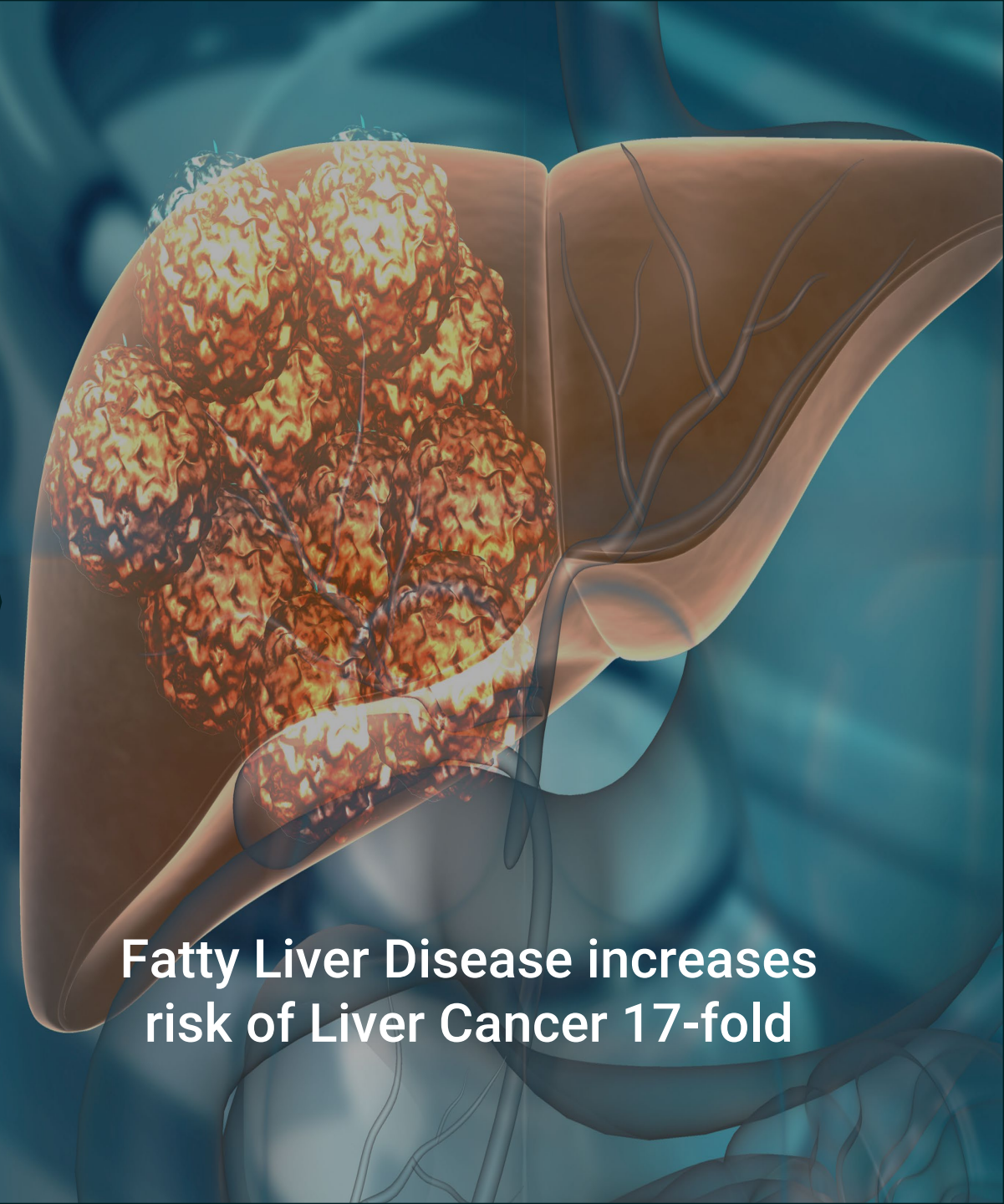
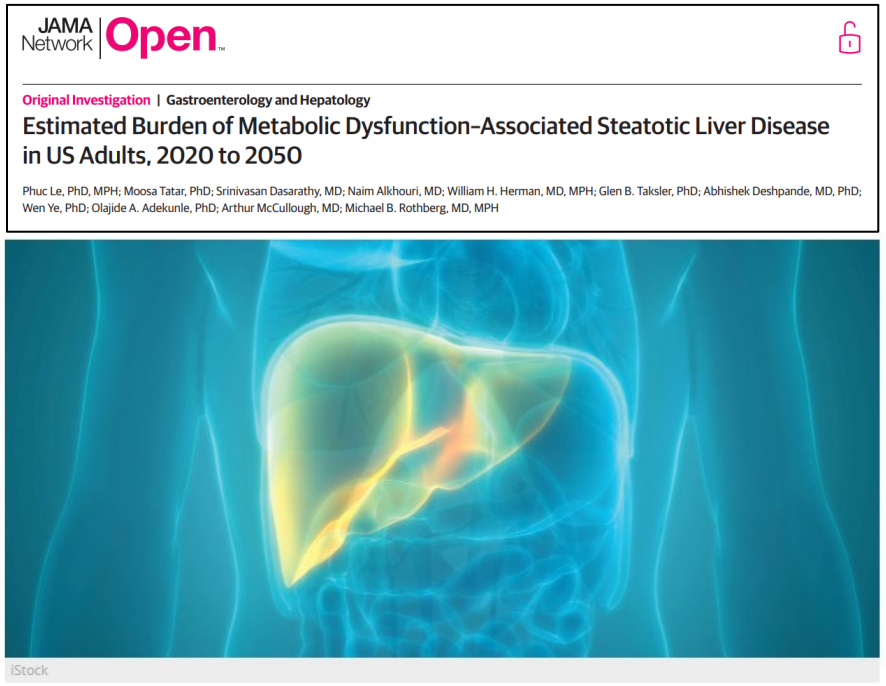
45% of US adults are obese
More than 25% have Fatty Liver Disease



Fatty Liver Disease increases
risk of Liver Cancer 17-fold

Growth in Fatty Liver Disease expected to drive an alarming increase in liver cancer cases[1]



JAMA Network Open

Original Investigation | Gastroenterology and Hepatology

Estimated Burden of Metabolic Dysfunction–Associated Steatotic Liver Disease in US Adults, 2020 to 2050

Phuc Le, PhD, MPH; Moosa Tatar, PhD; Srinivasan Dasarathy, MD; Naim Alkhouri, MD; William H. Herman, MD, MPH; Glen B. Taksler, PhD; Abhishek Deshpande, MD, PhD; Wen Ye, PhD; Olajide A. Adekunle, PhD; Arthur McCullough, MD; Michael B. Rothberg, MD, MPH

iStock

SCIENCE NEWS

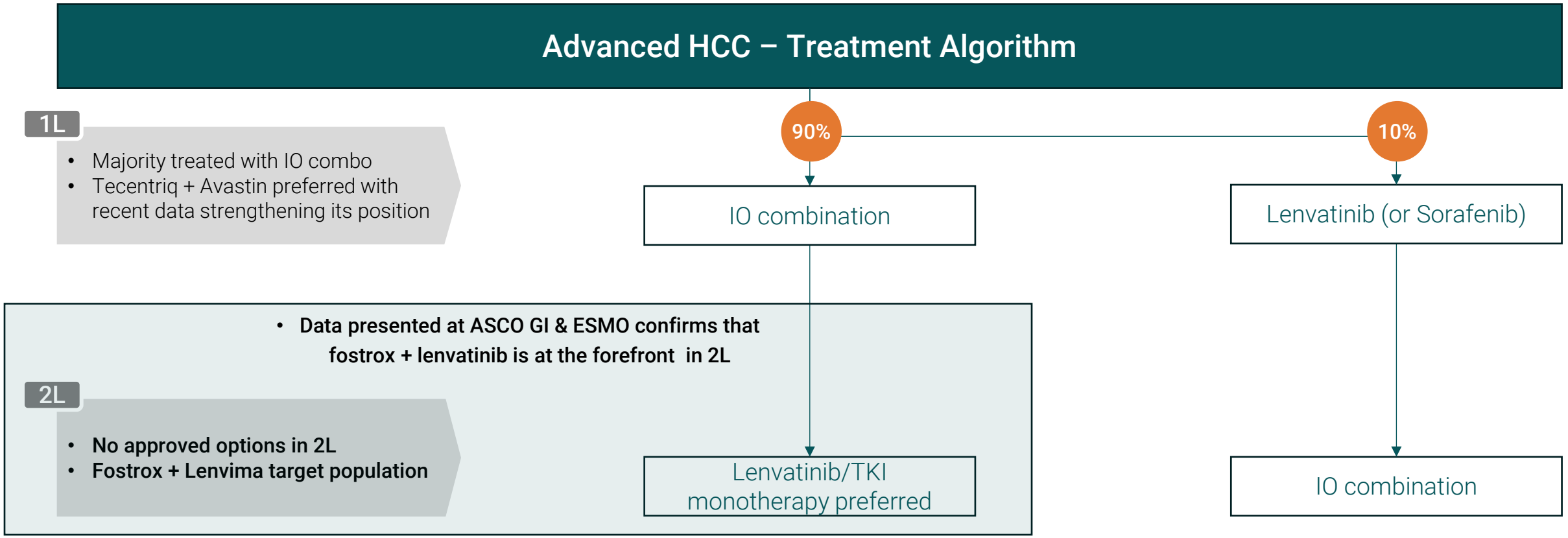
Fatty Liver Disease Is Expected to Skyrocket By 2050

A model predicts the rise in MASLD and MASH will drive an alarming increase in liver failure, liver cancer and liver transplants.

- Fatty Liver Disease (MASLD/MASH) expected to rise dramatically over the next 30 years
- The number of newly diagnosed liver cancer patients each year is expected to double
- HCC market growth further spurred by more and better treatments enabling patients to be treated longer

[1]JAMA Network Open. 2025;8(1):e2454707

Fostrox + Lenvima is at the forefront of development in population where no treatments are approved today



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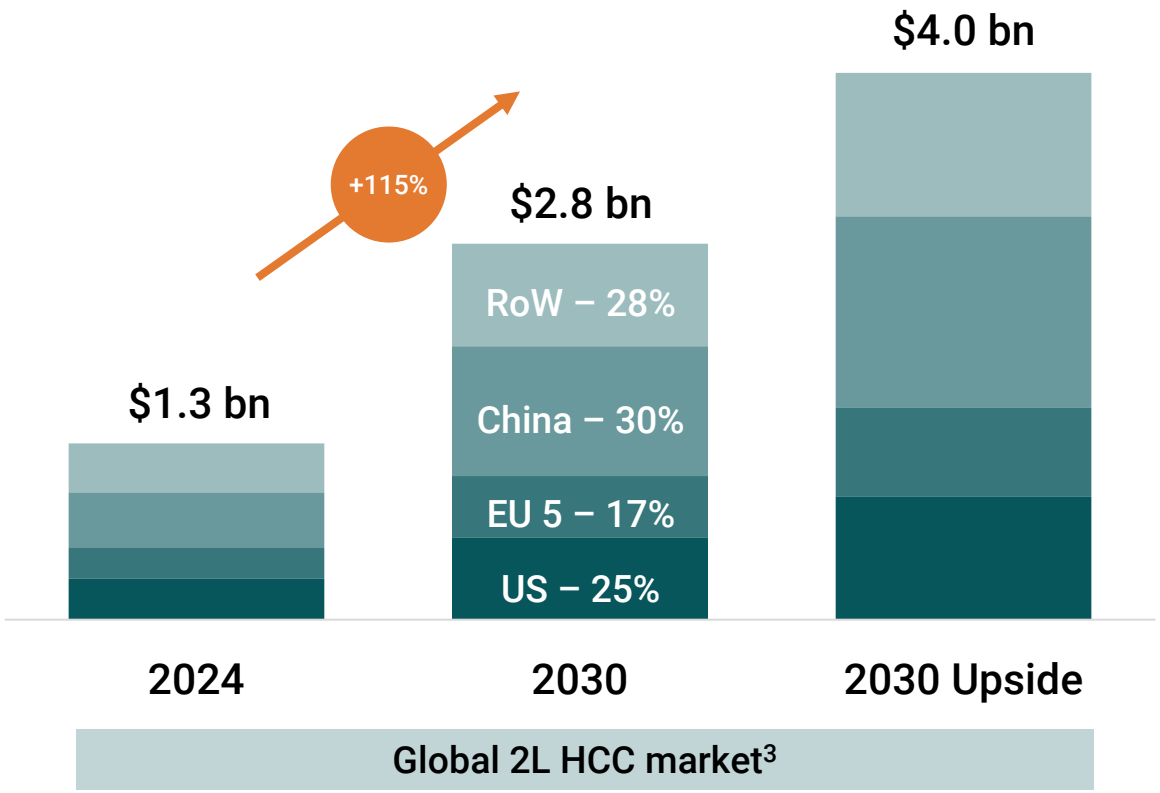
2nd line HCC – a ~$3bn commercial opportunity[3]



Growth driven by:

- HCC to increase **+122% in the US** and +**82% in China**[2] by 2030, caused by fatty liver disease
- With improved 1L treatment, more patients will be **fit enough for 2L, 50% → 70%**

2030 Upside:

- Average treatment duration increases to 10 months based on fostrox + Lenvima® study

[1]Rumguy et al. Journal of Hepatology 2022
[2]Huang et al., Nature Reviews, Gastroenterology & Hepatology, Vol 18, 2021
[3]GlobalData 2021 and internal analysis

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Absence of effective treatment options in 2nd line HCC

Treatment algorithm – major need for new 2nd line options

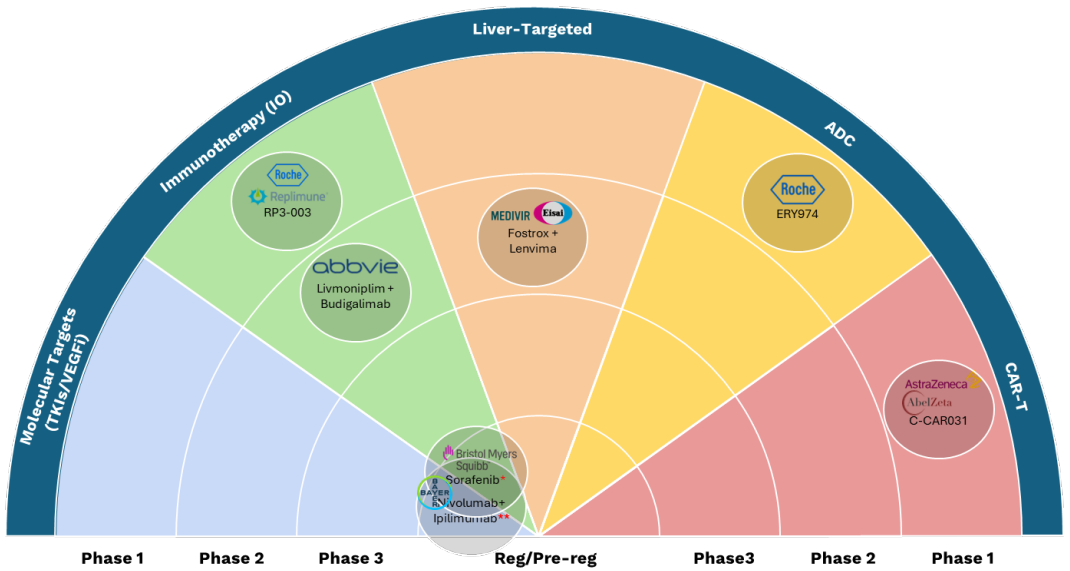
1st line treatment

- **IO combinations Standard of Care** – Tecentriq + Avastin
- Numerous studies ongoing evaluating various other IO combinations

2nd line treatment

- **No approvals or scientific evidence** to support treatment choice in 2nd line
- Few ongoing studies in 2nd line

Competitive landscape in 2nd line HCC highlights lack of novel mechanisms in development with fostrox + Lenvima at the forefront



"We are becoming greedy, trying to have 8 different regimens in the 1L setting and none of us know what to do after.

If I had my way, the focus should really be on 2L treatment and beyond"

Rachna T Schroff, University of Arizona Cancer Center
Late Breaking Abstract session at ESMO, September 2024

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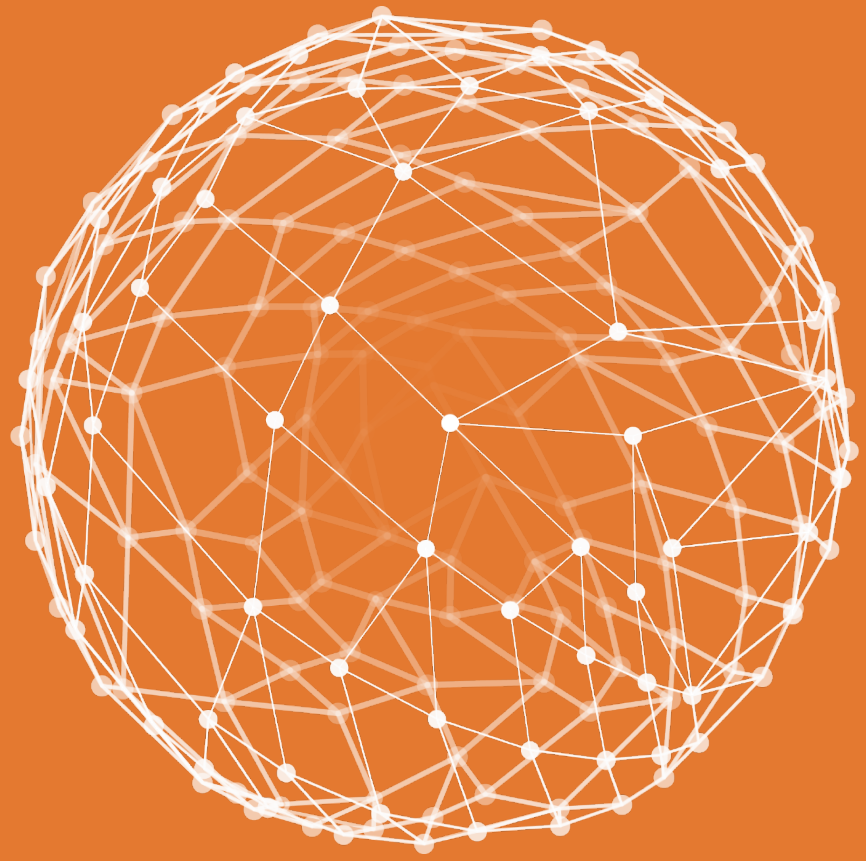
*Sorafenib was the first approved 1st-line treatment for HCC. Although approved for 2nd-line use, guidelines recommend against it due to a lack of evidence showing efficacy after immunotherapy combinations.
**Nivolumab + Ipilimumab were approved for patients post-sorafenib but are now moving into 1st line HCC treatment (positive phase III, awaiting approval (source)).

Fostrox – tailored for the specific needs of HCC



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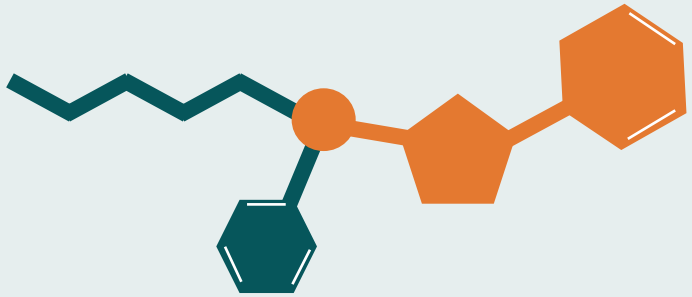
Fostrox – designed to selectively kill tumor cells in the liver

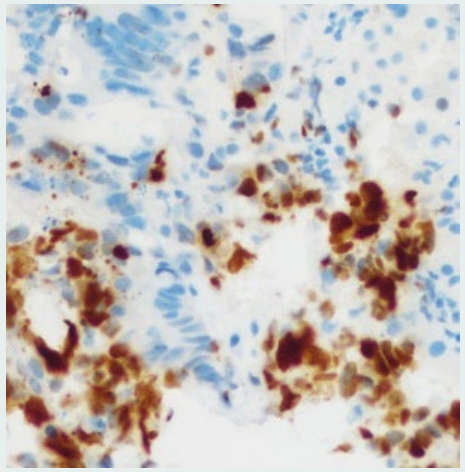
Prodrug transports inactive payload to the liver, where it is rapidly activated by liver enzymes[1]



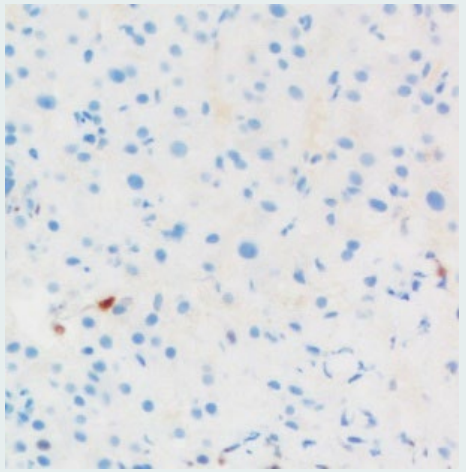
Liver-guided delivery – prodrug

Tumor-selective payload – troxacitabine

Kills tumor cells[2,3,4]



Spares healthy cells[2,3,4]



[1]Bethell, R. et al P-035, ILCA 2016
[2]Kukhanova, M et al J Biol Chem 1995
[3]Albertella, M. et al EASL Summit P01-05, 2018
[4]Öberg F. et al, EASL PO-221, 2022

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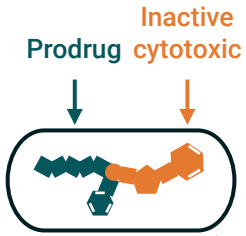
Fostrox MoA – tailored for HCC to achieve targeted DNA damage in liver tumor cells with minimal impact on healthy liver cells



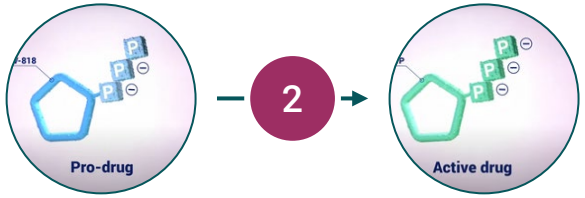
Fostrox taken as oral tablet

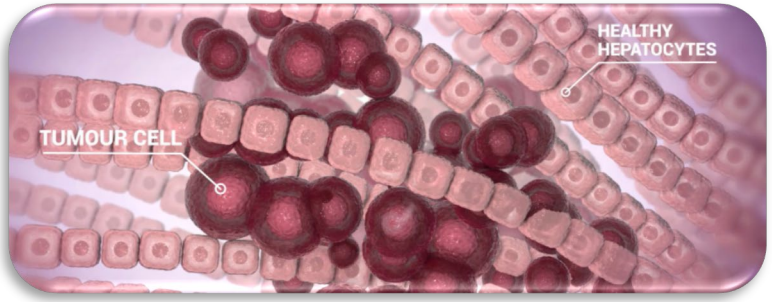
Prodrug transports inactive payload via first-pass metabolism to the liver[1]

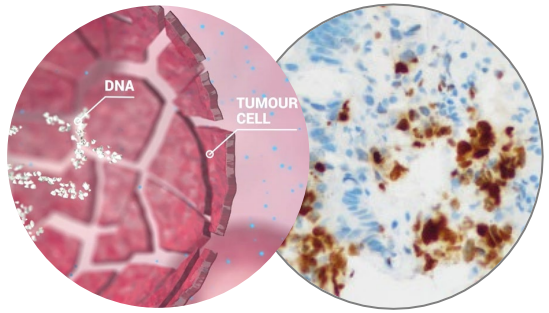
Mechanism of Action video

Rapidly activated by liver enzymes

Trapped inside liver cells for maximum liver exposure and minimal systemic spread[2,3,4]

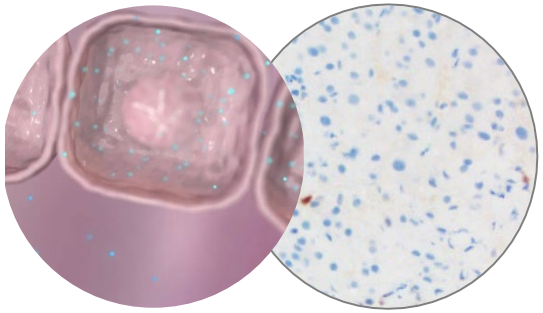






Cell death in tumor cells

Causes selective cell killing effect in tumor cells, sparing healthy liver cells as they very rarely divide[2,3,4]



No impact on healthy cells

[1]Bethell, R. et al P-035, ILCA 2016
[2]Kukhanova, M et al J Biol Chem 1995
[3]Albertella, M. et al EASL Summit P01-05, 2018
[4]Öberg F. et al, EASL PO-221, 2022

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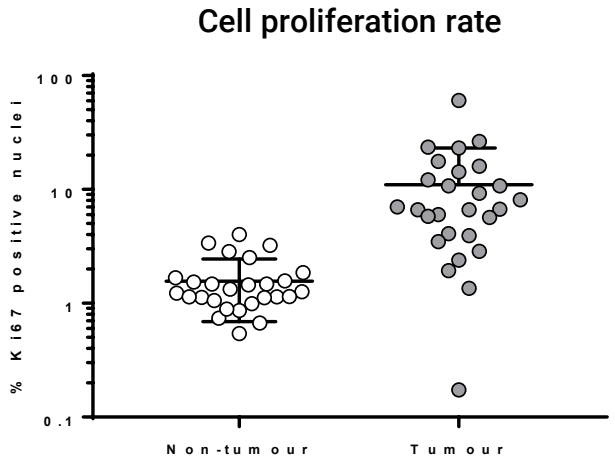
100-fold higher liver targeting vs IV administration & selective DNA damage in tumor cells enabling highly targeted mechanism

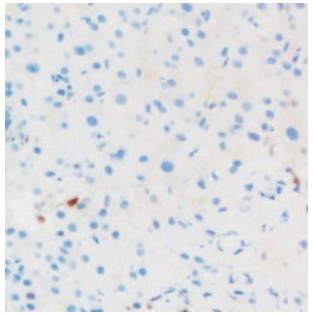
>100-fold higher liver targeting with fostrox than iv troxacitabine in rats

Compound	Route	Dose (µmol/kg)	AUC_{Liver} (nmol*h/g)	AUC_{Plasma} (µmol*h/L)	AUC ratio (Liver/ Plasma)
Troxa-citabine	*iv*	80	<1.2	80	**<0.016**
Fostrox	*oral*	80	10	5.4	**1.9**

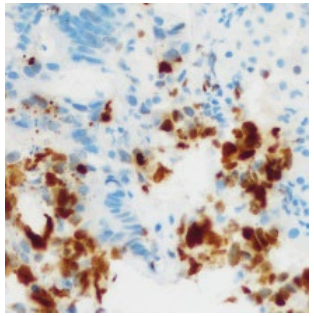
Liver tumor cells divide significantly more often than non-tumor cells[1]



Fostrox induces DNA damage in tumor cells, sparing normal liver tissue[2]



Normal liver tissue*



Tumor tissue*

Fostrox-induced DNA-damage indicated by pH2AX immuno-histochemistry (IHC) staining of liver biopsy from phase 1b monotherapy

[1]Albertella, M. et al EASL Summit P01-05, 2017
[2]Öberg F. et al, EASL PO-221, 2022
*Induced DNA damage indicated by pH2AX IHC staining (brown color) in patient biopsies

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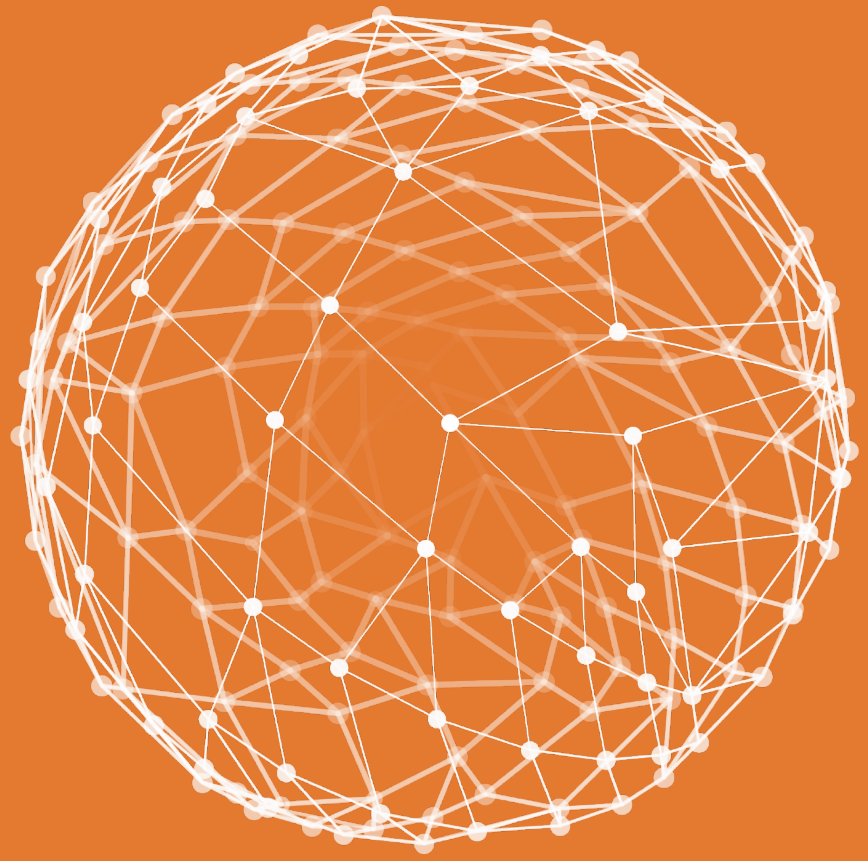
Fostrox + Lenvima shows promise of improved outcomes in 2L HCC



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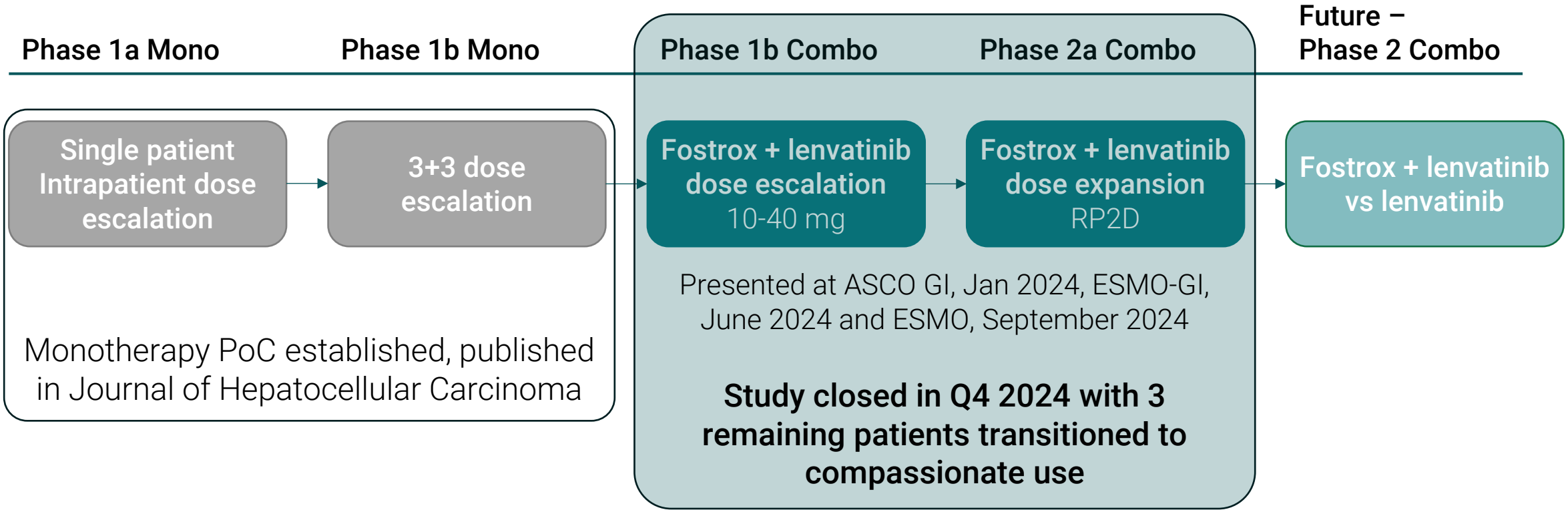
Fostrox Clinical Development Program; monotherapy PoC established, focus on combination approach in 2L HCC



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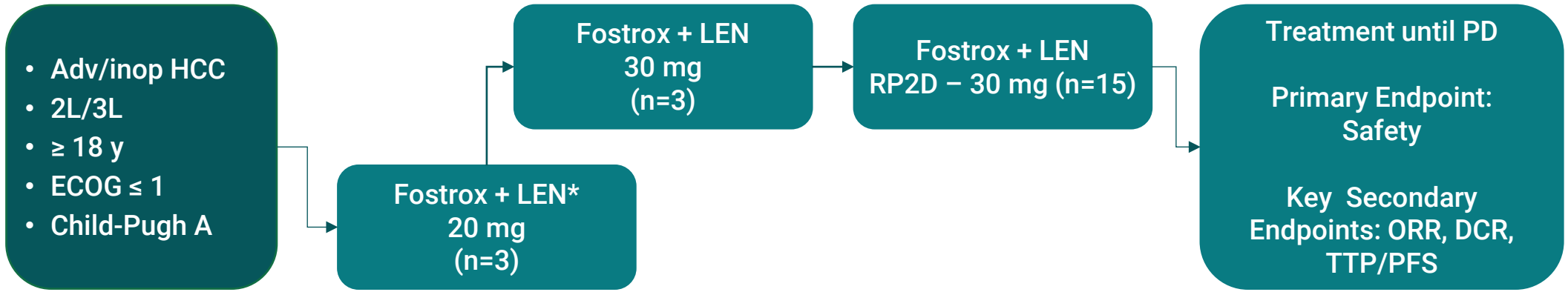
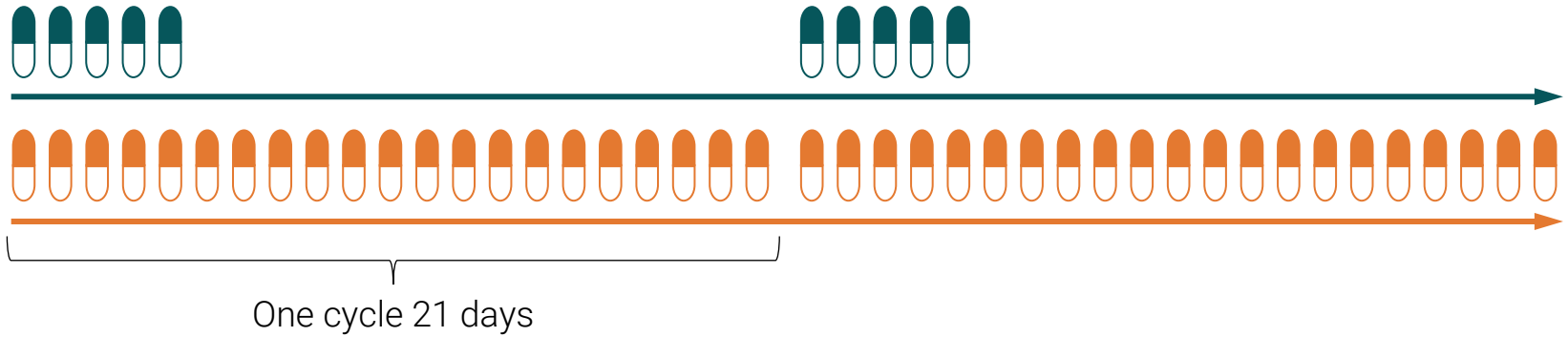
Fostrox + Lenvima phase 1b/2a study design



Patients were enrolled at 15 sites in the UK, Spain and South Korea. Imaging assessments (CT & MRI) every 6 weeks.

Fostrox: Oral QD
5 days in 21 days cycles

LEN: Oral QD continuous
(8 or 12 mg)



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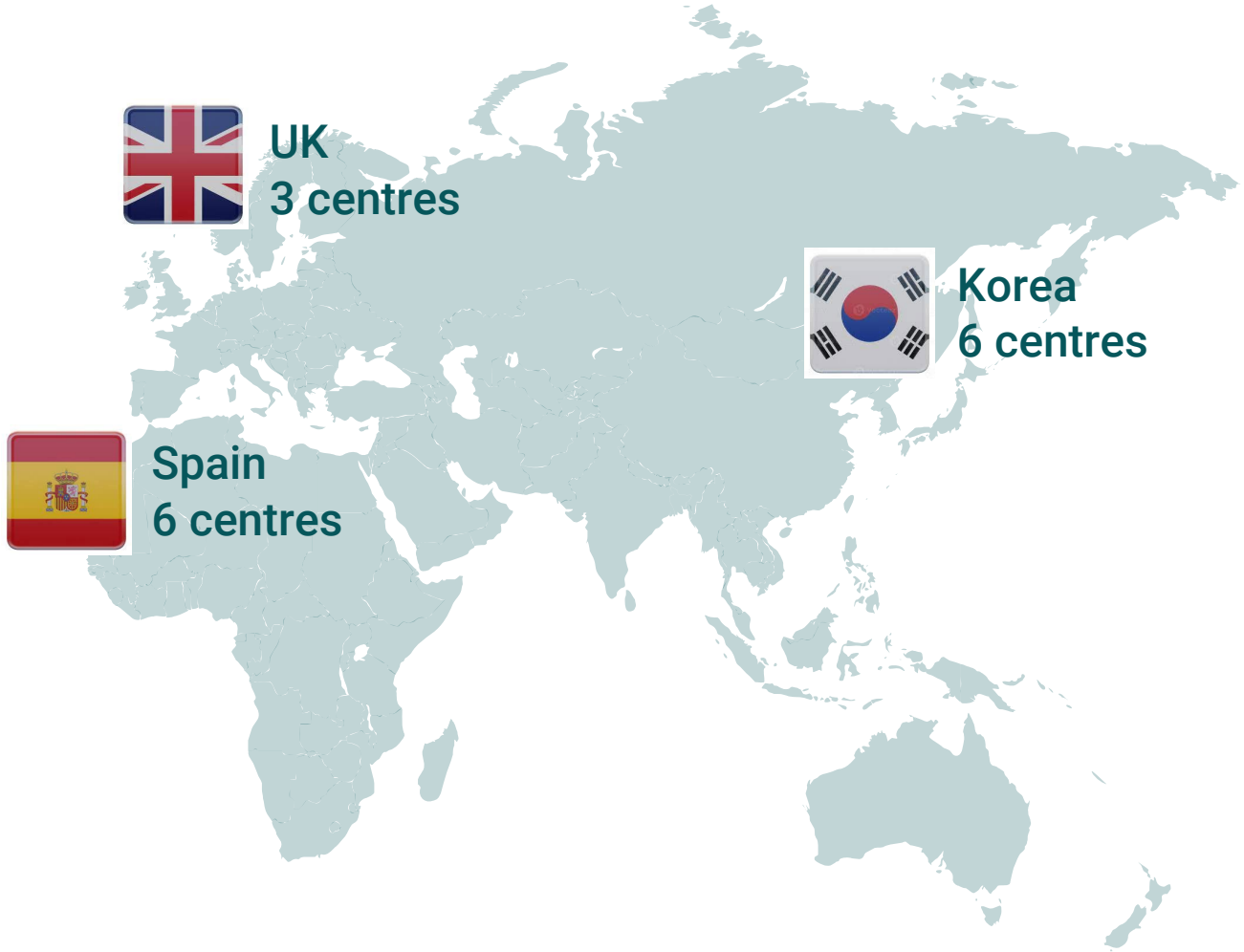
*LEN = lenvatinib

Global phase 1b/2a study with fostrox + Lenvima (TKI) positive, final data presented at EASL in February



Poster P02-13 presented by Dr. Jeff Evans, Glasgow, at EASL Liver Cancer Summit in February in Paris

EASL LIVER CANCER SUMMIT 2025
Paris, France 20-22 February

Final safety and efficacy results from the phase 1b/2a study of fostrox plus lenvatinib in second/third line advanced hepatocellular carcinoma progressed on immunotherapy

T.R. Jeffry Evans[1], Hong Jae Chon[2], Do Young Kim[3], Ho Yeong Lim[4], Teresa Macarulla[5], Carlos Gomez Martin[6], Victor Moreno[7], Min-Hee Ryu[8], Pia Baumann[9], Sujata Bhoi[9], Malene Jensen[9], Karin Tunblad[9], Hans Wallberg[9], Fredrik Öberg[9], Maria Reig[10], Jeong Heo[11]

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Introduction

Fostrox is a liver targeted oral prodrug of troxacitabine that selectively inhibits DNA replication in liver tumors[1].

Fostrox monotherapy was investigated in a phase 1a/1b dose escalating and expansion study in advanced solid tumors with an encouraging safety, tolerability profile and preliminary anti-tumor activity in hepatocellular carcinoma (HCC)[2].

Based on these results, a phase 1b/2a study of fostrox in combination with lenvatinib (LEN) in second line in locally advanced unresectable or metastatic hepatocellular carcinoma (HCC) (NCT03781934) was initiated and end of treatment data are reported here.

Aim

The aim of the phase 1b/2a study was to establish a recommended phase 2 dose of fostrox in combination with LEN and to evaluate safety & tolerability together with preliminary anti-tumor activity data.

Method

Figure 1: Study design of phase 1b/2a fostrox + LEN in advanced HCC

Table 1: Patient characteristics

	N = 21
Median age (range)	63 yrs (42 - 82)
Gender, Female / Male (%)	24 / 76
ECOG Performance status 0/1 (%)	71 / 29
Child-Pugh A (%)	100
Viral/Non-viral (%)	76* / 24
Extra hepatic lesion(s) Y/N (%)	67 / 33
AFP ≥400 ng/mL at baseline Y/N (%)**	48 / 52
Region, Asia / Europe (%)	67 / 33
Prior treatment lines; 2nd line/3rd line (%)	81 /19
Prior atezo/bevacizumab in 1L (%)	86
Prior local therapy (TACE, RFA etc)	76
PD on prior treatment (%)	100
Primary refractory on prior therapy (%)***	24
Starting dose fostrox, 20mg / 30mg (%)	14 / 86

Results

Safety and tolerability*

Table 2: Treatment emergent/related adverse events seen in >20% of patients

Adverse Events*	TEAE: No of pts (%)			TRAE: Grade ≥ 3* No of pts (%)	
	Grade 1/2	Grade 3	Grade 4	Fostrox	LEN
Hematologic AE					
Thrombocytopenia	13 (62)	5 (24)	2 (10)	5 (24)	6 (29)
Neutropenia (no febrile)	9 (43)	8 (38)	2 (10)	8 (38)	6 (29)
Anaemia	6 (29)	3 (14)		3 (14)	3 (14)
Leukocyte decrease	6 (29)	2 (10)		2 (10)	2 (10)
Other AE					
Hypothyroidism	11 (52)				
Diarrhea	11 (52)	1 (5)			1 (5)
Hand-foot syndrome	10 (48)	1 (5)			1 (5)
Fatigue	9 (43)				
Asthenia	2 (10)	2 (10)		1 (5)	2 (10)
Anorexia	8 (38)				
Proteinuria	7 (33)	2 (10)			1 (5)
Hypertension	8 (38)	3 (14)			2 (10)
Cough	5 (24)				
Pruritus	5 (24)				

* There was no grade 5 fostrox related AE and one grade 5 LEN related death (renal failure)

- With a median follow-up of 10.5 months (range 1.2 – 27.8 months) the combination of fostrox + LEN was shown to be safe and well tolerated with no unexpected new adverse events reported.
- Most common adverse events were laboratory test results with 33 events (out of 880 blood tests) of grade ≥ 3 neutropenia and thrombocytopenia with clinical relevance (fostrox: dose-delay, reduction or discontinuation) in 11 of these 33 occasions.
- No febrile neutropenia or low platelet count with bleeding was reported
- SAEs were reported in 8 patients with 17 events in total
  - No events were related to fostrox
  - 8 events in 6 patients were possibly related/related to LEN (asthenia, ischemic stroke, renal failure, hepatic encephalopathy, diarrhea)
- Fostrox dose reduction due to AE was done in 29% and discontinuation in 5%.
- LEN dose reduction due to AE was done in 52% and discontinuation in 10%

*Data cut Nov 30 2024

Clinical efficacy* (investigator review, RECISTv1.1)

Figure 2. Time to progression (TTP)

Median TTP, mo 10.9 (95% CI, 4.2-18.1)
DCR, % 24
Median DoR, mo 7.0 (95% CI, 1.6-NR)
DCR, % 81

Figure 3. Overall survival (OS)

Median OS, mo 13.7 (95% CI, 7.6 – NR)

Figure 4. Percentage change in total target lesion size over time

Figure 5. Best % change in target lesion size related to fostrox dose reduction

Target lesion tumor reduction was seen in >75% of patients and median duration of clinical benefit (CR, PR or SD>24 weeks*) was 11.2 months.

Fostrox dose reduction, required in 29% of patients, didn't impact response to treatment

Figure 6. Percentage change in AFP over time and best overall response

Figure 7. Best % change in target lesion size related to AFP at baseline

Reduced or stable AFP levels from baseline to 6 weeks were seen in 79% of patients and was associated with imaging response (RECISTv1.1).

Target lesion size reduction was seen independent of AFP level at baseline

Conclusions

- Fostrox + LEN was safe and well tolerated using the RP2D of fostrox 30 mg QD orally for 5 days in a 21-day cycle in combination with standard dose of LEN
- Encouraging efficacy was seen with a best objective response, median time to progression and overall survival of 24%, 10.9 months and 13.7 months, respectively
- The majority of patients had a reduction in AFP levels at 6 weeks which correlated with imaging response, and overall target lesion reduction was independent of baseline AFP levels
- Based on the promising result from this study, a randomized phase 2b study with fostrox + LEN versus placebo + LEN in second line advanced HCC is planned to enroll patients in Europe, Asia and in North America

Acknowledgement

Thanks to all the investigators, research teams, and clinical staff in Korea, Spain and the UK, whose dedication and expertise made this study possible. We also sincerely thank the patients and their families for participating and contribution to advancing medical knowledge in liver cancer.

Reference

1. Bethell, R. et al 2017, EASL International Liver Congress, Poster SAT-123 (www.medivir.com/media/7196/liver-targeting-with-the-novel-nucleoside-prodrug-troxacitabine.pdf)
2. J Hepatocell Carcinoma. 2024 Oct 24;11:2033-2047
3. Am J Cancer Res 2021;11(4):1121-1131

Abbreviations

Hepatocellular carcinoma (HCC), recommended phase 2 dose (RP2D), progressive disease (PD), best objective response (BOR), duration of response (DoR), disease control rate (DCR), time to progression (TTP), duration of clinical benefit (DoCB), Eastern Cooperative Oncology Group Performance Status (ECOG PS), Response evaluation criteria in solid tumors (RECIST 1.1), Computerized tomography (CT), Magnetic Resonance Imaging (MRI), Trans arterial chemoembolization (TACE), radio frequency ablation (RFA), Common Terminology Criteria for Adverse Events (CTCAE), Immunotherapy (IO), target lesion (TL), treatment emergent adverse event (TEAE), treatment related adverse event (TRAE), lenvatinib (LEN), Kaplan Meier (K-M), serious adverse event (SAE), opaque decline daily (QD), alfa feto protein (AFP), overall survival (OS)

Contact information

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Patient characteristics reflecting generous inclusion criteria

Patient characteristics[1]	N = 21
Mean age (range)	62 yrs (42 - 82)
Gender, Female / Male (%)	24 / 76
ECOG Performance status 0/1 (%)	71 / 29
Child-Pugh A (%)	100
Viral/Non-viral (%)	76* / 24
Extra hepatic lesion(s) Y/N (%)	67 / 33
AFP ≥400 ng/mL at baseline Y/N (%)**	45 / 55
Region, Asia / Europ (%)	67 / 33
Prior treatment lines; 2nd line/3rd line (%)	81 /19
Prior atezolizumab/bevacizumab in 1L (%)	86
Prior local therapy (TACE, RFA etc)	70
PD on prior treatment (%)	100
Primary refractory on prior therapy (%)***	24
Starting dose fostrox, 20mg / 30mg (%)	14 / 86

*HepB-81% and HepC-19%; **AFP- NA for 1 pt; ***Active treatment ≤ 12 weeks. Data NA for 3 patients

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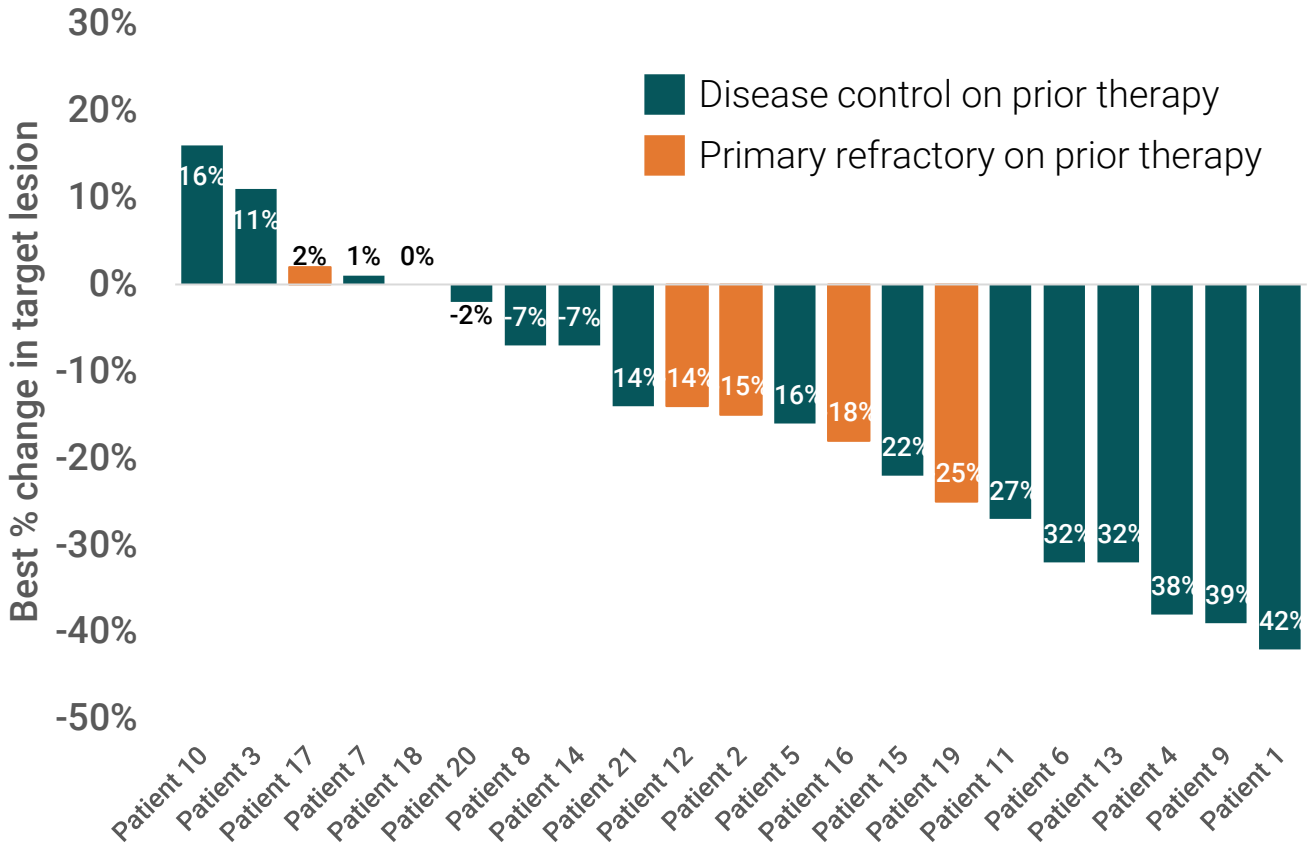
[1]Chon et al., ESMO 2024, Poster 986.

More than 75% of patients experiencing tumor shrinkage[1]

Best percentage change in target lesion size related to treatment response in first line



- Median duration of response 7.0 months
- Longest duration of response still ongoing at 19.5 months
- Patients benefitted from treatment independent of outcome in previous line of therapy

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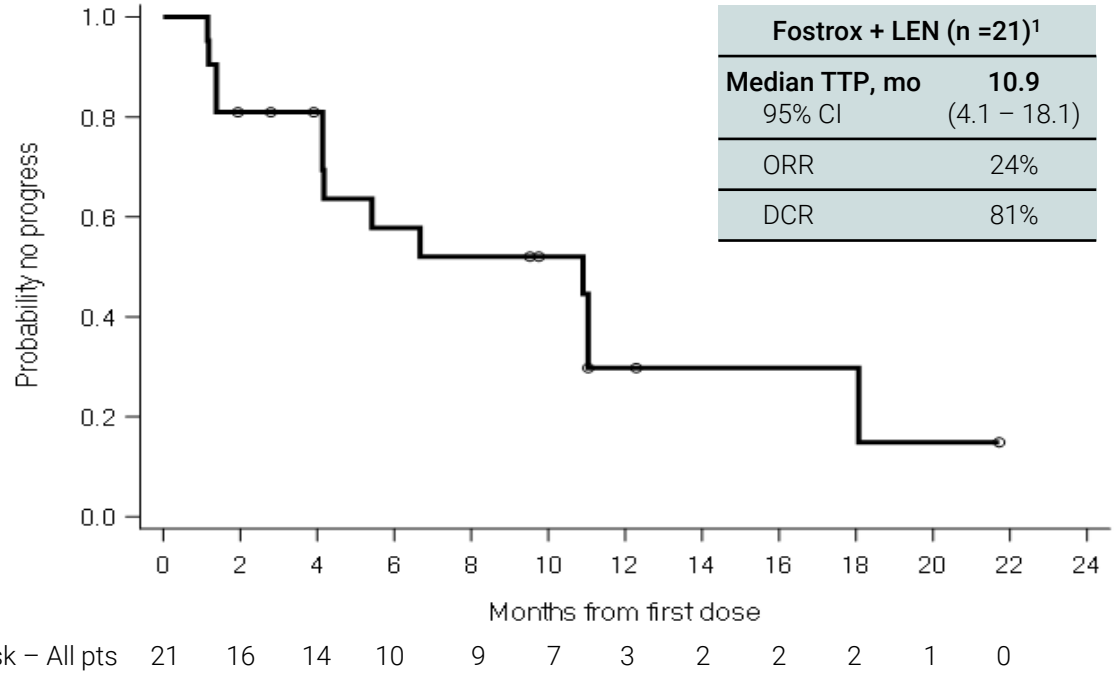
[1]Chon et al., ESMO 2024, Poster 986.

Median TTP 10.9 months, indicating substantially improved efficacy compared with Lenvima alone[1]

Median time to progression (TTP) with fostrox + LEN – investigator review, RECISTv1.1



Fostrox + LEN (n =21)[1]	
Median TTP, mo	**10.9**
95% CI	(4.1 – 18.1)
ORR	24%
DCR	81%

- Median time to progression 10.9 months
- Median follow-up of 10.5 months
- Longst running patient still on treatment after three years (Aug 2025)

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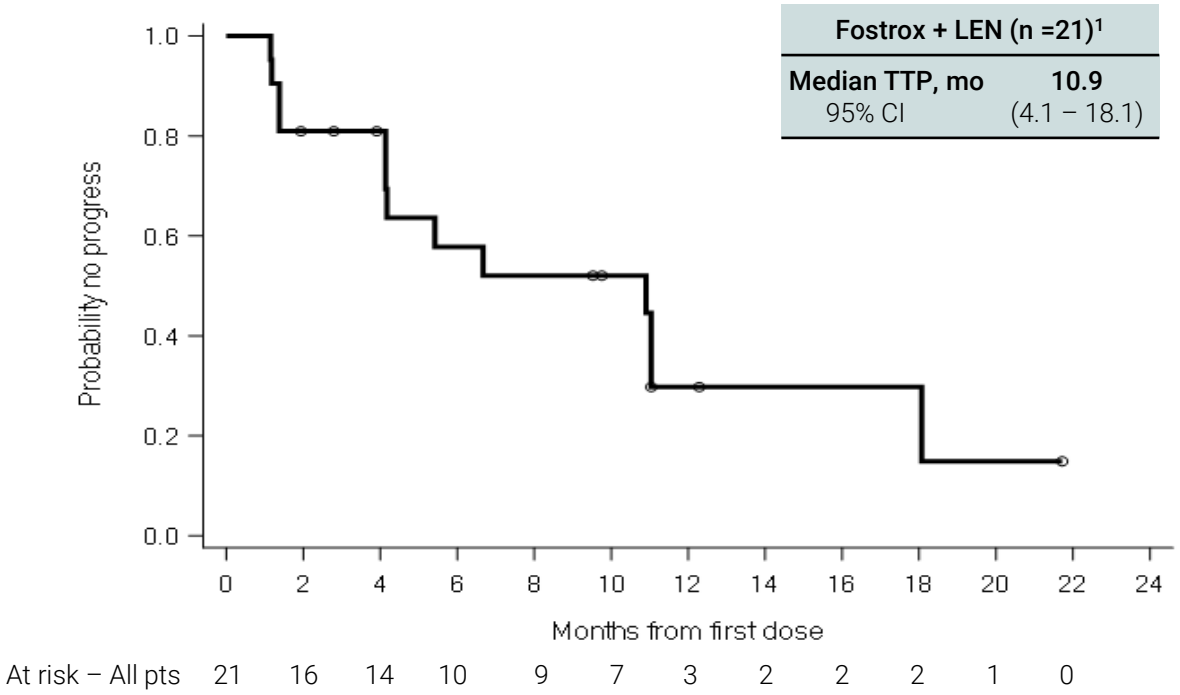
[1]Chon et al., ESMO 2024, Poster 986.

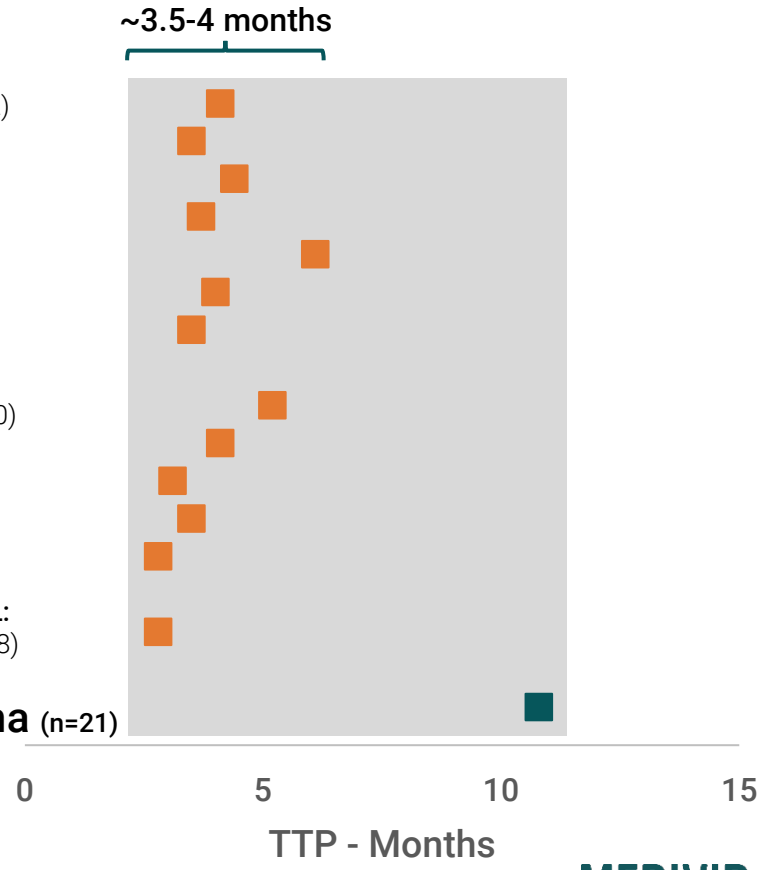
Median time to progression (TTP) 10.9 months, remarkably longer than Lenvima monotherapy and other 2L HCC treatments

Median TTP (Kaplan-Meier) with fostrox + Lenvima



Fostrox + LEN (n =21)[1]	
Median TTP, mo	**10.9**
95% CI	(4.1 – 18.1)

Median TTP/PFS vs previous studies in 2L HCC



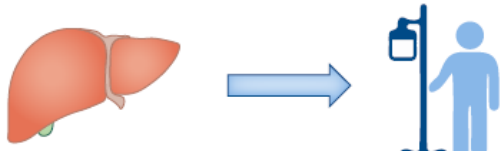
[1]Chon et al., ESMO 2024, Poster 986.

Korean Cancer Study Group prospective study data with Lenvima post Tecentriq + Avastin, aligns with other 2nd line outcome data

Second-line lenvatinib after atezolizumab-bevacizumab in advanced HCC

Study design

HCC progressed on 1st-line atezolizumab-bevacizumab

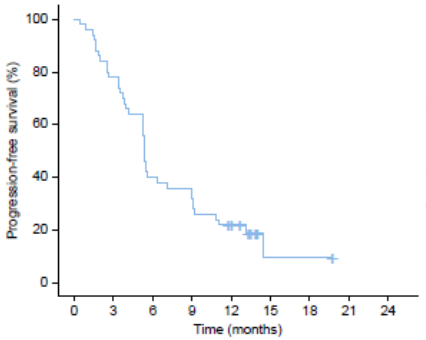


2nd-line lenvatinib

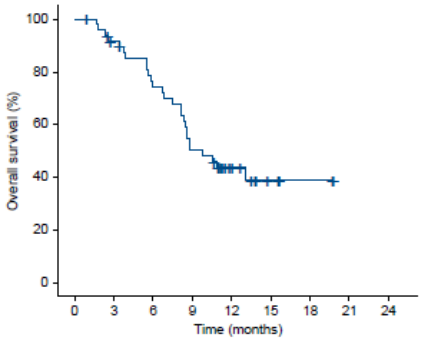
- **Investigator-initiated, multicenter, single-arm phase 2 study**
- **50 patients enrolled from 13 sites in Korea**
- **Primary end point:** PFS (>median 4.5 months)
- **Secondary endpoints:** OS, ORR, DCR, DoR, and safety.

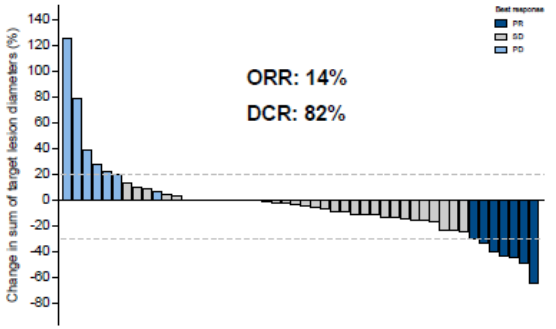
Results

Median PFS: 5.4 months



Median OS: 9.8 months





ORR: 14%

DCR: 82%

- **The most common AE were diarrhea (42.0%), hypothyroidism (32.0%), and anorexia (30.0%).**
- **Grade ≥3 AEs occurred in 46.0% of patients.**

Conclusion

- **Lenvatinib demonstrated promising efficacy and a manageable safety profile as a second-line treatment for patients with HCC progressing on atezolizumab-bevacizumab.**
- **These findings offer prospective evidence supporting lenvatinib as a viable treatment option in the post-atezolizumab-bevacizumab context.**

[1]Kim et al., Journal of Hepatology, Sept 04 2025

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Similar patient characteristics across the Lenvima monotherapy study and the Phase 1b/2a fostrox + Lenvima study

Patient characteristics	N = 50 Lenvima monotherapy 13 sites in Korea[1]	N = 21 Fostrox + Lenvima 15 sites in Korea, UK & Spain[2]
Mean age (range)	66 (32-86)	62 yrs (42 - 82)
Gender, Female / Male (%)	18 / 82	24 / 76
Child-Pugh A (%)	100	100
BCLC stage A/B or C (%)	12 / 88	0 / 100
Viral/Non-viral (%)	72 / 28	76* / 24
AFP ≥400 ng/mL at baseline Y/N (%)**	44 / 56	48 / 52
Region, Asia / Europe (%)	100 / 0	67 / 33
Prior treatment lines; 2nd line/3rd line (%)	100 / 0	81 /19
Prior atezolizumab/bevacizumab in 1st line (%)	100	86
Prior TACE therapy (%)	58	70

*HepB-81% and HepC-19%; **AFP- NA for 1 pt

[1]Kim et al., Journal of Hepatology, Sept 04 2025
[2]Chon et al., ESMO 2024, Poster 986

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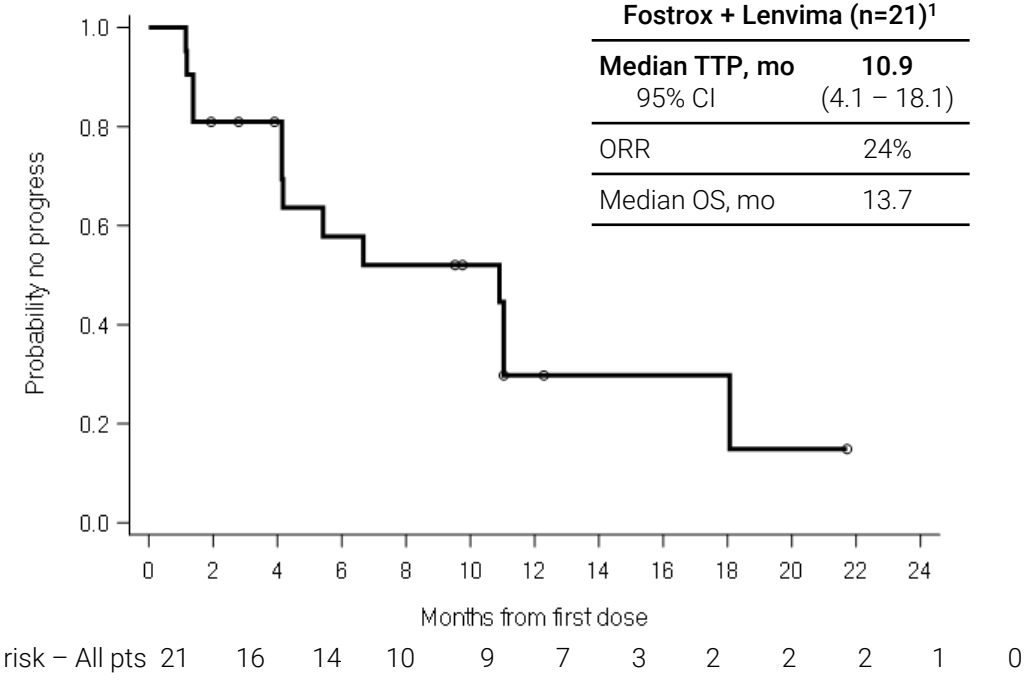
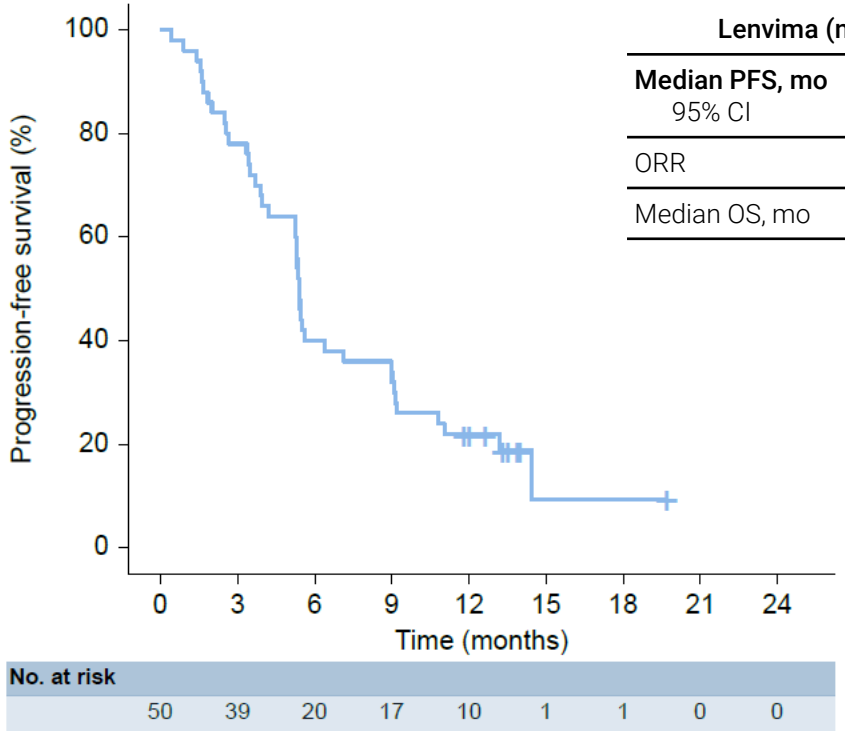
Fostrox + Lenvima phase 1b/2a data showed substantially better outcome data compared to the Lenvima montherapy study

Median TTP – Fostrox + Lenvima[1]



Fostrox + Lenvima (n=21)[1]	
Median TTP, mo 95% CI	**10.9** (4.1 – 18.1)
ORR	24%
Median OS, mo	13.7

Median PFS – Lenvima monotherapy[2]



Lenvima (n=50)[2]	
Median PFS, mo 95% CI	**5.4** (4.2 – 7.1)
ORR	14%
Median OS, mo	9.8

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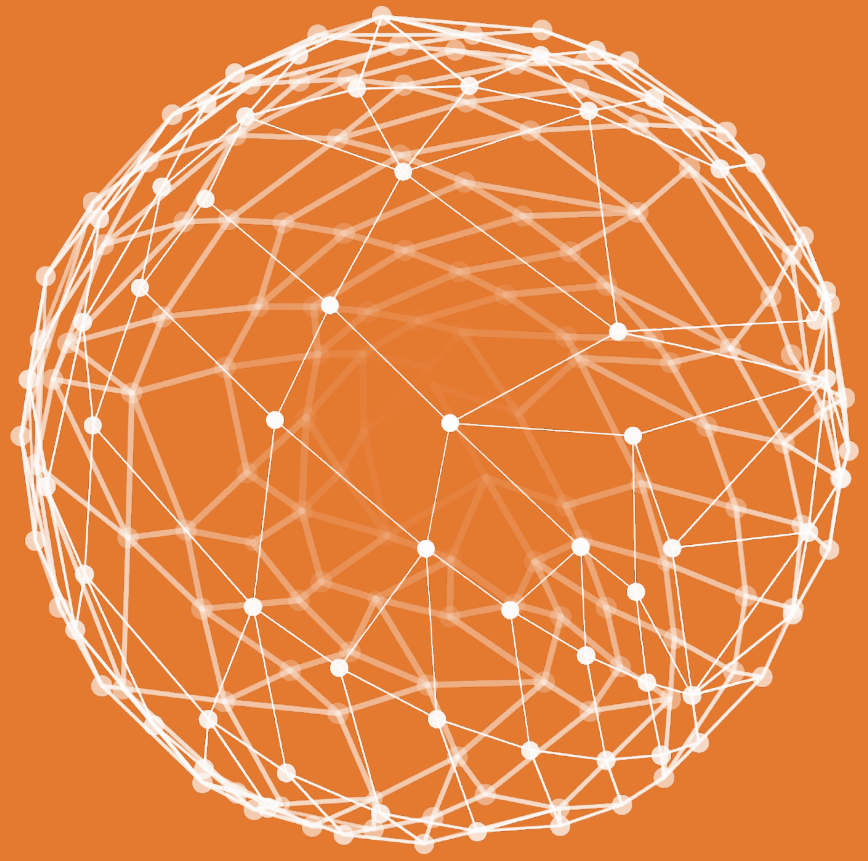
[1]Chon et al., ESMO 2024, Poster 986
[2]Kim et al., Journal of Hepatology, Sept 04 2025

Fostrox + Lenvima shows encouraging tolerability enabling patients to remain on treatment long-term



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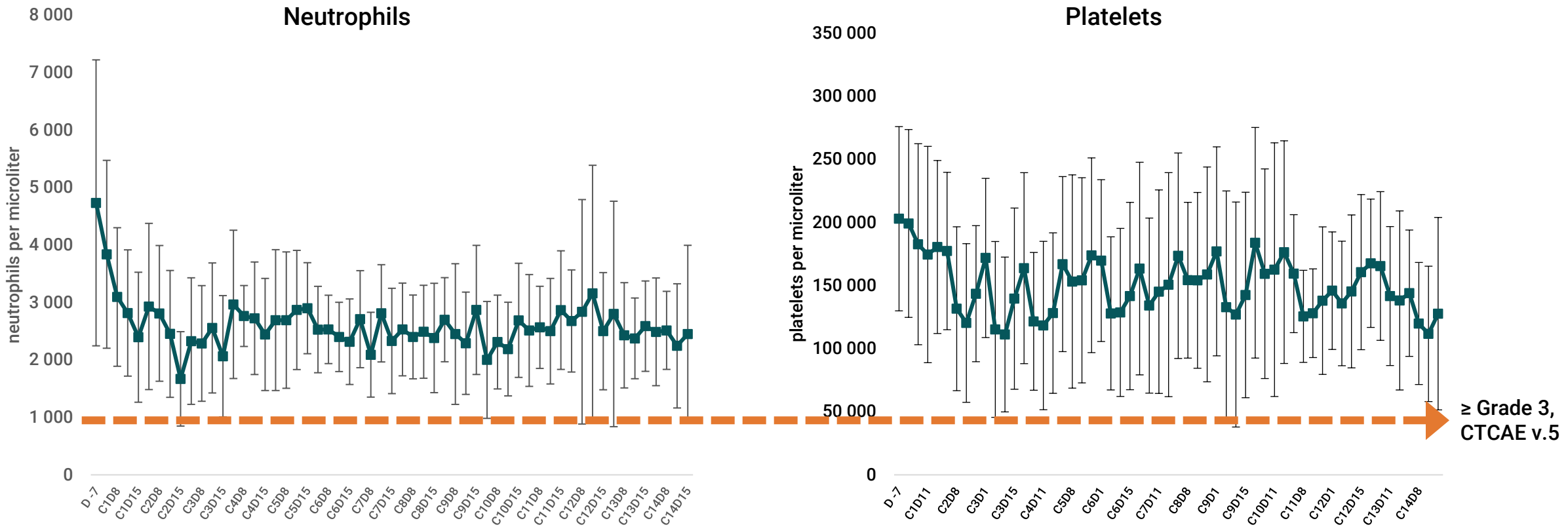
Absolute neutrophile and platelet counts were stable over the course of treatment, enabling long-term use[1]

Longitudinal neutrophil & platelet counts, at all time points measured over first 10 months of treatment



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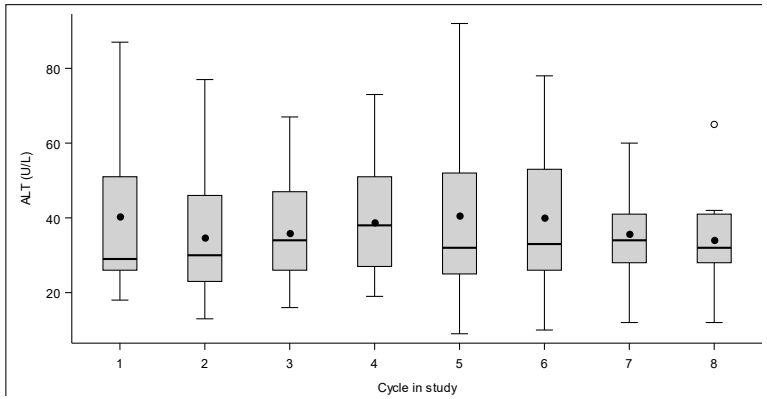
[1]Chon et al., ESMO 2024, Poster 986.

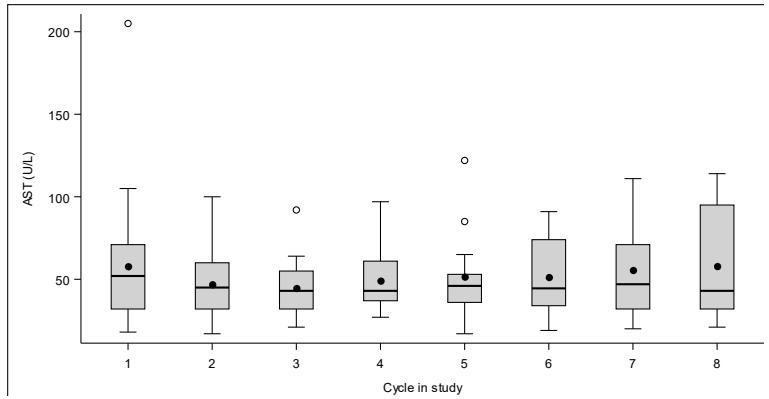
Stable liver function during treatment with fostrox + Lenvima – no deterioration in liver enzymes or change in ALBI score

ALT change over duration of treatment



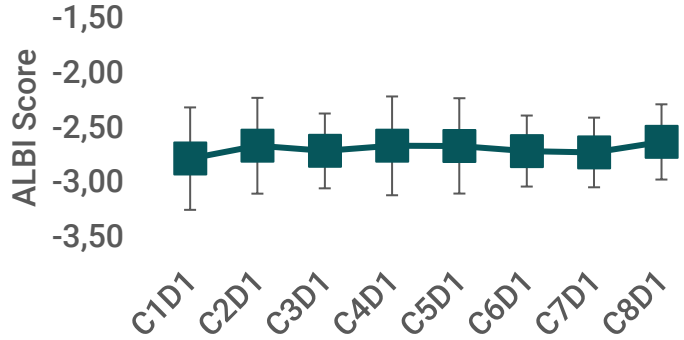
Patients At risk	21	21	16	15	14	14	11	10

AST change over duration of treatment



Patients At risk	21	21	16	15	14	14	11	10

ALBI score change over duration of treatment



Patients At risk	21	21	16	15	14	14	11	10

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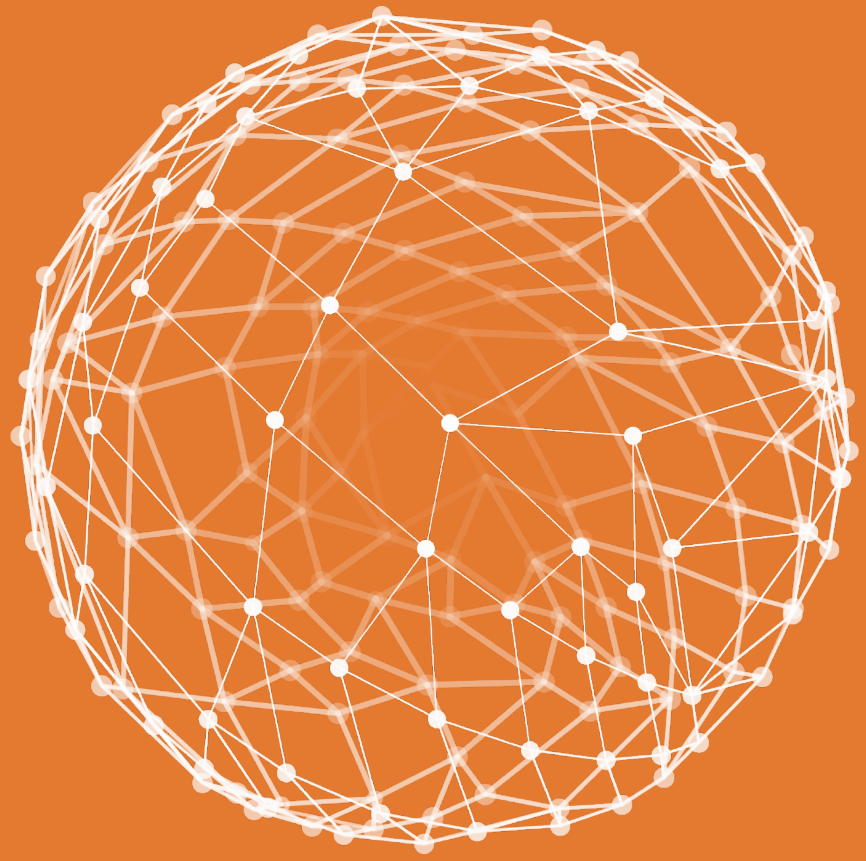
[1]Chon et al., ESMO GI 2024, Poster 176.

FLEX-HXX: Phase 2 study enables rapid generation of randomized, comparative data to confirm benefit of fostrox combination with Lenvima in 2nd line HCC



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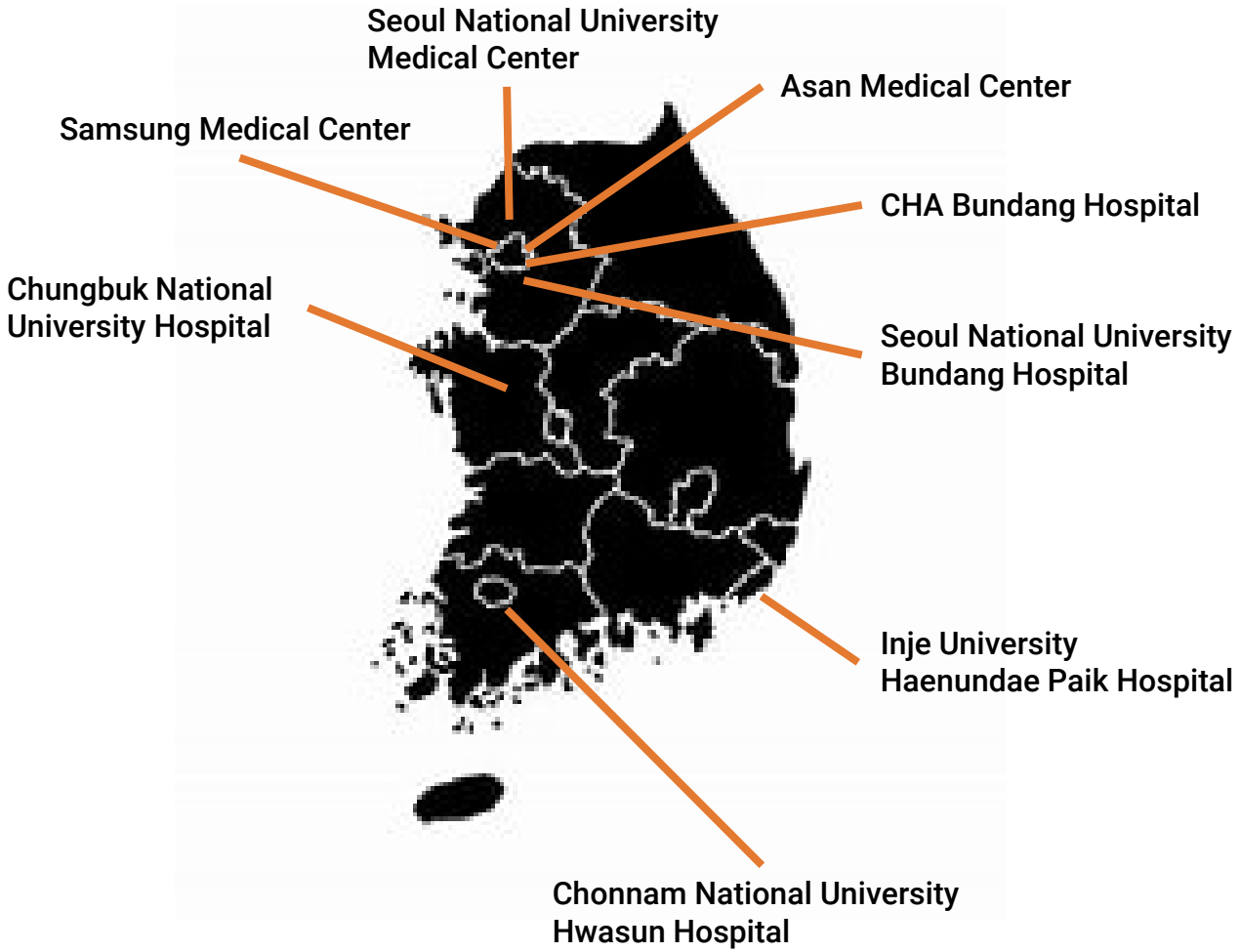
FLEX-HCC
Fostrox + Lenvatinib Combination for Advanced HCC



Primary Investigator

Dr. Hong Jae Chon

CHA Bundang Hospital,
Seoul, Korea

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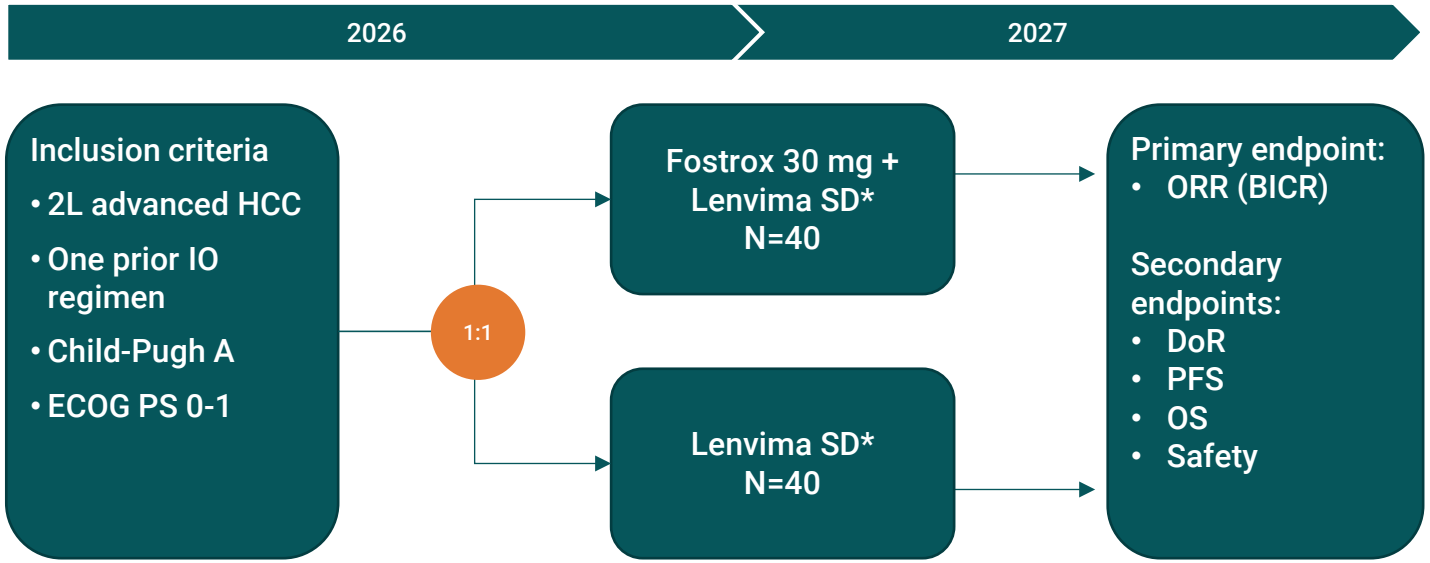
FLEX-HCC: Randomized, comparative phase 2 study to confirm benefit for fostrox + Lenvima combination in 2nd line HCC



*standard weight based dose in HCC

Response assessments every 6 week with CT or MRI

Study design:

- 80 pts randomized to fostrox + Lenvima or Lenvima alone
- 8 sites in Korean Cancer Study Group
- Efficacy evaluated by Blinded Independent Central Review (BICR)

Estimated study timelines:

- Enrollment time: 12 mo
- Topline results H2 2027

Key benefits:

- Generation of robust comparative efficacy and safety data in collaboration with an established research consortium
- Enables rapid data read out

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Fostrox (fostroxacitabine bralpamide)
The first oral, liver-targeted treatment tailored for HCC

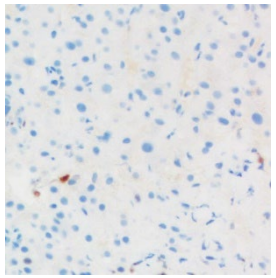
Oral, liver-activated small molecule inducing DNA damage in tumor cells, sparing healthy liver cells[3]

Unique, liver-targeted approach in HCC

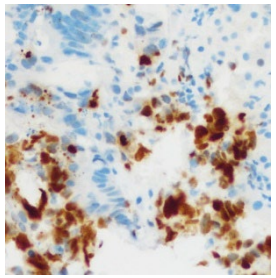
Liver-guided delivery – prodrug

Tumor-selective payload – troxacitabine

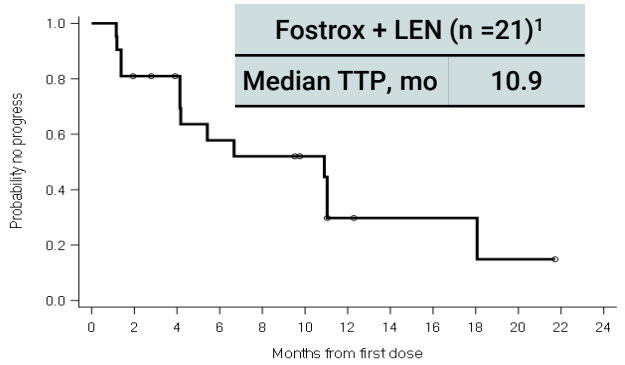
No DNA damage in healthy liver tissue

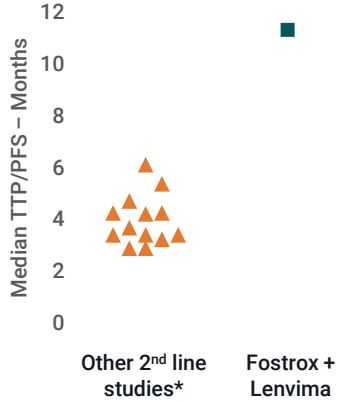


DNA damage in tumor tissue



10.9 months time to progression, substantially better than SoC[1,2]





*see slide 20 for details regarding individual study data

Absence of effective treatment options in 2nd line enables first-to-market opportunity for fostrox + Lenvima

- No 2nd line treatments approved in advanced HCC
- FLEX-HCC Phase 2 study initiated, in collaboration with Dr Hong Jae Chon and the Korean Cancer Study Group, to confirm superior benefit of fostrox + Lenvima vs Lenvima alone in 2nd line HCC

Market opportunity in 2nd line HCC >$2.5bn, with significant upside potential

>$2.5bn

2nd line HCC market by 2030, fastest growing cause of cancer death in US[4]

Significant upside in liver metastasis from other solid tumors

[1]Chon et al., ESMO, 2024, Poster 986
[2]Based on data from previous 2L phase 3 HCC studies with Stivarga, Cyramza & Cabometyx and investigator initiated prospective & retrospective 2L studies with Lenvatinib
[3]Evans et al ASCO GI, 2021
[4]Ma et al., Cancer, June 15, 2019; 2089-2098

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Key patent approval in Japan for fostrox + Lenvima extending protection until 2041, complementing previous approval in EU

Medivir receives Notice of Allowance for fostrox plus lenvatinib combination patent by Japan Patent Office

2025-07-08

Medivir AB (Nasdaq Stockholm: MVIR), a pharmaceutical company focused on developing innovative treatments for cancer in areas of high unmet medical need, announces today thar it has received a Notice of Allowance by the Japan Patent Office (JPO) for the company's patent application covering claims for the combination of fostroxacitabine bralpamide (fostrox) with lenvatinib (Lenvima) for the treatment of hepatocellular carcinoma (HCC) and cancer metastases to the liver.

- Covers the combination of fostrox + Lenvima for the treatment of HCC and metastases to the liver
- Now approved in Japan, EU and Australia which indicates likelihood of other key regions to follow
- Generates critical extension of patent protection until 2041

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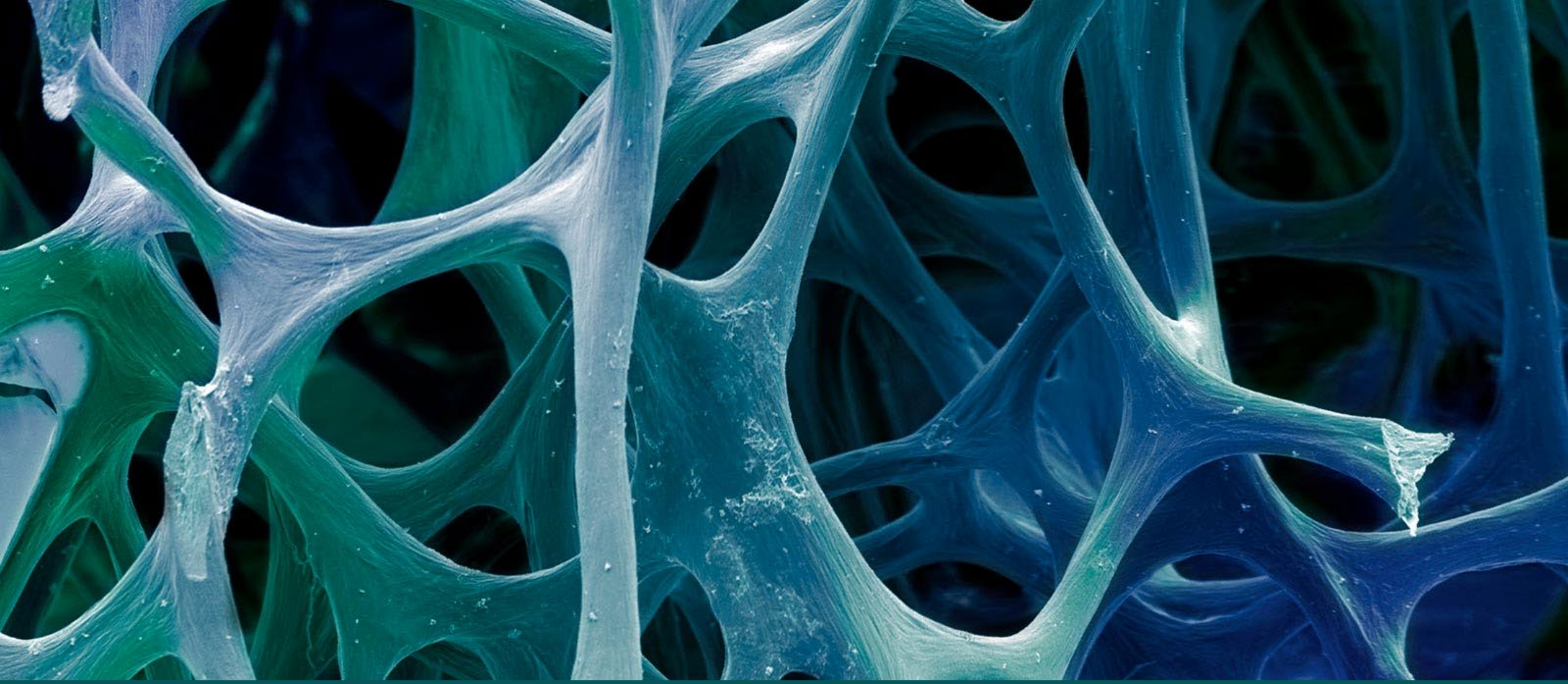

MIV-711 – Highly selective oral, cathepsin-K inhibitor for Potential treatment of Osteogenesis Imperfecta

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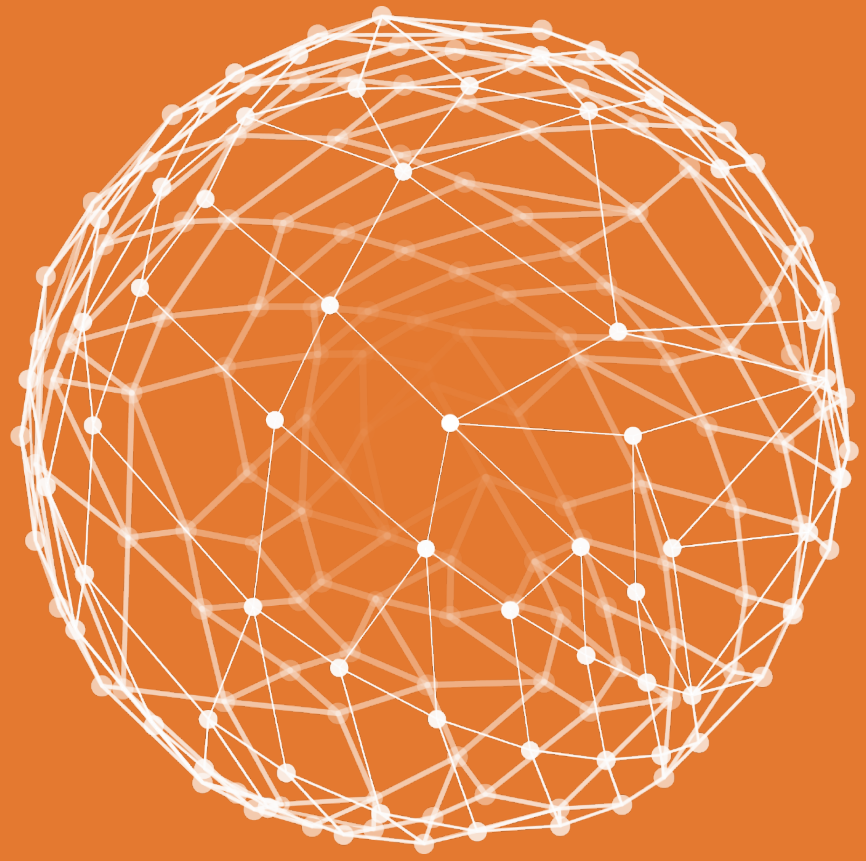
MIV-711 – Clear, scientific rationale for treatment of Osteogenesis Imperfecta



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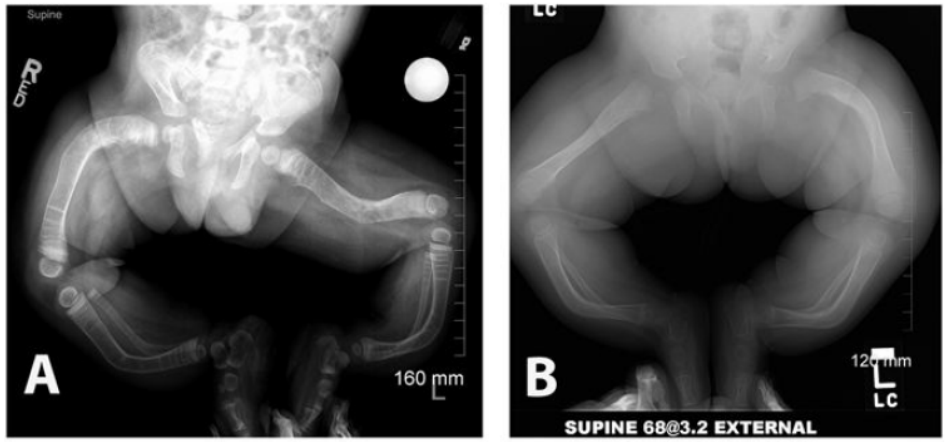
Osteogenesis Imperfecta (OI) – a rare disease from pediatric to adulthood with significant unmet medical need

Clinical rationale & unmet medical need

- Heterogenous rare disorder with 85% having dominant inherited mutations in genes for collagen 1 (COL1A1/COL1A2), causing varying degrees of severity and impact on life length
- Characterized by defective bone and cartilage causing fragile bone structure (brittle bone) leading to frequent fractures that can lead to deformities, pain and impacted mobility
- There are no approved medical treatments in OI



Significant unmet medical need

A. One-year-old infant diagnosed with severe type III OI. Note the severe bowing of the legs and the lack of bone modeling in both femurs and tibiae.

B. A nine-month-old infant with moderate type IV OI.

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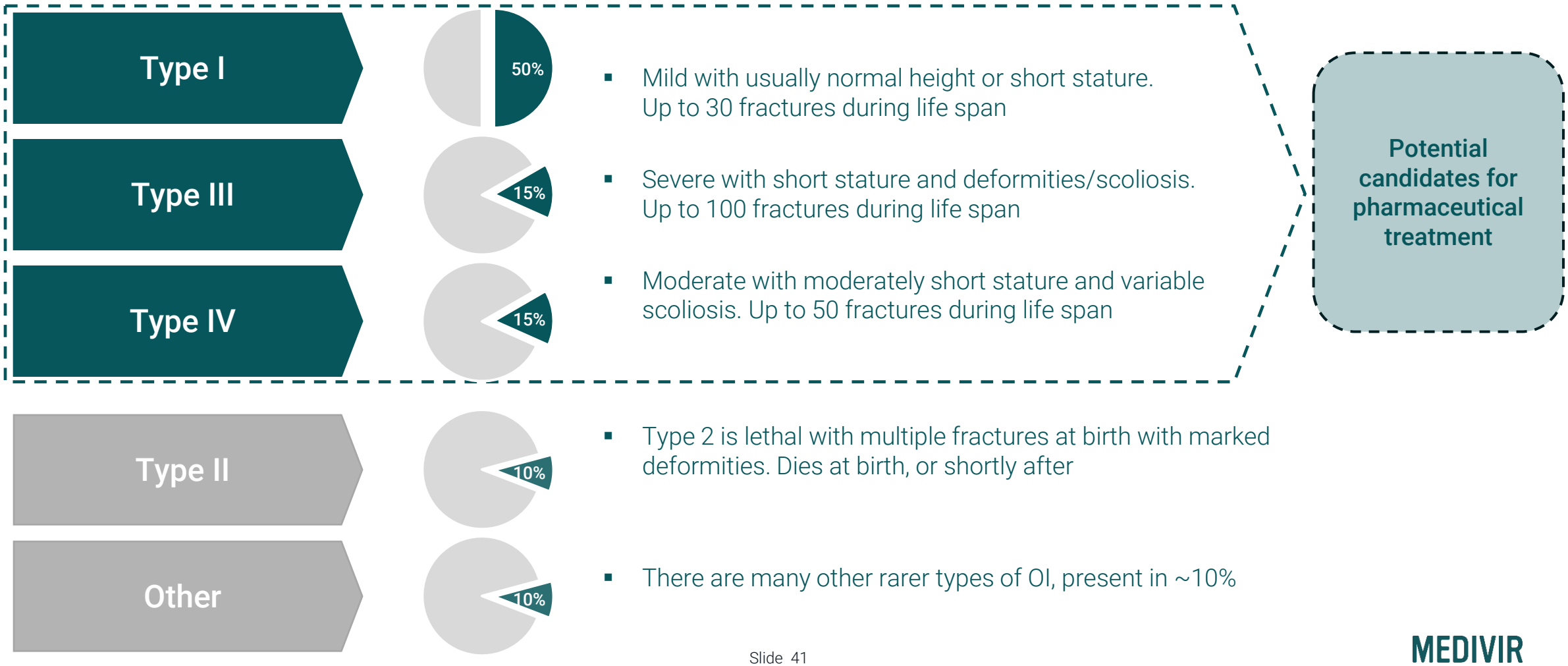
OI are divided into subtypes according to clinical severity [1,2]



[1]Sillence DO, Rimoin DL, Danks DM. Clinical variability in osteogenesis imperfecta-variable expressivity or genetic heterogeneity. Birth Defects Orig Artic Ser 1979; 15: 113–29.
[2]https://www.orpha.net/

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Bone remodelling is a continuous process requiring interplay between osteoclasts & osteoblasts

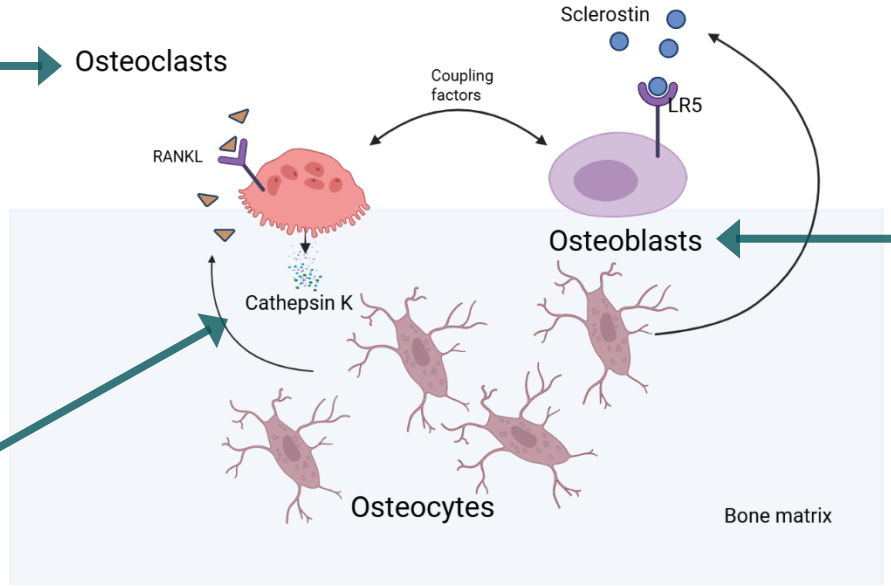
Osteoclasts

Cells responsible for resorbing old bone

Cathepsin K

Enzyme secreted by osteoclasts driving bone resorption

Elevated in OI



Osteoblasts

Cells responsible for synthesizing new bone.

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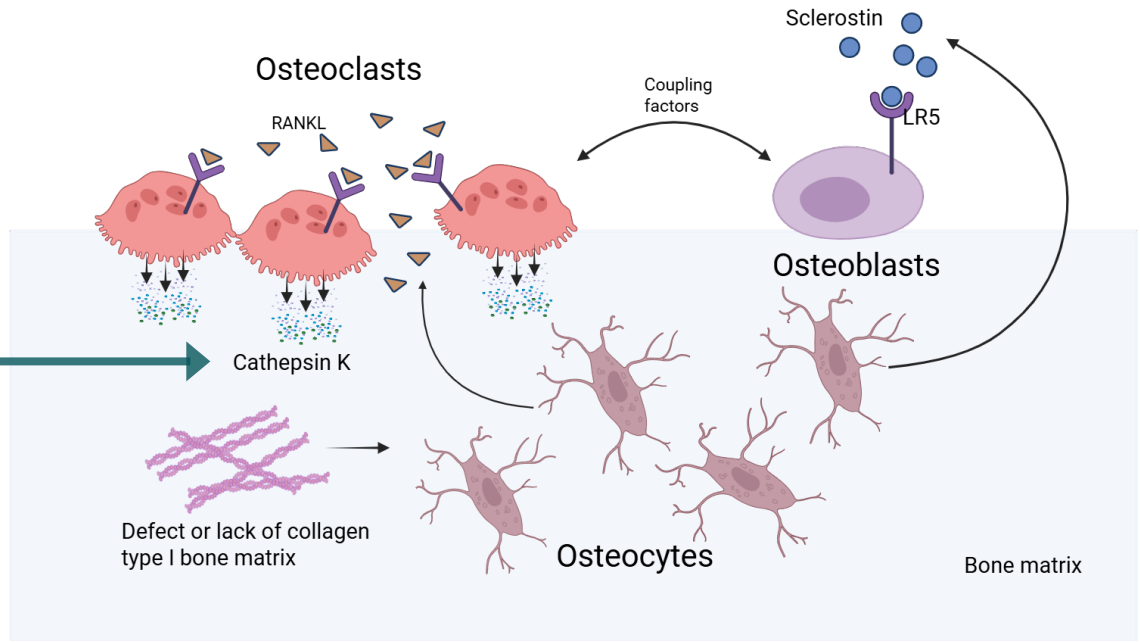
Cathepsin K activity is increased in Osteogenesis Imperfecta and drives increased bone resorption

1. Type I collagen is the major component in bone, making up ~90% of bone matrix. It is the skeleton of the bone and provides flexible strength and regulates the mineralization process
2. The OI mutations leads to reduced and/or defect Type I collagen resulting in increased bone resorption by osteoclasts and reduced formation of qualitative bone
3. Studies in children with OI show high levels of Cathepsin K



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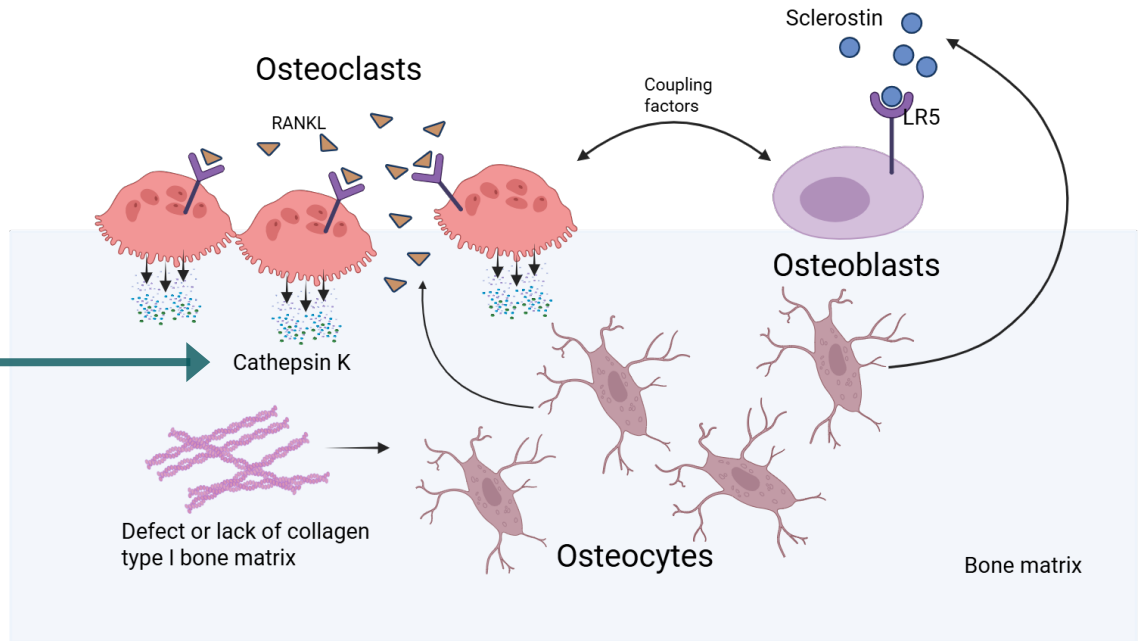
Rauch et al 2000, Baron et al 1983, Braga et al 2004; Garnero et al 2009; Rousseau et al 2010)

MIV-711, a selective cathepsin K inhibitor, restores balance between bone resorption and bone formation

1. MIV-711 is a highly selective inhibitor of Cathepsin K, preventing degradation of type I collagen
2. While MIV-711 inhibits the in OI increased bone degrading osteoclast activity, continuous bone remodeling is maintained as the osteoclast-osteoblast coupling is preserved
3. As a result, MIV-711 helps restore the balance between bone resorption and bone formation in the continues bone remodeling



MEDIVIR

Rauch et al 2000, Baron et al 1983, Braga et al 2004; Garnero et al 2009; Rousseau et al 2010)

Inhibiting cathepsin K prevents bone resorption and restores bone remodelling and formation of new bone

Cathepsin K inhibition

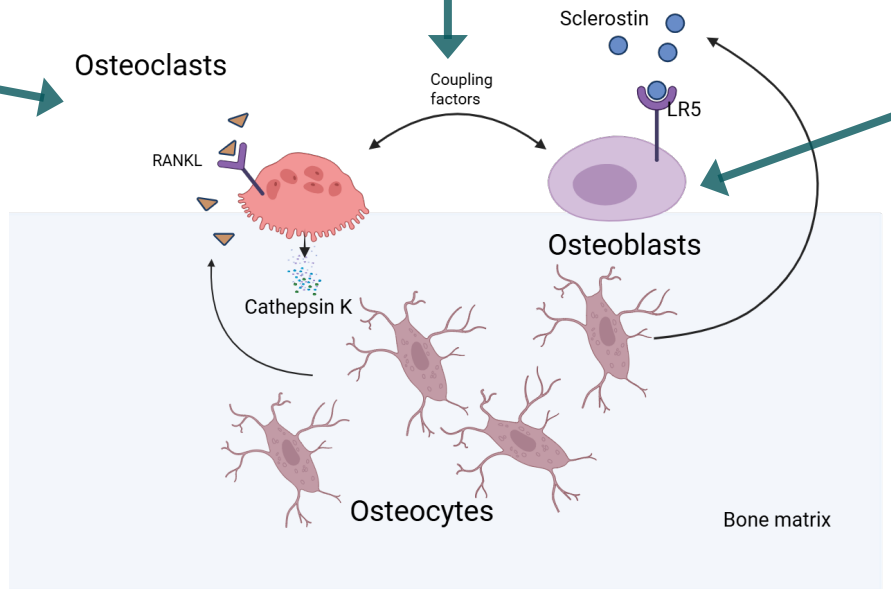
Prevents bone resorption by selective inhibition of cathepsin K

+ Effectively prevents bone resorption while saving osteoclast functionality
+ Preserves osteoclast – osteoblast coupling → induces formation of new bone

Bisphosphonates

Prevents bone resorption by killing osteoclasts

+ Effectively prevents bone resorption
- Lost coupling between osteoblast and osteoclast
- Negative impact on formation of new bone
- Not approved in OI



Anti-sclerostin mAb

Induces bone formation via osteoblast…

+ Effectively induces new bone formation
+ Indirect reduction of bone resorption
- Benefit diminishes after 6-12 months due to induction of escape pathway activation
- Failed phase 3 study in Q4 2025

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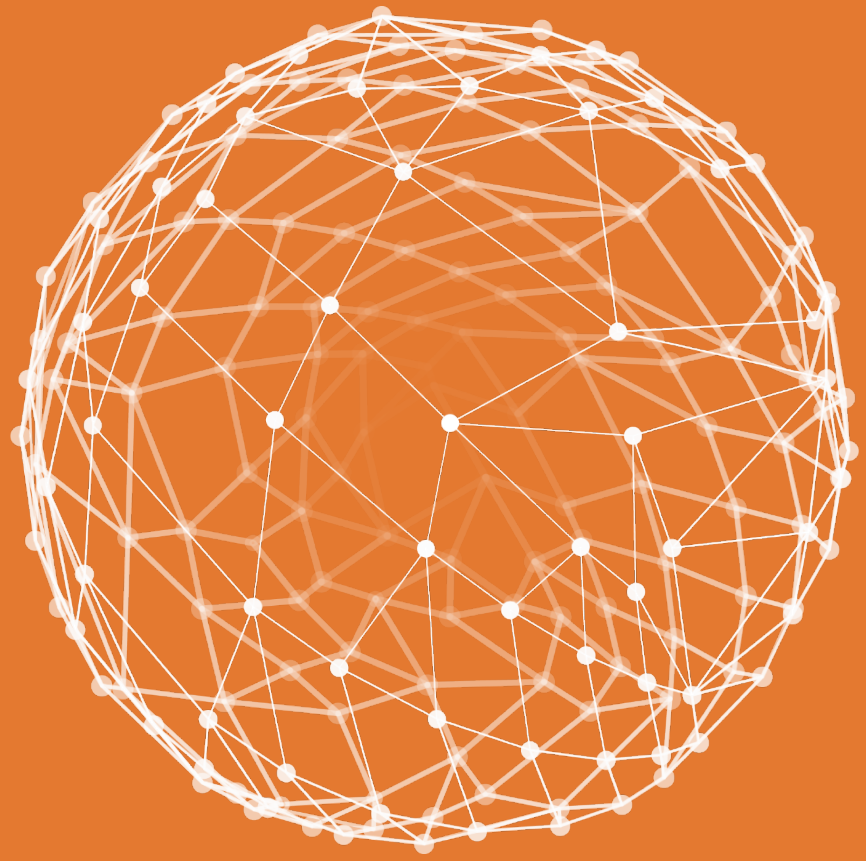
Proven disease-modifying benefit across multiple bone-related disorders setting up clinical PoC



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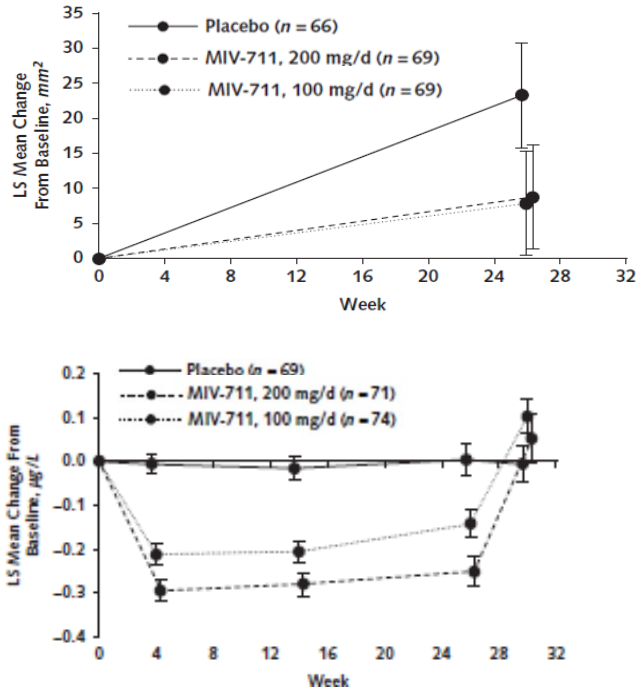
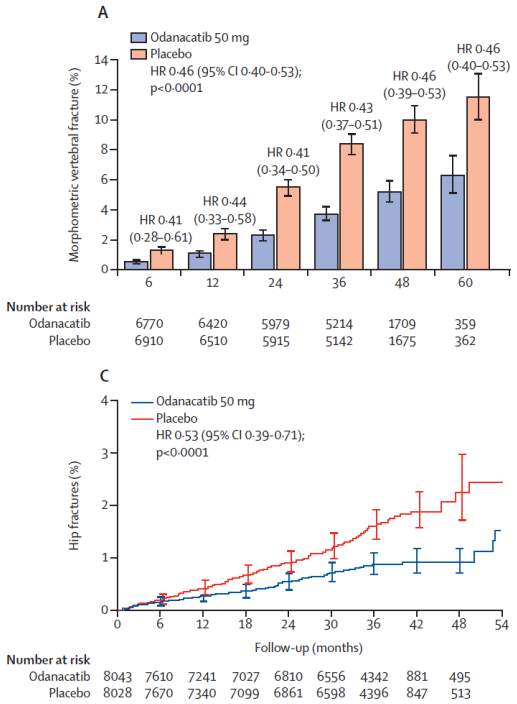
Cathepsin K inhibition showing significant benefit across multiple bone-related disorders

Cathepsin K inhibition – Significant bone & cartilage benefit in **Osteoarthritis**[1]



Cathepsin K inhibition – prevents fractures in **Osteoporosis**[2]



Cathepsin K inhibition – promising signals in **Osteogenesis Imperfecta**[3]

- Significant and dose dependent improvement in bone volume & quality vs placebo in OI mouse model
- Orphan Drug Designation granted by US FDA

[1]Conaghan et al, Annals of Internal Medicine 2019
[2]McClung et al., The Lancet Diabetes & Endocrinology, P899-911, Dec 2019
[3]Data on file

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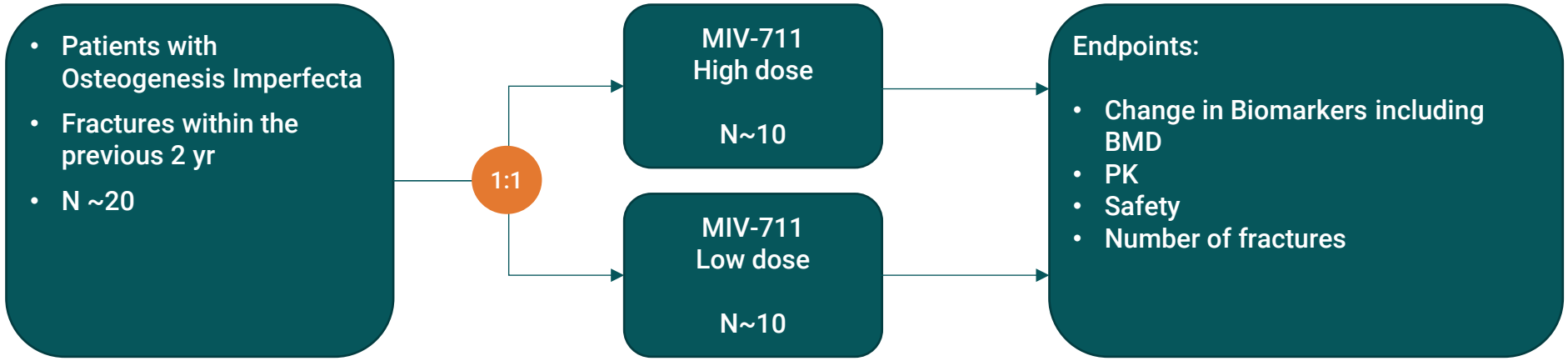
Draft design of phase 2 randomized POC study with MIV-711 in OI to inform next pivotal development phase



Phase 2 POC study in Osteogenesis Imperfecta

- ~20 patients randomized 1:1 to two dose arms with MIV-711 oral treatment once daily for 12 months
- Enrollment in Europe
- Patients eligible for this study are already known at sites positively impacting enrollment

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Highly experienced scientific expert council to support design of clinical development program

Dr. Andreas Kindmark

- Associate Professor and Senior Consultant in Endocrinology at Uppsala University Hospital, specializing in the investigation of skeletal metabolic disorders
- He is heading the Uppsala University Hospital units for National Highly Specialized care for OI and for Skeletal Dysplasias.
- His research interests span from epidemiological studies, through effects of hereditary factors influencing bone metabolism, and he heads a research group working to identify genes and genetic variants affecting bone health.

Dr. Richard Keen

- Director, Centre for Metabolic Bone Disease, Royal National Orthopaedic Hospital, Stanmore, UK
- He is the lead for adult metabolic bone disease in the Musculoskeletal GeCIP Domain of NHS England's 100,000 Genome Projectand Medical Advisor to the UK Brittle Bone Society.
- Published over 60 scientific papers in peer-reviewed journals

Dr. Oliver Semler

- Head of department of Rare Skeletal Diseases in childhood at Division of Paedriatic Endocrinology, Metabolic Diseases and Osteology, University of Cologne, Germany
- His clinical and research work focuses on osteogenesis imperfecta and other skeletal dysplasias.
- A highly active researcher with numerous publications spanning OI pathophysiology, treatment, and clinical management.

Dr. Bente Lomholt Langdahl

- Clinical Professor, Department of Endocrinology and Internal medicine, Aarhus University Hospital, Denmark
- She specializes in metabolic bone disorders with particular focus on osteoporosis and osteogenesis imperfecta.
- She is head of the Clinical Bone Research Center at Aarhus University Hospital.
- She has authored more than 300 peer-reviewed scientific papers, contributing extensively to the understanding of osteoporosis, rare bone diseases including osteogenesis imperfecta and genetics of bone disorders.

Dr. Marelise Eekhoff

- Internist-endocrinologist and professor at Amsterdam UMC, Netherlands, where she leads the center for rare bone disorders, including osteogenesis imperfecta, fibrodysplasia ossificans progressiva, genetic osteoporosis, fibrous dysplasia/MAS and Camurati Engelman among others.
- She is a highly active researcher, contributed extensively to scientific literature spanning rare skeletal diseases, pre- and clinical pathophysiology, treatment and clinical management.
- She is responsible for a significant number of clinical studies.

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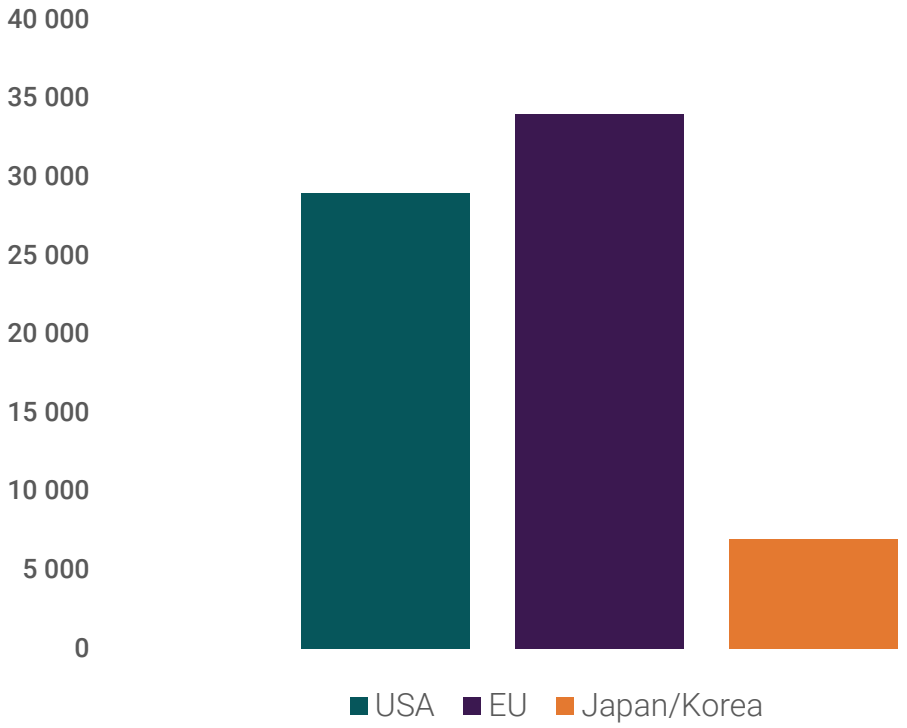
Significant market opportunity with no approved treatment options for patients with Osteogenesis Imperfecta[1,2]

Estimated prevalent OI population



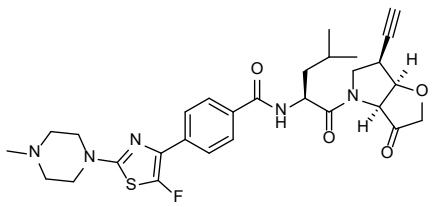
Sizeable market opportunity – MIV-711

- Significant unmet medical need with no approved treatment options
- Anti-sclerostin antibody (setrusumab), phase 3 failure in Q4 2025
- Market opportunity across USA, EU and Japan/Korea >$2.5bn annually
- Potential for rare Pediatric Disease Designation & Priority Voucher

[1] Sillence DO, Rimoin DL, Danks DM. Clinical variability in osteogenesis imperfecta-variable expressivity or genetic heterogeneity. Birth Defects Orig Artic Ser 1979; 15: 113–29.
[2]https://www.orpha.net/

MIV-711 – Highly selective cathepsin K inhibitor in development for patients with Osteogenesis Imperfecta (OI)

3rd generation, highly selective cathepsin K inhibitor

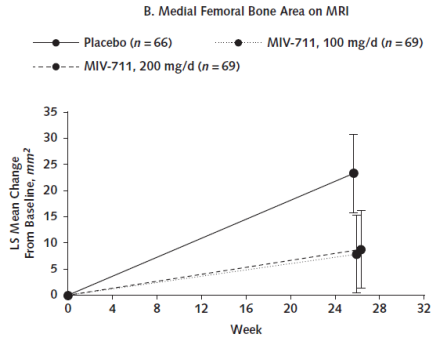


Inhibits cathepsin K, the main protease of bone-degrading osteoclasts, to restore bone matrix quality

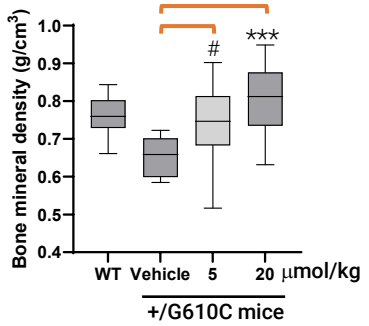
- ~250 subjects in phase 1/2 Osteoarthritis study, confirming ability to prevent cartilage degradation
- PoC established in Osteogenesis Imperfecta animal model, increasing bone volume & quality
- MOA enabling long-term bone formation & anti-resorption

Proven ability to prevent cartilage & bone degradation & improve bone quality

OA – prevention of cartilage loss[1]



OI – Improved bone volume & quality[2]



Phase 2 proof-of-concept study underway With ODD granted

- Significant clinical exposure and proven benefit across multiple bone-related diseases
- Orphan drug designation (ODD) approved in the US
- Funding completed for phase 2 proof-of-concept study

Total market opportunity in Osteogenesis Imperfecta >$2.5bn across key markets

- At least 70.000 potential patients estimated across the US, EU and Japan and Korea
- No approved treatment options available
- Potential for rare pediatric disease designation (RPDD)

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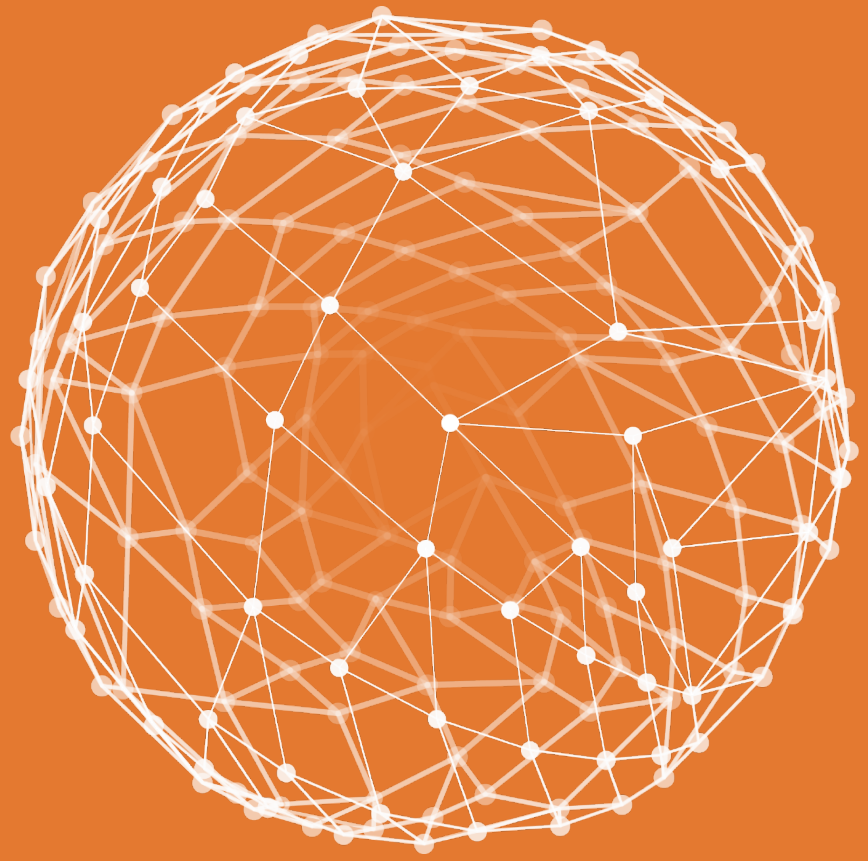
[1]Conaghan et al, Annals of Internal Medicine 2019
[2]Data on file

PARTNERED PROGRAMS

MEDIVIR

VBX-1000 (MIV-701) – The first, potential disease-modifying treatment of periodontal disease in dogs



MEDIVIR

Exclusive licensing agreement with French biotech Vetbiolix for the development of MIV-701 (VBX-1000) in animal health

Medivir's partner Vetbiolix announces initiation of randomized, placebo-controlled study to confirm clinical benefit with VBX-1000 (MIV-701)

2026-02-12

- Speedy inclusion of the first 10 dogs, out of a total of 51, within one month of study start
- Top-line data expected by Q4 2026
- Blockbuster potential as the first disease-modifying therapy to halt bone loss in canine periodontitis

Stockholm, Sweden — Medivir AB (Nasdaq Stockholm: MVIR), a pharmaceutical company focused on developing innovative treatments in areas of high unmet medical need, announced today that its partner Vetbiolix, a France-based veterinary biotechnology company, has initiated First Patient First Visit in a pilot randomized, double-blind, placebo-controlled, dose-ranging clinical study evaluating VBX-1000 (MIV-701) for efficacy and safety in dogs with alveolar bone loss due to Stage 2–3 periodontitis.

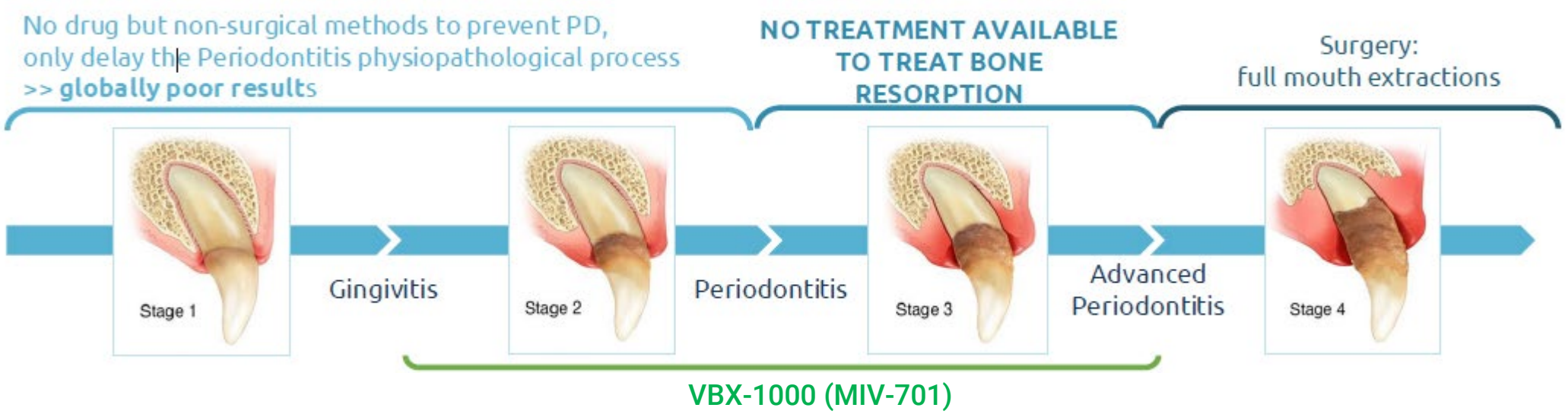
Positive POC established in periodontitis in dogs with topline results from randomized, placebo-controlled study expected during Q4 2026

Global, exclusive, licensing agreement to develop and commercialize MIV-701 (VBX-1000) in animal health

Substantial revenue upside through future royalties on net sales and a meaningful share of any partnering payments from third-party collaborations.

MEDIVIR

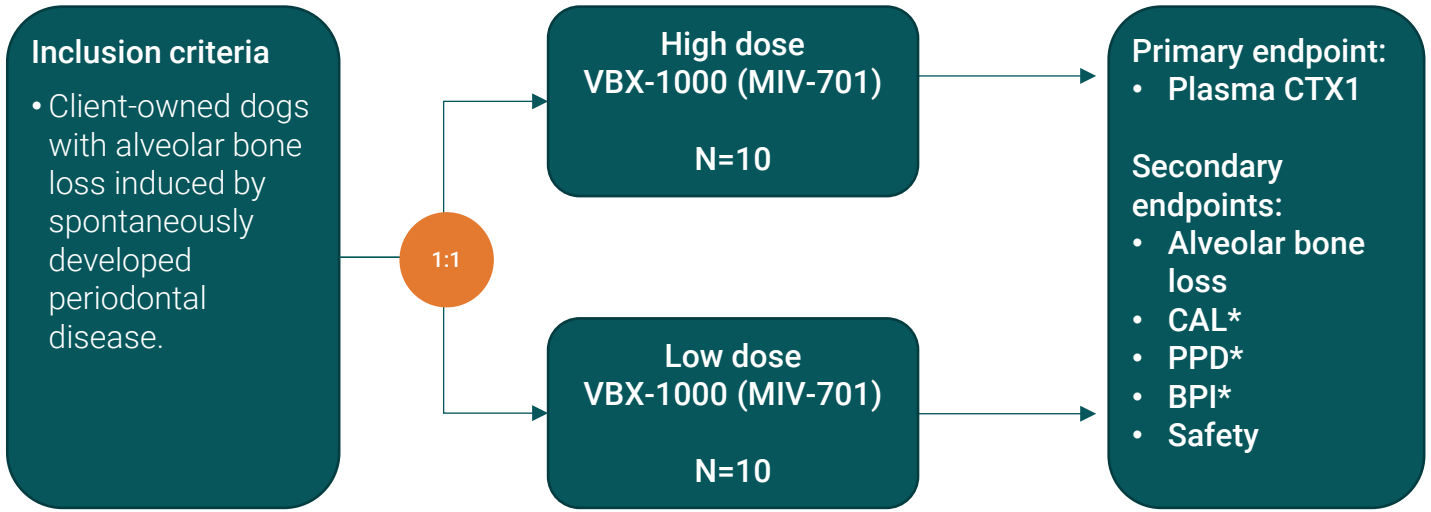
VBX-1000 (MIV-701) – Potential game changer for the treatment of periodontitis in animal health



- 80% of all dogs and cats over 3 years suffer from periodontal disease (PD), causing pain, tooth loss & infections
- No therapeutic treatments available to stop/reduce bone resorption in dogs and cats
- VBX-1000 (MIV-701) targets periodontitis as the first disease-modifying treatment.

VETBIOLIX NEW ROUTE TO ANIMAL HEALTH MEDIVIR

Landmark POC Study in periodontal disease (PD) in dogs showing significant & clinically relevant improvements



Efficacy assessment on primary and secondary end-point measurements = Day 90 vs baseline

Study outcomes:

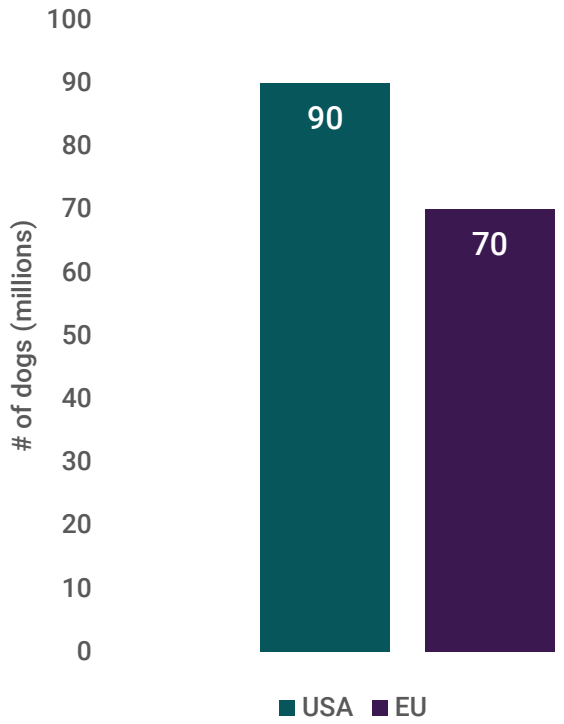
- The first treatment to show disease-modifying benefit
- Highly significant effect on the primary end-point of the study (plasma CTX1) at high dose as proof of target engagement in the patient
- Significant & clinically relevant improvements on the secondary end-points at high dose
- No safety concerns identified to date

VETBIOLIX NEW ROUTE TO ANIMAL HEALTH MEDIVIR

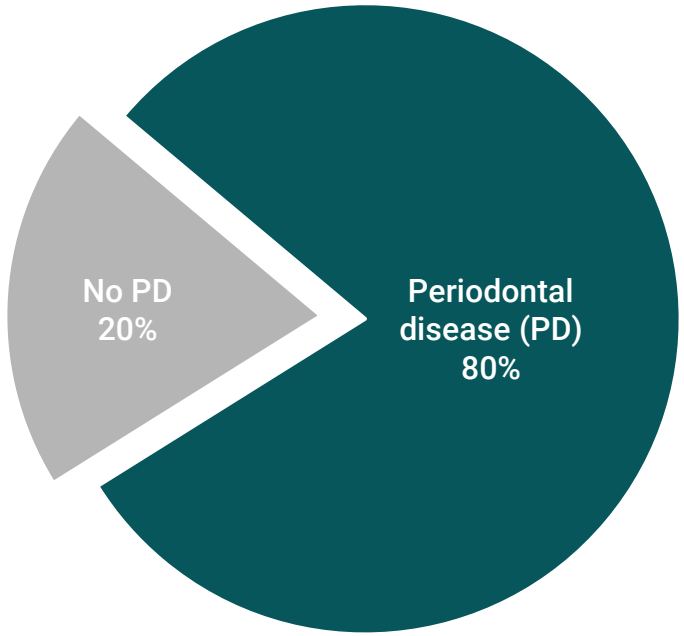
*CAL = Clinical Attachment Loss, PPD = Periodontal Probing depth, BPI = Bleeding on probing Index

Significant financial upside potential through royalty revenues & share of potential Vetbiolix partnership payments

Estimated total dog population



Share of dogs >3 years with PD

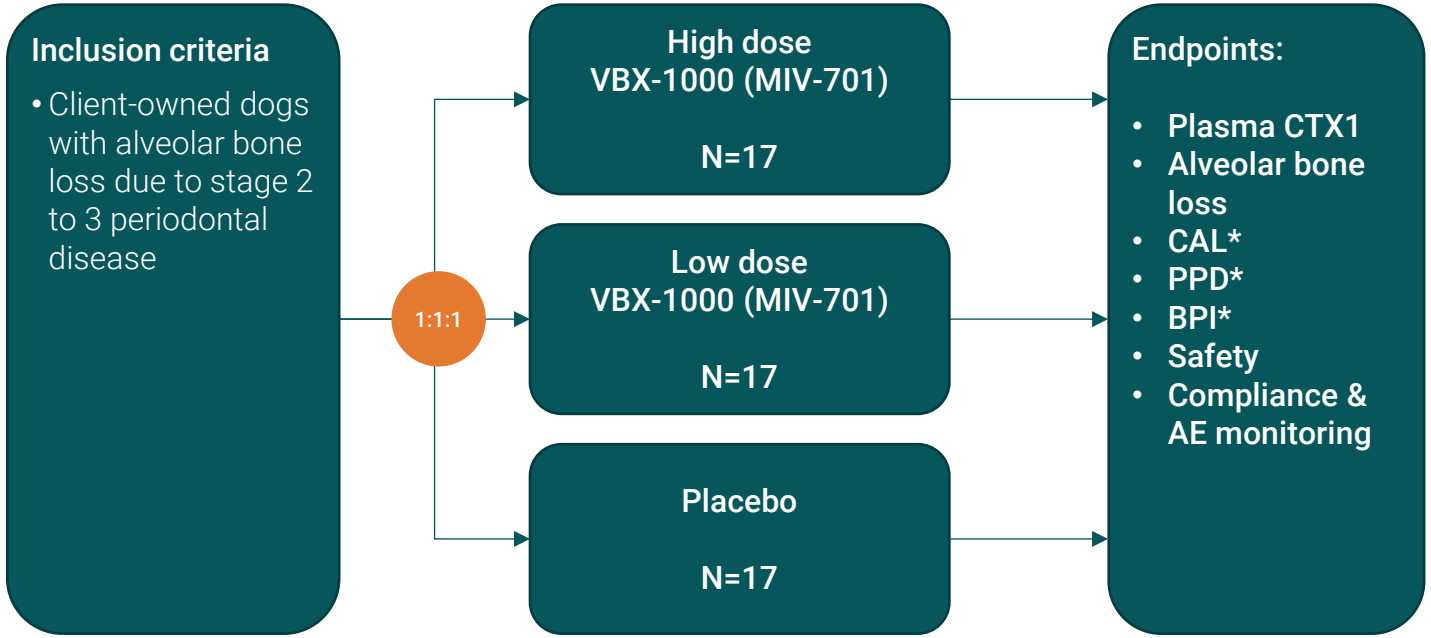


Sizeable market opportunity – MIV-701

- Significant unmet medical need with no approved treatment options
- Blockbuster potential for VBX-1000 (MIV-701) as the first & only disease-modifying treatment in development
- Significant financial upside potential through royalties & substantial share of potential partnership payments.
- Potential for annual royalty revenues equivalent to current market cap five years after global launch.

VETBIOLIX NEW ROUTE TO ANIMAL HEALTH MEDIVIR

Randomized, double-blind, placebo-controlled pilot study in dogs to confirm the efficacy of VBX-1000 (MIV-701)



Efficacy assessment on primary and secondary end-point measurements = Day 90 vs baseline

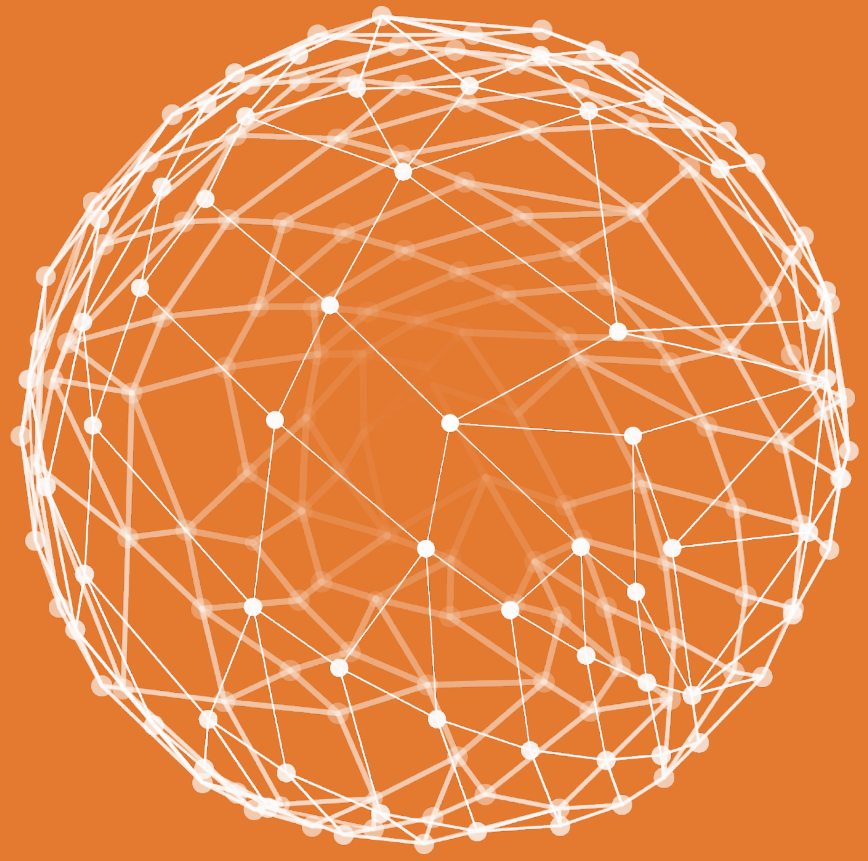
Estimated study timelines:

- 10 dogs dosed to date (Feb 12)
- Top-line results expected during Q4 2026

VETBIOLIX NEW ROUTE TO ANIMAL HEALTH MEDIVIR

*CAL = Clinical Attachment Loss, PPD = Periodontal Probing depth, BPI = Bleeding on probing Index

Remetinostat – Phase 3 ready topical HDAC-inhibitor for non-melanoma skin cancer



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Remetinostat – Efficacy and safety shown in 3 skin cancers

Three Phase II trials completed

Cutaneous T-Cell Lymphoma (MF-CTCL)

- Open label, multicenter Phase II study (60 patients) results showed 40% ORR, and reduced pruritus (itching) in 80% of patients

Basal Cell Carcinoma (BCC)

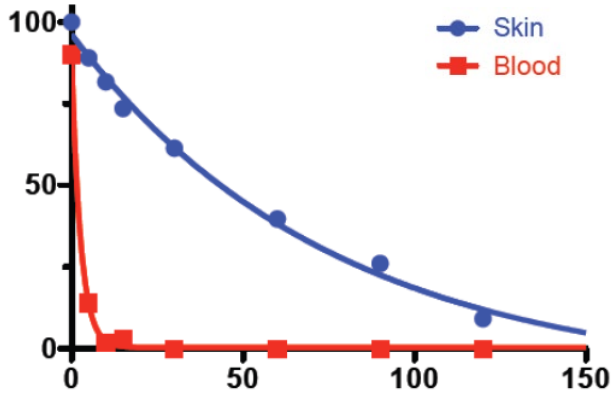
- Open label Phase II study (25 patients, Stanford ISS) results showed 70% ORR

Squamous cell Carcinoma (SCC)

- Open label Phase II study (4 patients, Stanford ISS) results showed 100% ORR

Unique topical HDAC-inhibitor

Stability of remetinostat



- Rapid breakdown by esterases in human blood (t½ ~4 mins)
- Negligible levels of systemic exposure translates to reduced risk of HDACi class-associated toxicities

Global, exclusive licensing agreement signed with Biossil, Inc

1)

MEDIVIR

Medivir enters exclusive licensing agreement with Biossil, Inc. for remetinostat

Medivir enters exclusive licensing agreement with Biossil, Inc. for remetinostat

2025-10-23

Stockholm, Sweden — Medivir AB (Nasdaq Stockholm: MVIR), a pharmaceutical company focused on developing innovative treatments for cancer in areas of high unmet medical need, announces today that it has entered into an exclusive licensing agreement, through which Biossil, Inc. will receive global, exclusive development rights for remetinostat, a clinical-stage topical HDAC inhibitor. Biossil is a Toronto-based AI-native drug developer focused on developing novel therapies for heterogenous diseases with urgent unmet medical needs.

Positive phase 2 data in basal cell carcinoma (BCC) and cutaneous T-cell lymphoma (CTCL)

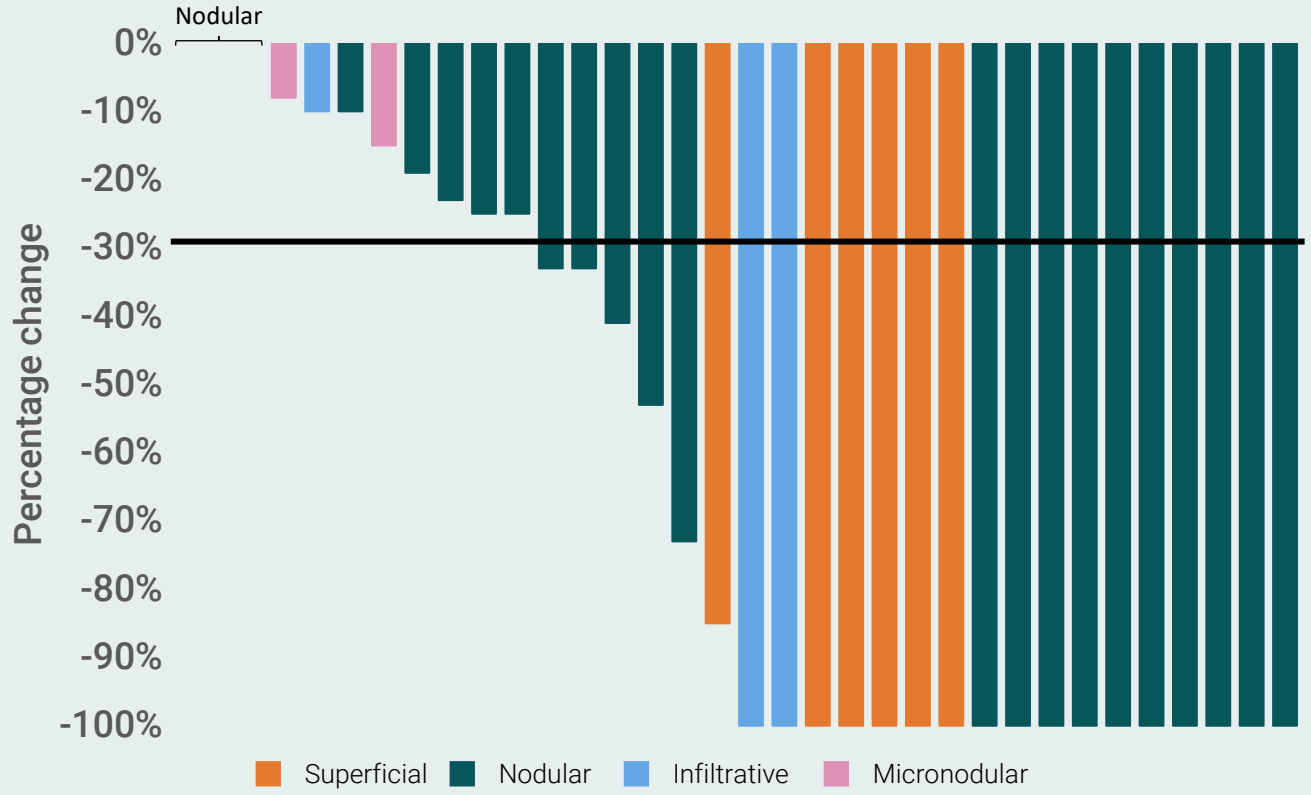
Global, exclusive, licensing agreement to develop and commercialize remetinostat

Total, potential milestone payments of approximately USD 60 million
Mid-single digit royalties on future net sales & sub-licensing revenue share.

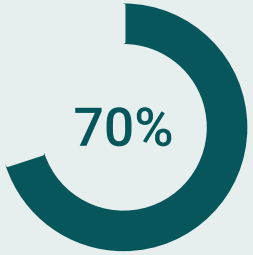
MEDIVIR

Remetinostat is an effective topical treatment for reducing BCC disease burden in a clinically significant manner

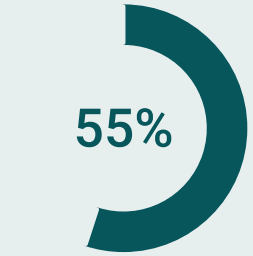
Encouraging efficacy across all subtypes of BCC lesions



>50% of lesions achieving complete pathological resolution across full cohort



Overall response rate

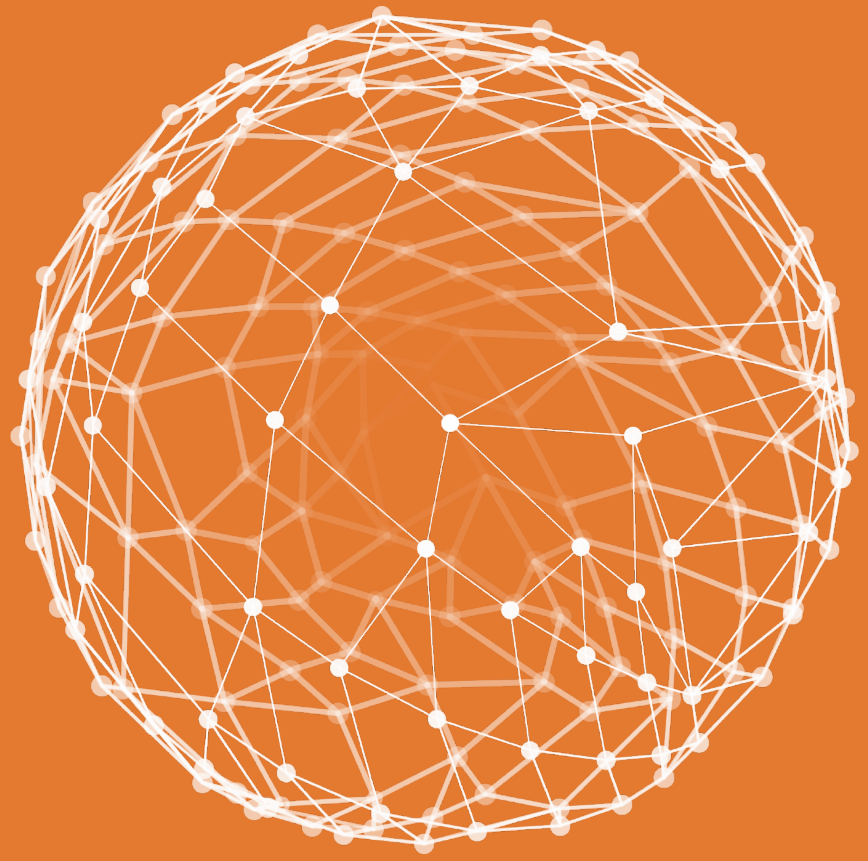


Complete pathological resolution

MEDIVIR

[1]J. Kilgour et al, Clinical Cancer Research published online 6 August 2021

MET-X – AMR resistance bypass drug candidate



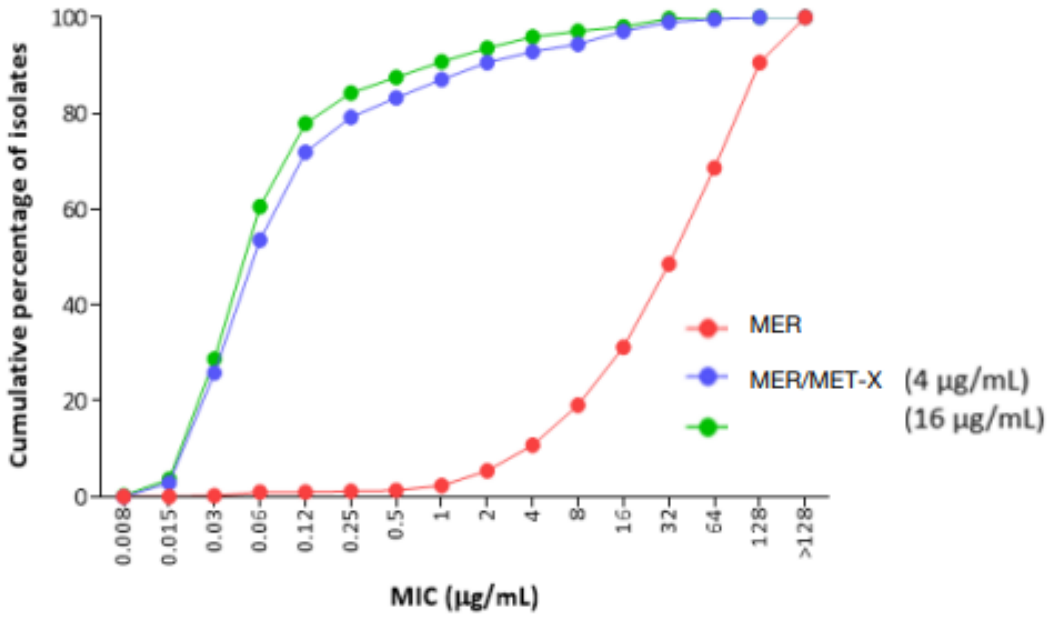
MEDIVIR

MET-X (MBLI) – FDA QIDP Designation received & sub-licensing agreement signed to enter clinical development

Potential best-in-class Metallo-β-Lactamase Inhibitor

- MET-X is a potent broad-spectrum MBL inhibitor in combination with β-lactams to restore their activity, targeting one of the most serious global threats from AMR. (Anti Microbial Resistance)
- Moving towards clinic, received FDA QIDP designation in 2023
- Medivir to receive revenue share on all commercialisation revenue by Infex Therapeutics
- In 2025, Infex signed a license agreement with Venus Remedies Ltd for the clinical development, set to receive upfront, milestone and double-digit royalty payments

MET-X restores activity of Meropenem*



*Restoration of meropenem activity in critical threat Gram-negative pathogens (519 clinical isolates of MBL-positive Enterobacterales). Clinical isolate panel containing NDM (n=385), IMP (n=44) and VIM (n=90) producers

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Thank You!