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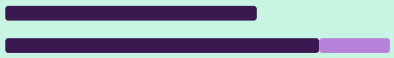
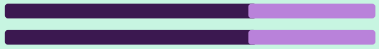
A pipeline of first-in-class programs targeting patient populations without approved treatment options

PROJECT	PARTNER	DISEASE AREA	PRE-CLINICAL / PH 1 / PH 2 / PH 3 / ON MARKET	FINANCIALS	POTENTIAL NEXT EVENT(S)
IN-HOUSE PROGRAMS					
Fostroxacitabine bralpamide	In-house development	HCC (mono) HCC (combo)		100% Medivir	▪ Phase 2 start
MIV-711	In-house development	OI Perthes disease		100% Medivir	▪ Phase 2 PoC study ▪ Phase 2 PoC study
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	▪ Phase 2/3 study start
MIV-701	Vetbiolix	Periodontal disease in dogs		Royalties & revenue share agreement on Vetbiolix partnering	▪ Phase 2 study results
MET-X	Infex Therapeutics	Critical MBL Infections		Revenue Share Agreement	▪ Phase 1 study start

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Main Projects & Potential New indication

- Fostrox in primary liver cancer: Blockbuster potential
- MIV-711 in Osteogenesis Imperfecta: Blockbuster potential
- MIV-701 in periodontitis in dogs: around SEK700m (Peak royalty)

- MIV-711 in Perthes disease (Perthes sjukdom)
  - Orphan Drug Designation & Rare Pediatric Disease Designation

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Transformational progress

Two directed share issues of SEK 140 million & SEK 45 million to Carl Bennet AB and other institutional investors, enabling MIV-711 phase 2 studies in two rare, bone-related disorders, Osteogenesis Imperfecta & Perthes Disease and providing funding into 2030

Phase 1b/2a data for fostrox + lenvatinib published in Clinical Cancer Research, first patient dosed in the randomized FLEX-HCC phase 2 study with recruitment progressing to plan for topline results in 2027

VBX-1000 (MIV-701) placebo-controlled study to confirm disease-modifying benefit & unlock blockbuster potential progressing well, 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
- 3 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. Angelica Loskog, Ph D, Clin. Immunology**
  - CEO Lokon Pharma & scientific advisor at Nexttobe VC
  - 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.

- **Dr. Kristian Tryggvasson, PhD, Biology, MBA**
  - Founder BioLamina and Alder Therapeutics
  - More than 20 years' of industrial drug and product development experience

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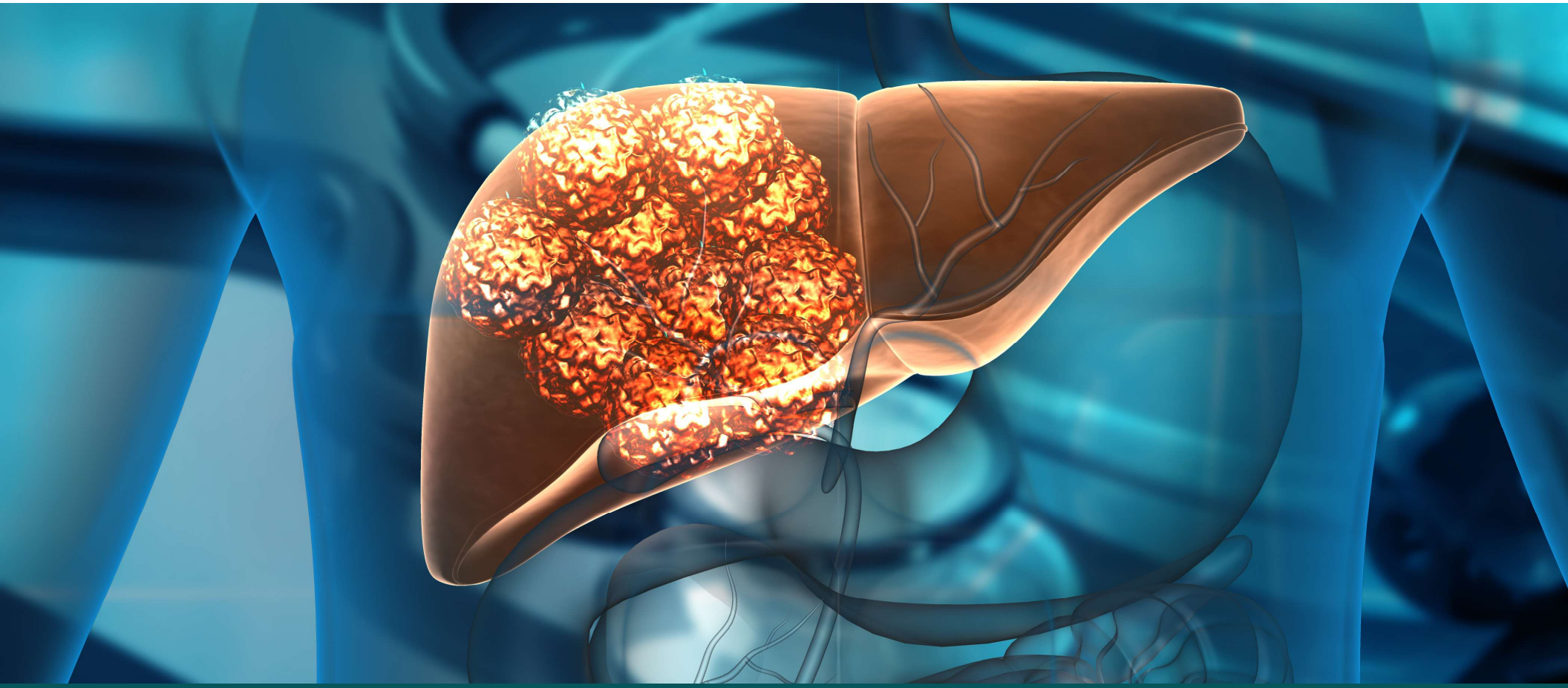

Improving life for advanced liver cancer (HCC) patients

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

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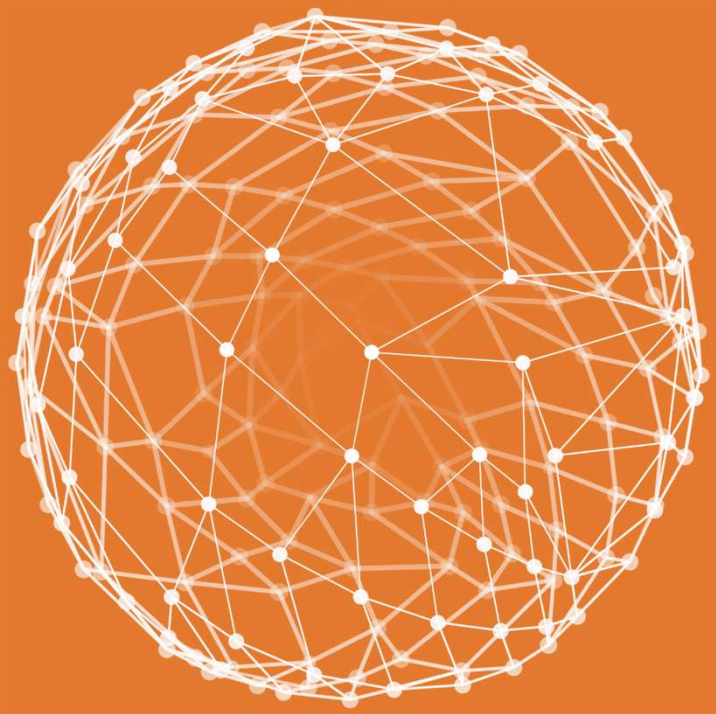
First-to-market opportunity in 2nd line HCC market valued >$2.5bn



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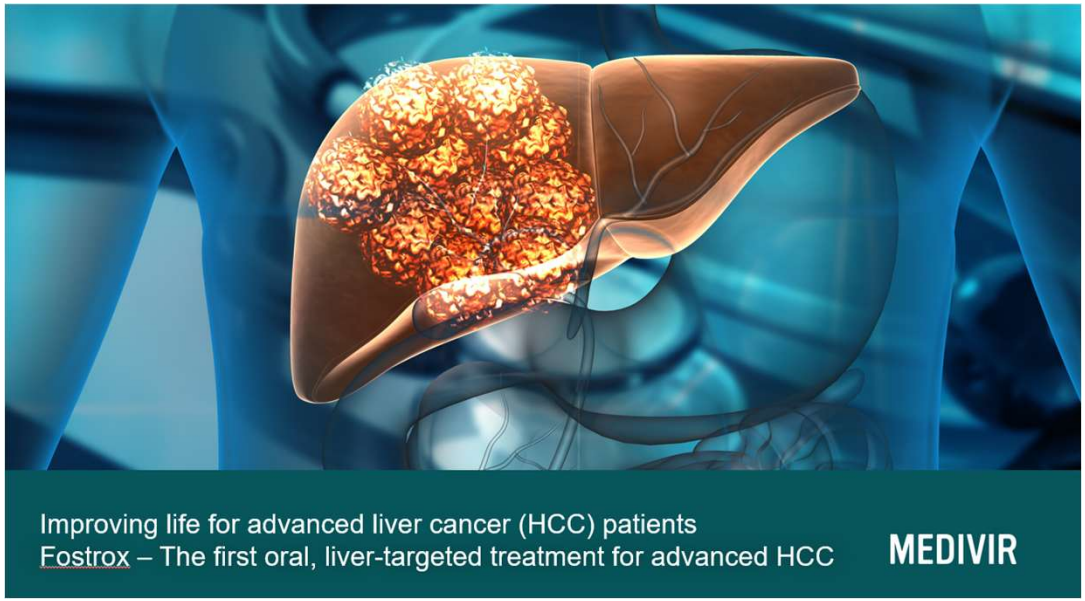
Primary Liver Cancer (HCC)

- 3rd leading cause of cancer death globally
- 70,000 new patients per year in Europe and USA
- High unmet medical need for effective second-line treatment



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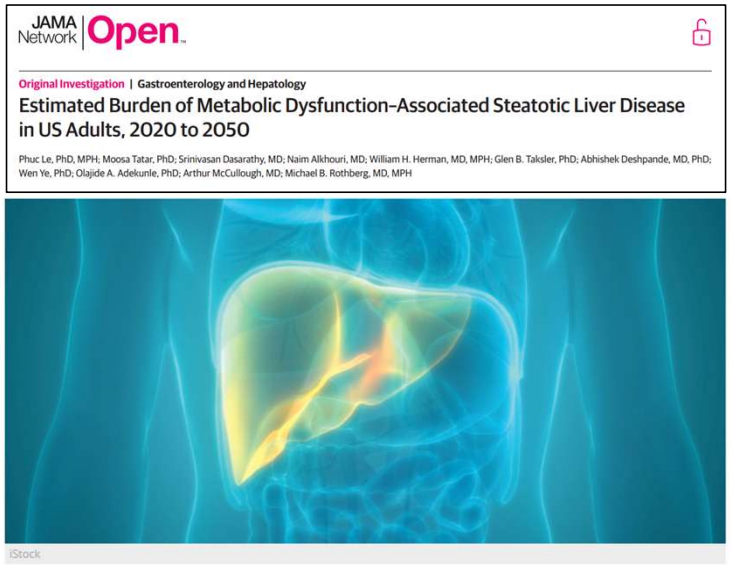
45% of US adults are obese
More than 25% have Fatty Liver Disease
Fatty Liver Disease increases
risk of Liver Cancer 17-fold

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

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SCIENCE NEWS

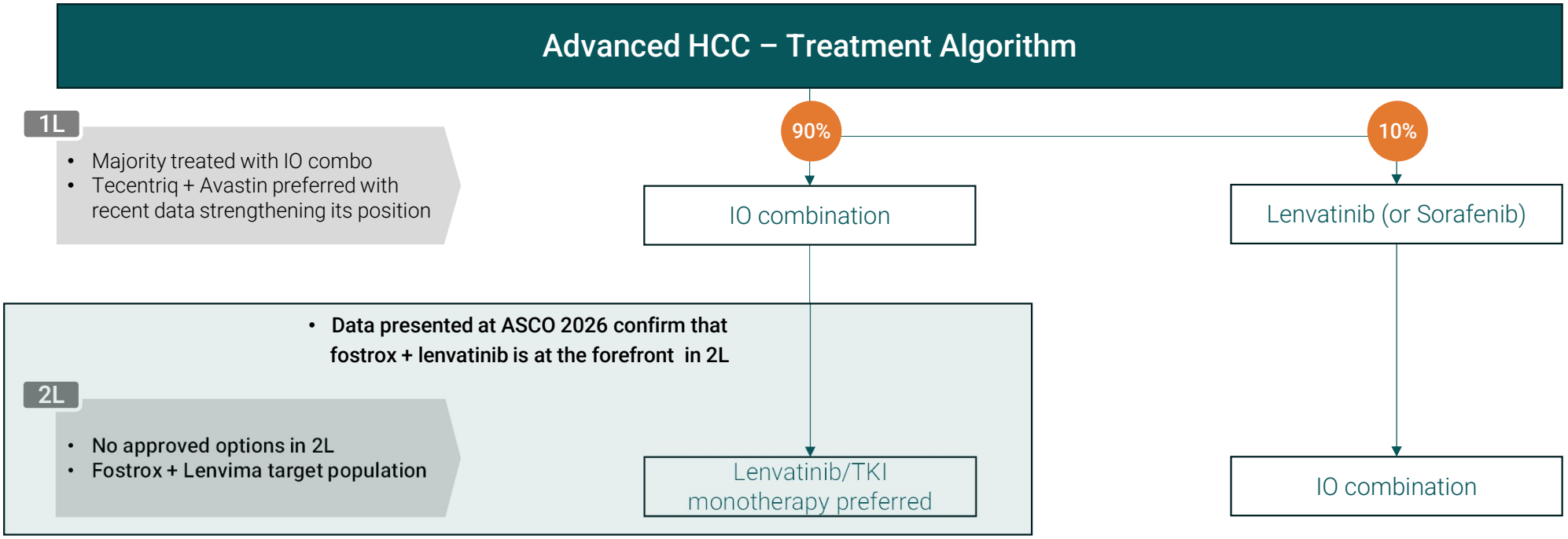
Fatty Liver Disease Is Expected to Skyrocket By 2050

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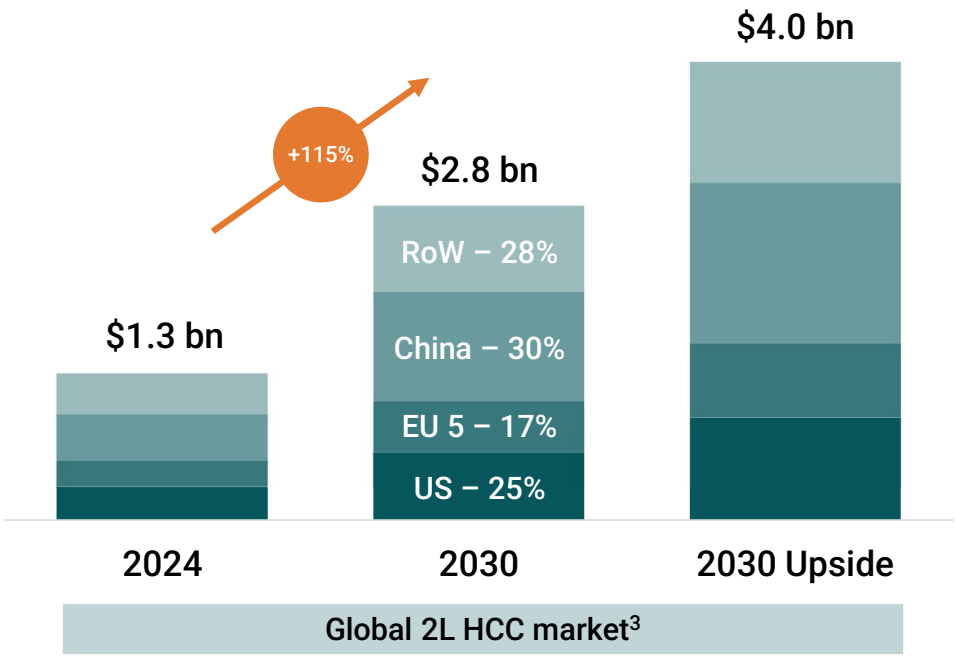
2nd line HCC – a ~$3bn commercial opportunity[3]



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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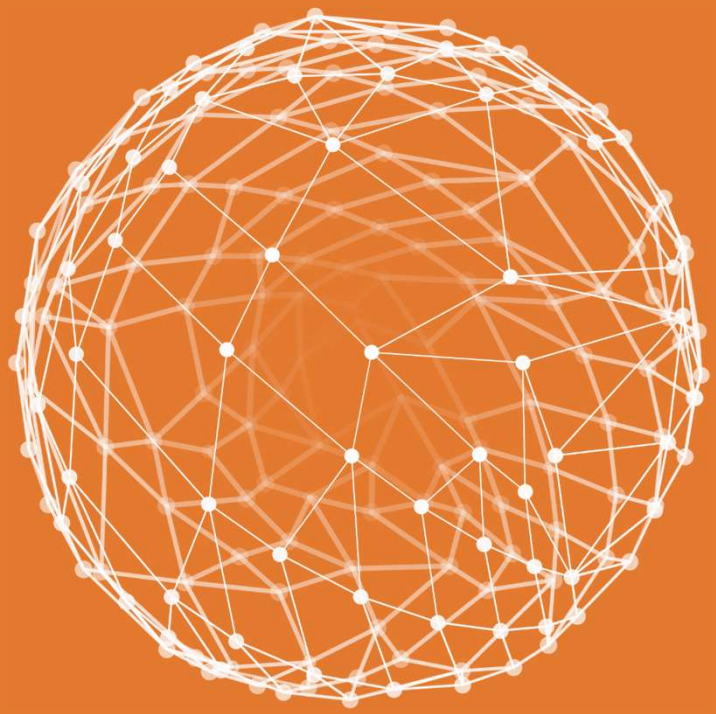
Fostrox – tailored for the specific needs of HCC



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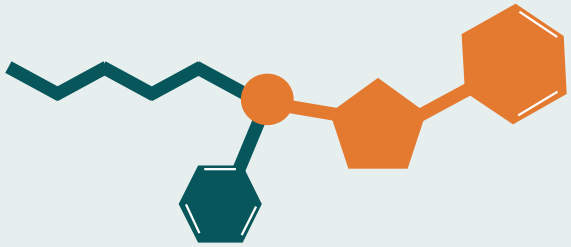
Fostrox – designed to selectively kill tumor cells in the liver

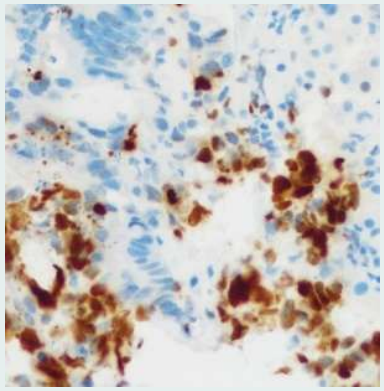
Prodrug transports inactive payload to the liver, where it is rapidly activated by liver enzymes[1]



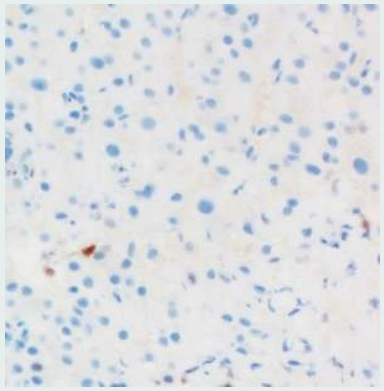
Liver-guided delivery – prodrug

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

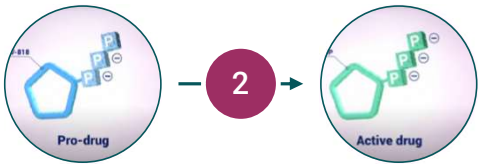
Fostrox taken as oral tablet

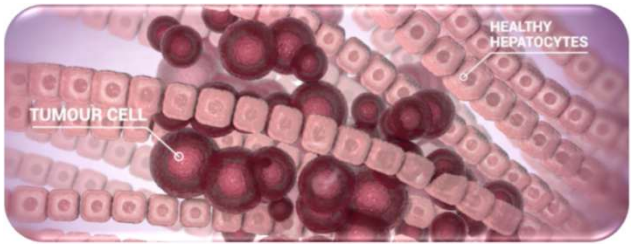
Prodrug transports inactive payload via first-pass metabolism to the liver[1]

Mechanism of Action video

Rapidly activated by liver enzymes

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





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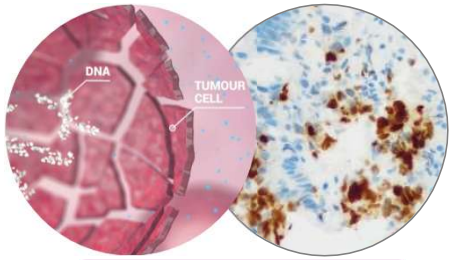
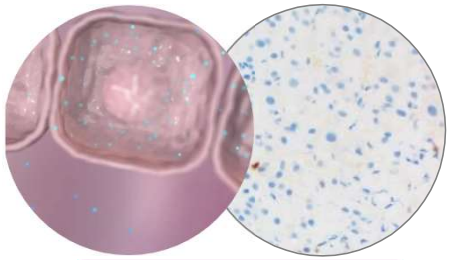


Cell death in tumor cells

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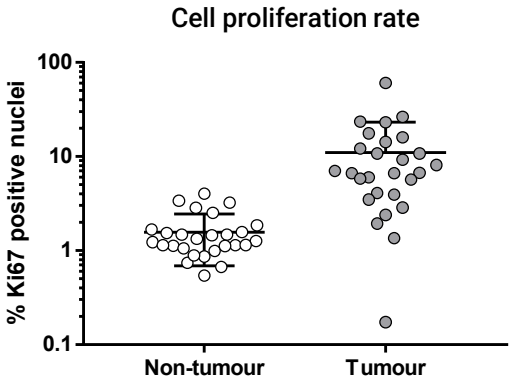
100-fold higher liver targeting vs IV administration & selective DNA damage in tumor cells enabling highly targeted mechanism

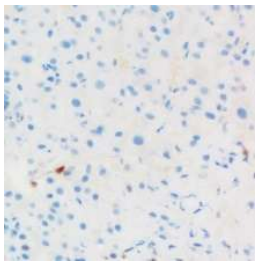
>100-fold higher liver targeting with fostrox than iv troxacitabine in rats

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**

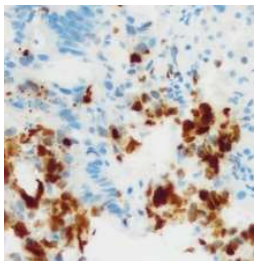
Liver tumor cells divide significantly more often than non-tumor cells[1]



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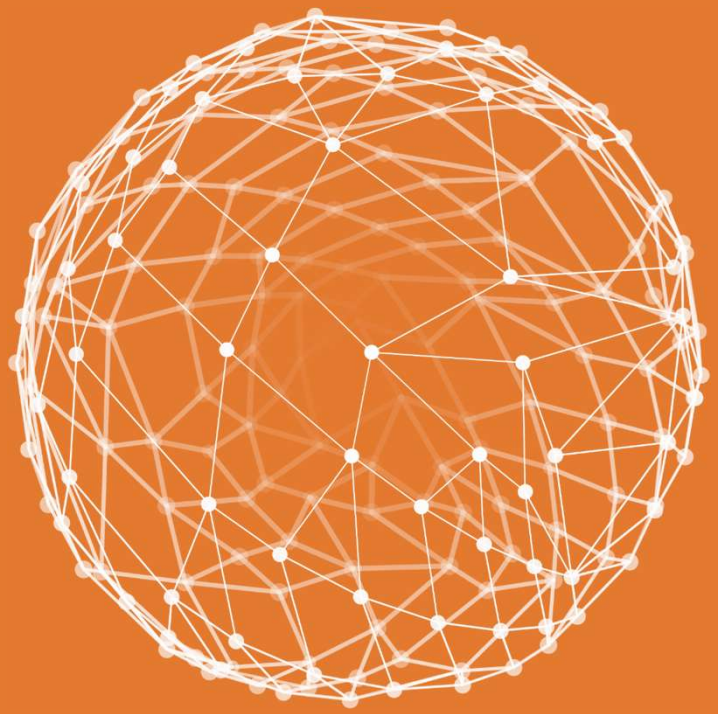
Fostrox + Lenvima shows promise of improved outcomes in 2L HCC



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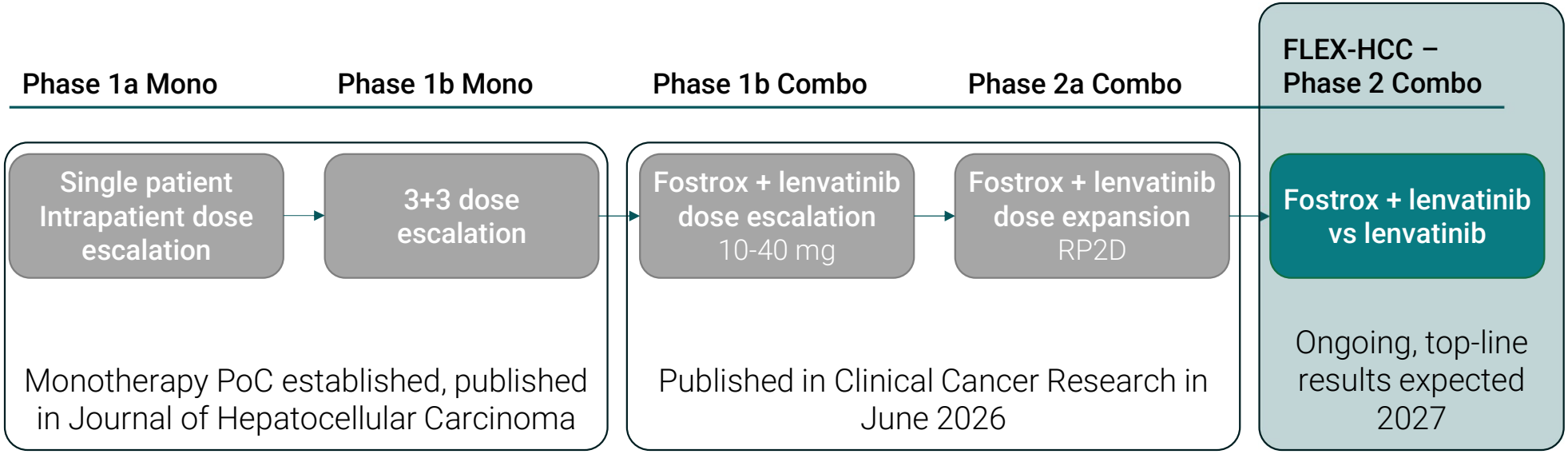
Fostrox Clinical Development Program; monotherapy PoC established, focus on combination approach in 2L HCC



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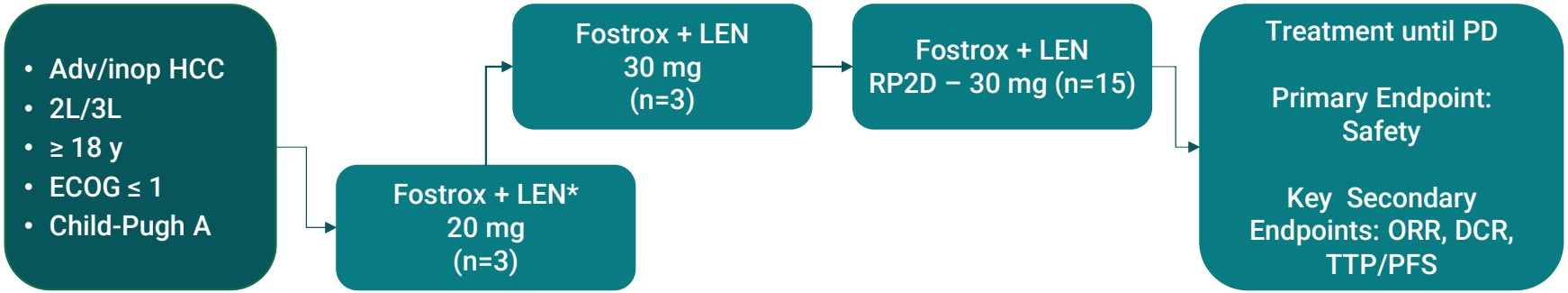
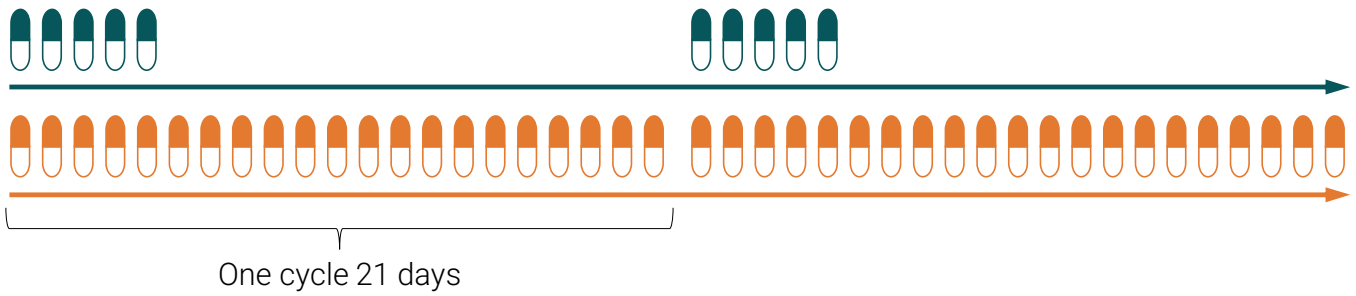
Fostrox + Lenvima phase 1b/2a study design



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, data published in Clinical Cancer Research[1]

CLINICAL CANCER RESEARCH | CLINICAL TRIALS: NOVEL MECHANISMS

A Phase Ib/IIa Study of Fostrox in Combination with Lenvatinib as Second-line Therapy in Patients with Advanced Hepatocellular Carcinoma

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

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[1]Chon et al., Clinical Cancer Research 2026.

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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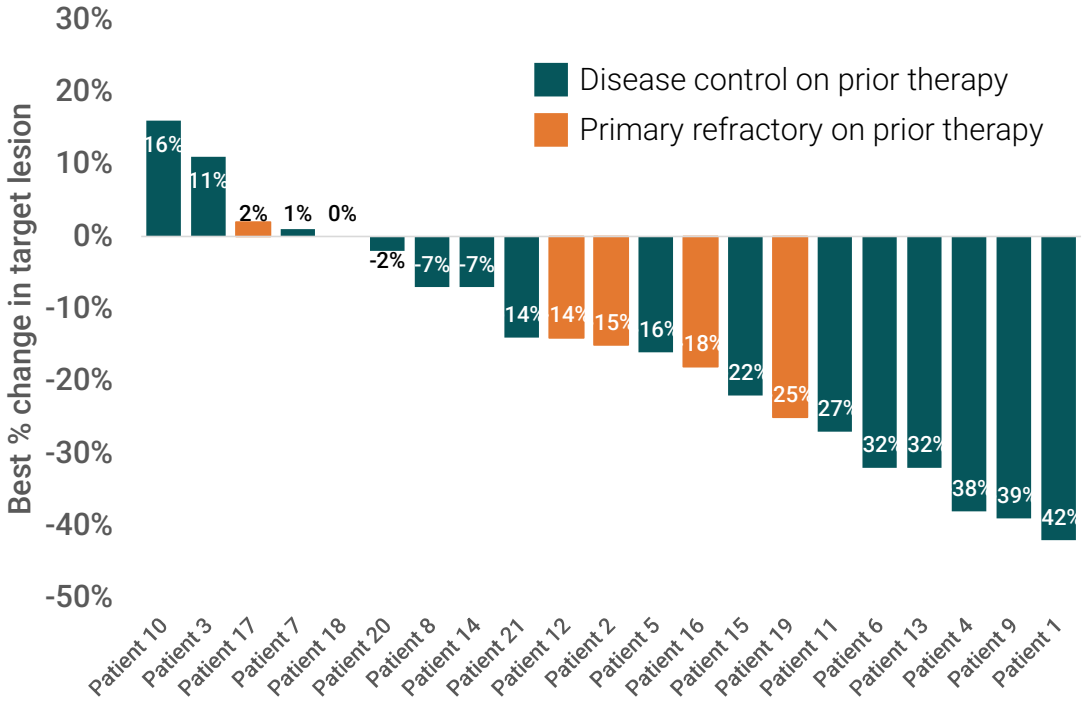
[1]Chon et al., Clinical Cancer Research 2026.

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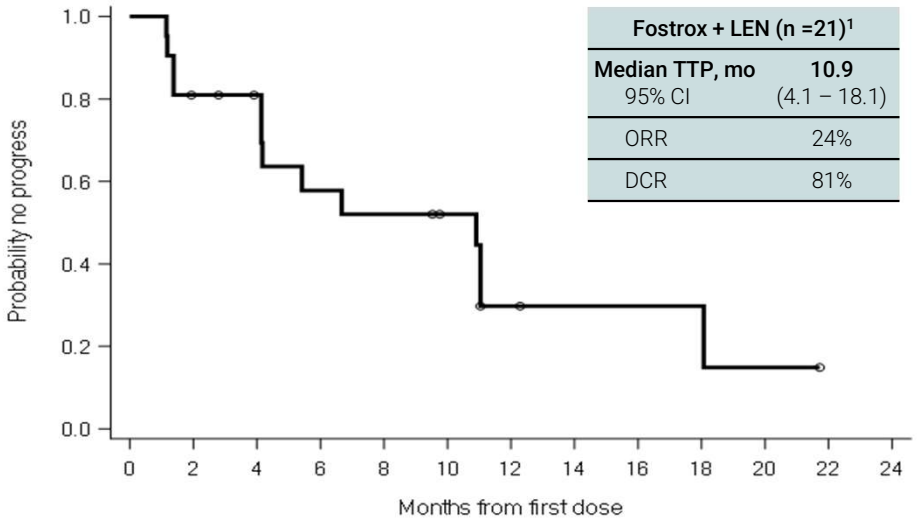
[1]Chon et al., ESMO 2024, Poster 986.

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 95% CI	**10.9** (4.1 – 18.1)
ORR	24%
DCR	81%

At risk – All pts	21	16	14	10	9	7	3	2	2	2	1	0

- 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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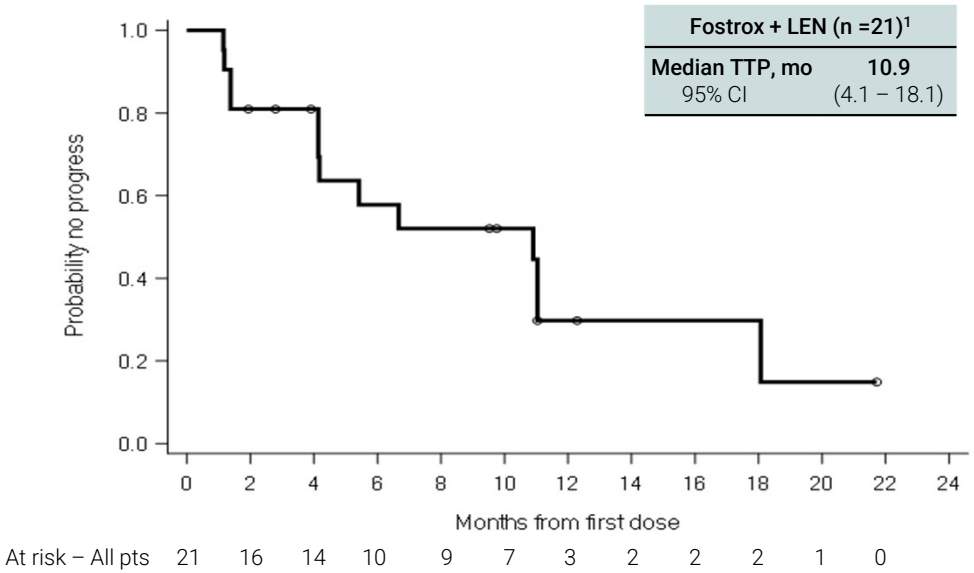
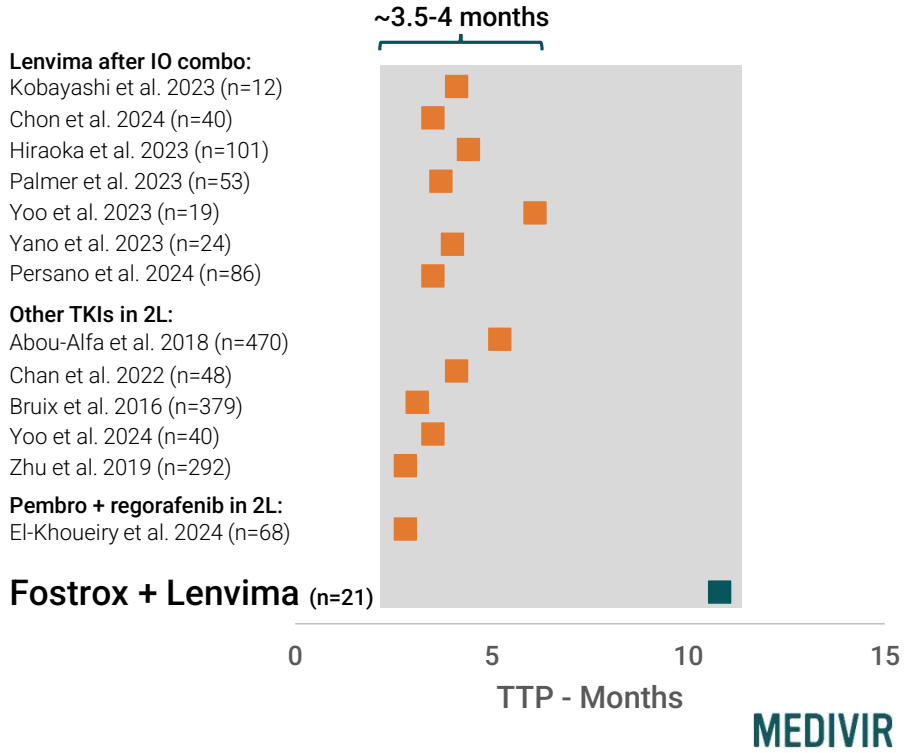
[1]Chon et al., Clinical Cancer Research 2026.

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



Median TTP/PFS vs previous studies in 2L HCC



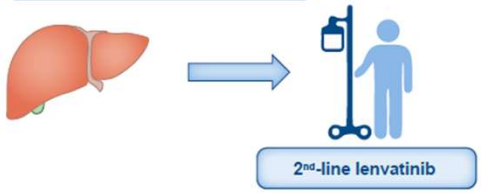
[1]Chon et al., Clinical Cancer Research 2026.

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

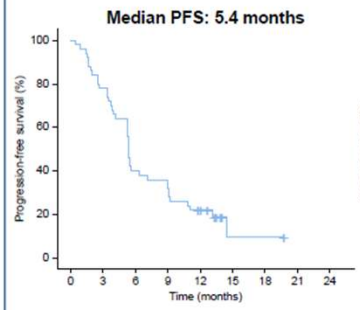
Study design

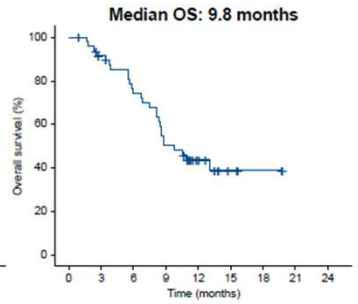
HCC progressed on 1st-line atezolizumab-bevacizumab

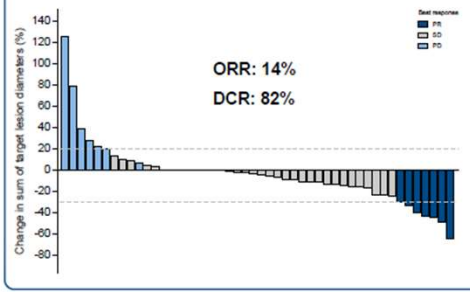


- **Investigator-initiated, multicenter, single-arm phase 2 study**
- **50 patients enrolled from 13 sites in Korea**
- **Primary end point:** PFS (>median 4.5 months)
- **Secondary endpoints:** OS, ORR, DCR, DoR, and safety.

Results







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.**

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

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., Clinical Cancer Research 2026

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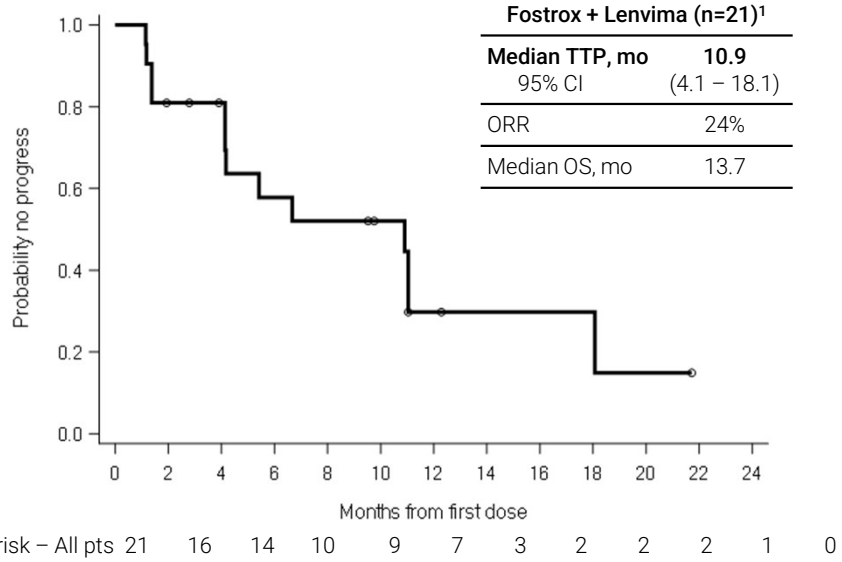
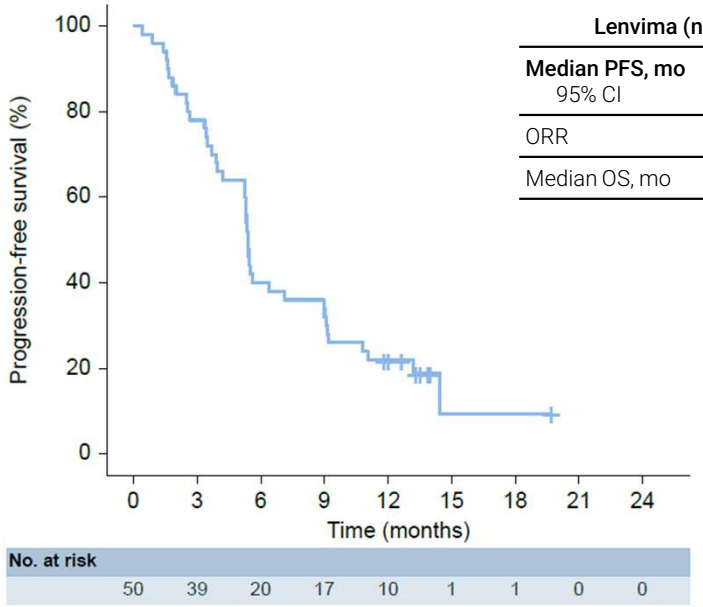
Fostrox + Lenvima phase 1b/2a data showed substantially better outcome data compared to the Lenvima montherapy study

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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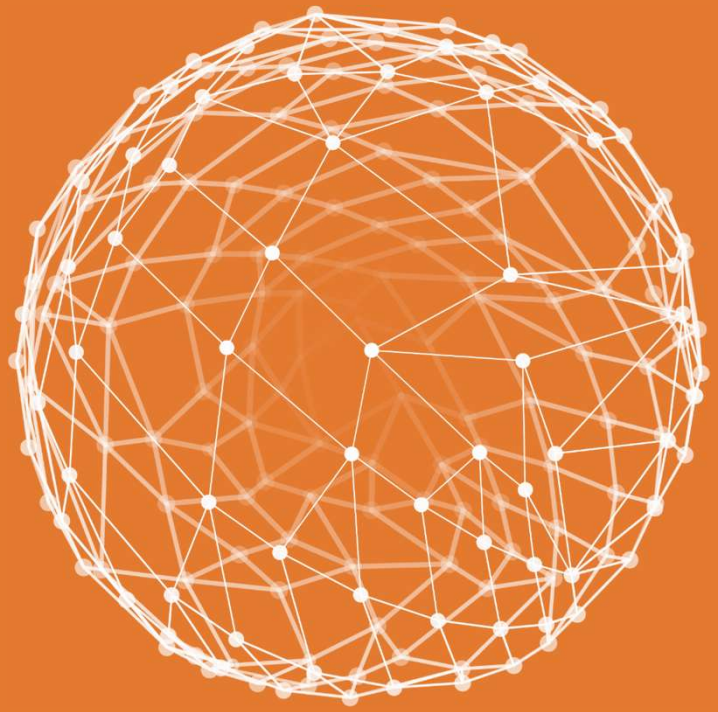
[1]Chon et al., Clinical Cancer Research 2026
[2]Kim et al., Journal of Hepatology, Sept 04 2025

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



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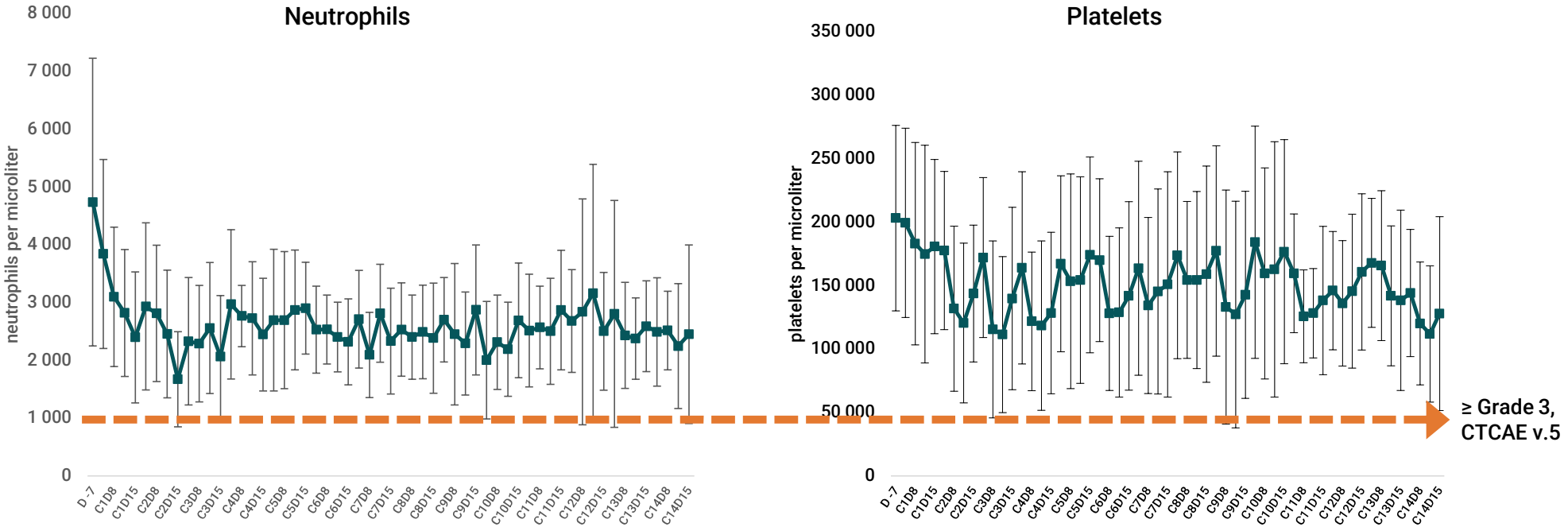
Absolute neutrophile and platelet counts were stable over the course of treatment, enabling long-term use[1]

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



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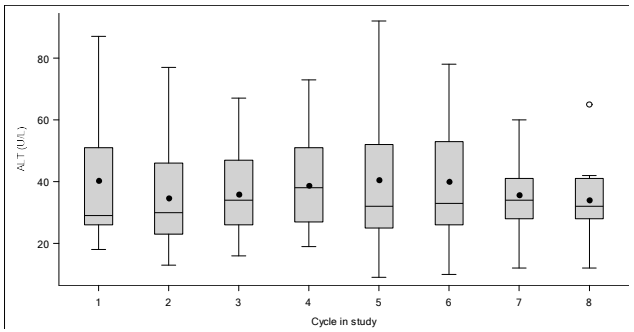
[1]Chon et al., Clinical Cancer Research 2026.

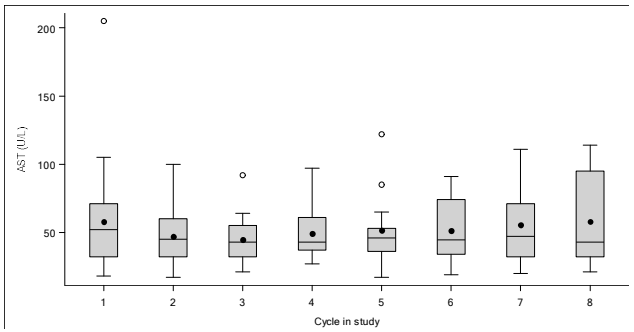
Stable liver function during treatment with fostrox + Lenvima – no deterioration in liver enzymes or change in ALBI score

ALT change over duration of treatment



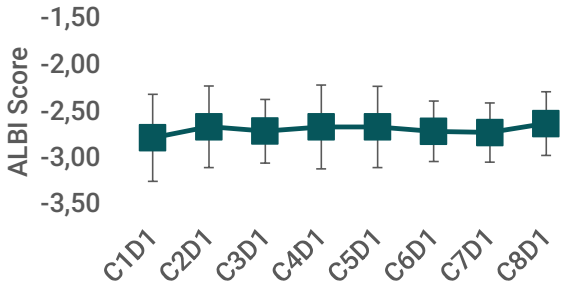
Patients At risk	21	21	16	15	14	14	11	10

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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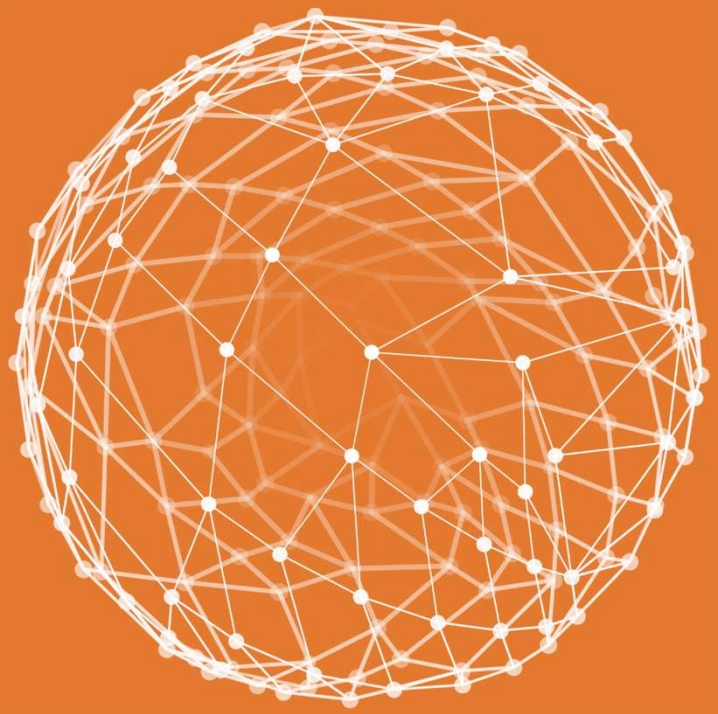
[1]Chon et al., Clinical Cancer Research 2026.

FLEX-HCC: 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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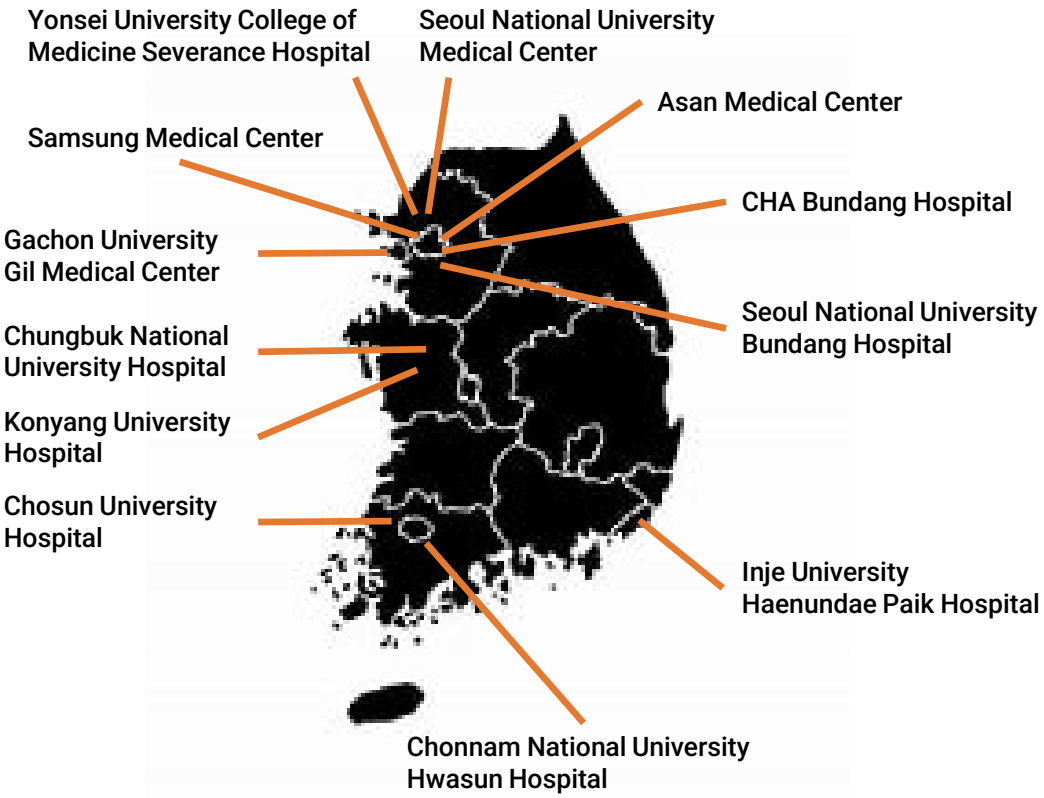
FLEX-HCC
Fostrox + Lenvatinib Combination for Advanced HCC



Primary Investigator

Dr. Hong Jae Chon

CHA Bundang Hospital

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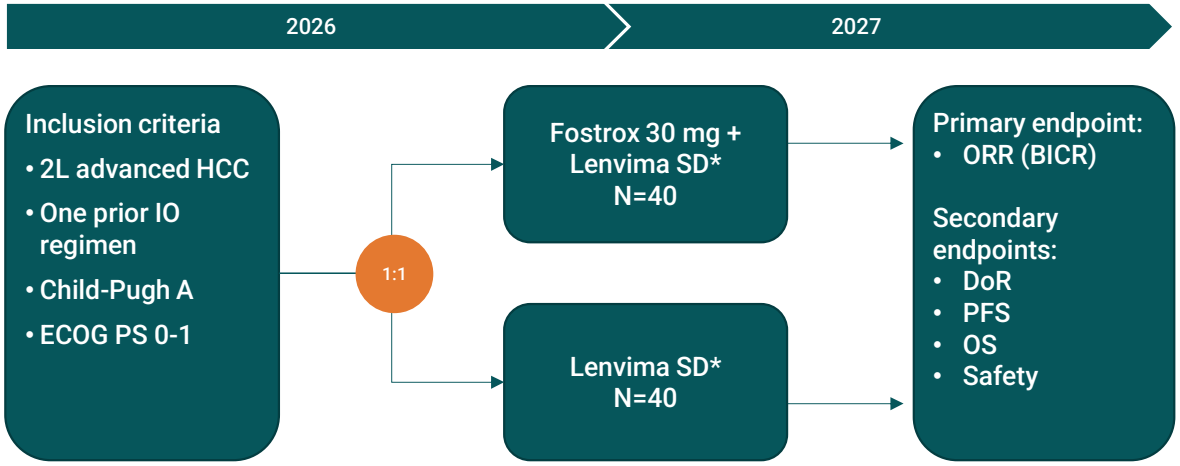
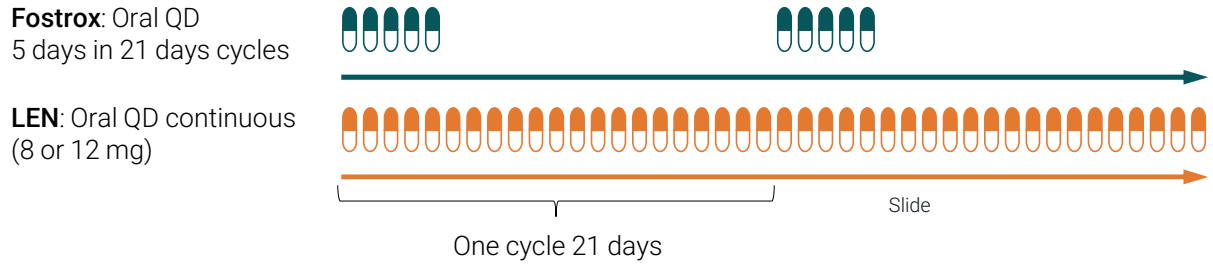
FLEX-HCC: Randomized, comparative phase 2 study to confirm benefit for fostrox + Lenvima combination in 2nd line HCC



*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

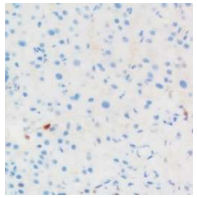
Oral, liver-activated small molecule selectively targeting tumor cells[3]

Unique, liver-targeted approach in HCC

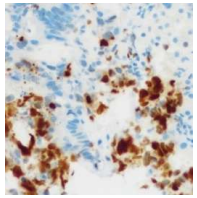
Liver-guided delivery – prodrug

Tumor-selective payload – troxacitabine

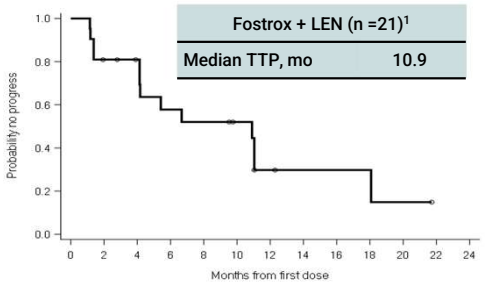
No DNA damage in healthy liver tissue



DNA damage in tumor tissue



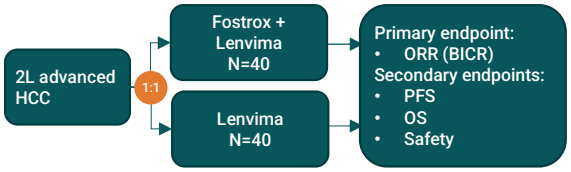
Promising efficacy in 2nd line HCC in combination with lenvatinib[1,2]



- Substantially better efficacy than seen in previous 2nd line studies including ImBrave251
- Well tolerated combination – final safety and efficacy data published in Clinical Cancer Research, June 2026

First-to-market opportunity in patient population worth >$2.5bn annually

- No 2nd line treatments approved in advanced HCC
- FLEX-HCC phase 2 study enrolling – first patient dosed in May 2026, recruitment in line to include all patients in 12 months



- **> $2.5bn** 2nd line HCC market by 2030[4]

[1]Chon et al., Clinical Cancer Research, 2026
[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]GlobalData 2021 and internal analysis

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US Notice of Allowance extends fostrox + lenvatinib patent protection to April 2041

Medivir receives Notice of Allowance for the fostrox plus lenvatinib combination patent from the US Patent and Trademark Office

JULY 20, 2026

The patent covers the combination of fostroxacitabine bralpamide (fostrox) with lenvatinib for the treatment of hepatocellular carcinoma (HCC) and cancer metastases to the liver. Once granted, the patent provides protection and market exclusivity until at least April 2041.

Covers the combination of fostrox + Lenvima for the treatment of HCC and metastases to the liver

Together with the approvals in the EU, Japan, Australia and Canada, protection is secured in key markets

Provides market exclusivity until at least April 2041

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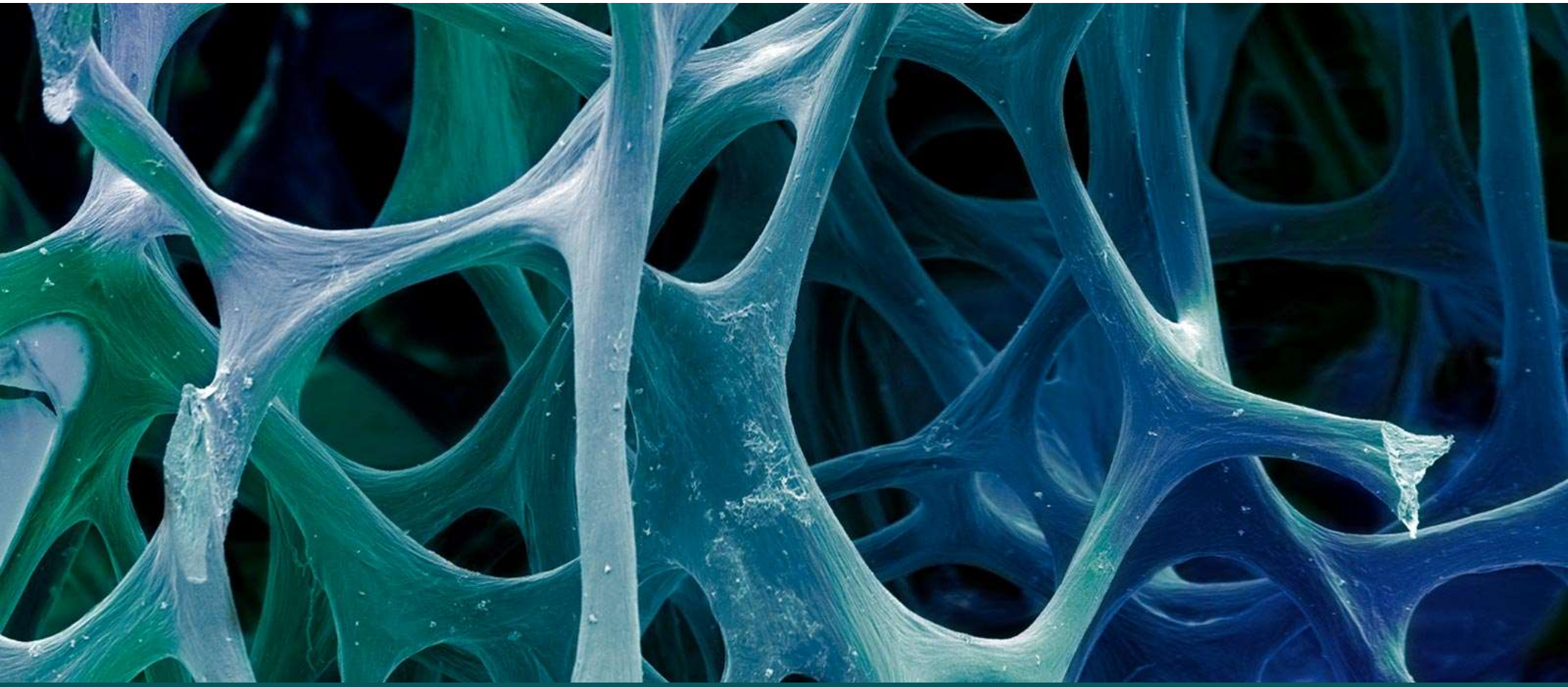

MIV-711 – Highly selective oral, cathepsin-K inhibitor for Potential treatment of Osteogenesis Imperfecta

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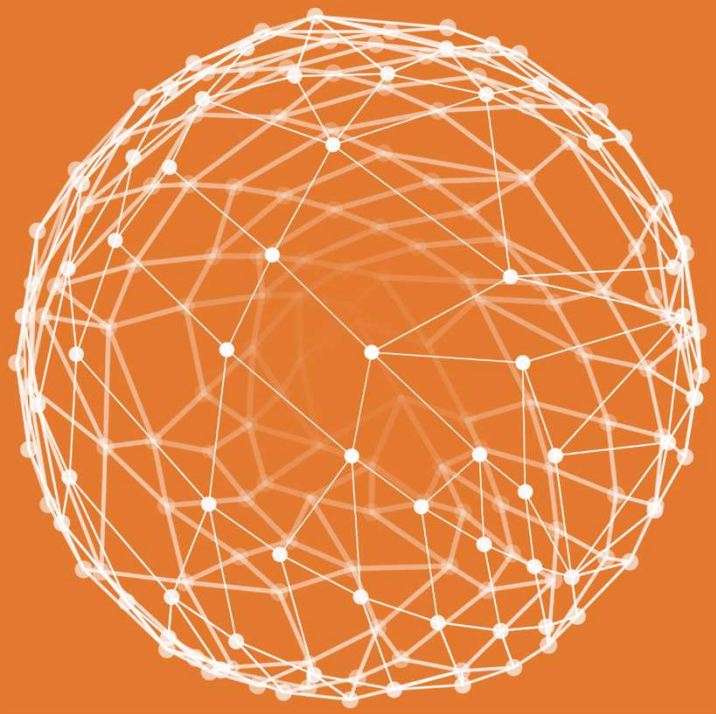
MIV-711 – Clear, scientific rationale for treatment of Osteogenesis Imperfecta



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Osteogenesis Imperfecta (brittle bone disease)

- Rare, life-threatening disease: 1 in 15,000 births
- Genetic mutations – born with the disease
- Defective or insufficient collagen production
- Fragile bones that fracture easily
  - Children suffer up to five fractures per year
  - Bone deformities and shorter stature
  - Chronic pain
  - Inability to participate in normal activities

- No approved disease-modifying drug available

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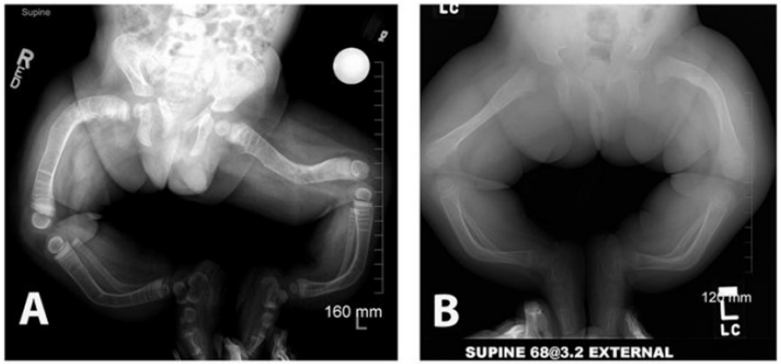
Osteogenesis Imperfecta (OI) – a rare disease from pediatric to adulthood with significant unmet medical need

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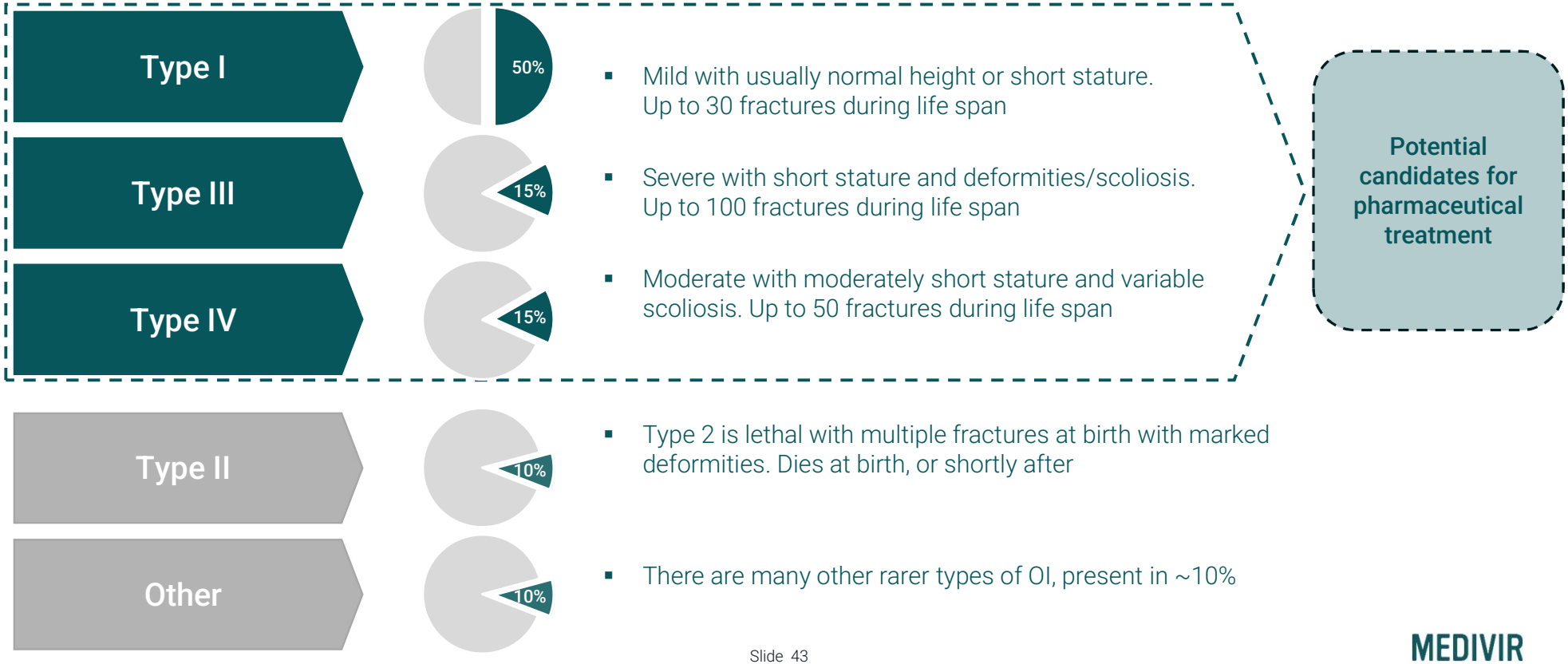
OI are divided into subtypes according to clinical severity [1,2]



[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

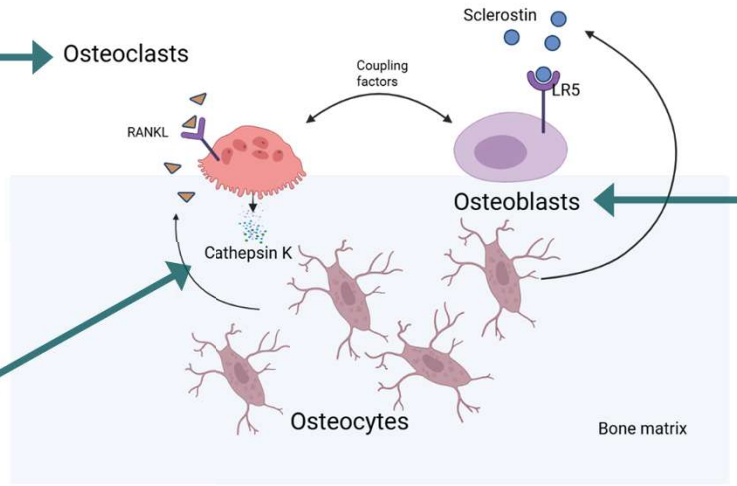
Osteoclasts

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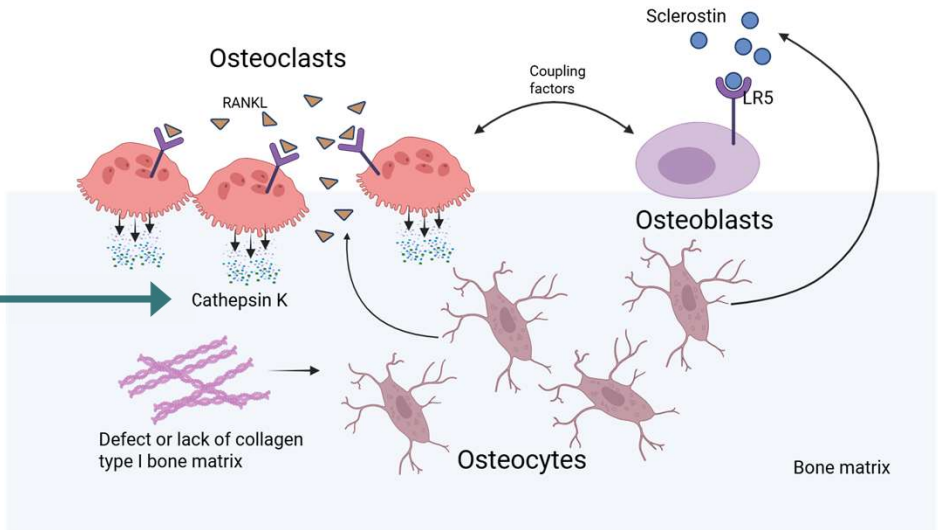
Cathepsin K activity is increased in Osteogenesis Imperfecta and drives increased bone resorption

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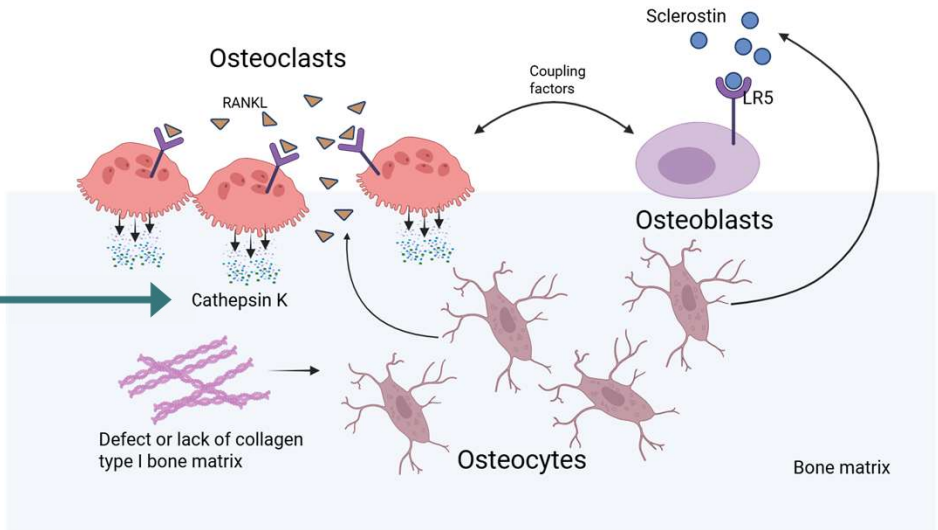
Rauch et al 2000, Baron et al 1983, Braga et al 2004; Garnero et al 2009; Rousseau et al 2010)

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



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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

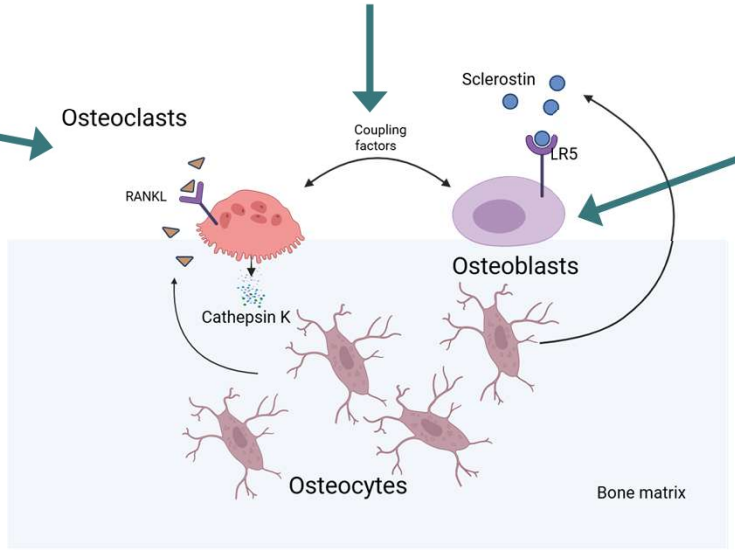
Prevents bone resorption by selective inhibition of cathepsin K

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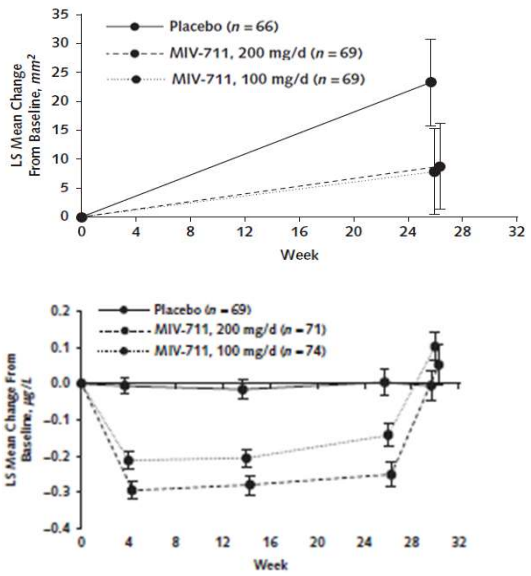
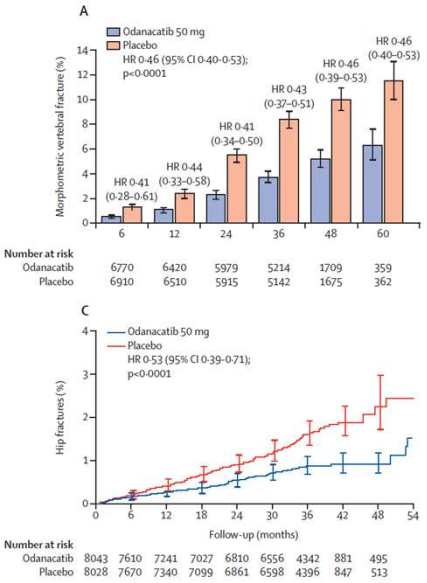
Cathepsin K inhibition showing significant benefit across multiple bone-related disorders

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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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

- Consultant Rheumatologist and Director, Centre for Metabolic Bone Disease at Royal National Orthopaedic Hospital, Stanmore, UK.
- He specialises in clinical research and development of novel therapies for adults with rare metabolic bone conditions. He is the Chair of the UK Brittle Bone Society's Scientific Advisory Board.
- He has 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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Clinical, regulatory and patient organization input on MIV-711 in Osteogenesis Imperfecta

External engagements and interactions

- **Scientific Expert Council:** study design in the context of current/ future management of OI
- **Former FDA regulatory pediatric clinical expert:** advice on Rare Pediatric Disease Designation (RPDD) filing and next clinical development step in adult and pediatric OI
- **Chairman of Osteogenesis Imperfecta Federation Europe (OIFE**): patient experience and general unmet need in OI
- **Annual meeting Swedish organization for OI (SFOI):** patient input on MIV-711 POC study design in OI and patient insights overall

Take aways to shape PoC study design

- Strong engagement from external experts with scientific support for MIV-711 mechanism in OI patients, including high interest in POC study
- Existing clinical/non-clinical package & significant unmet medical need supports development in pediatric population
- Potential for Rare Pediatric Disease Designation & Priority Voucher confirmed
- Patient organizations emphasize significant medical need across all subtypes, including mild Type 1

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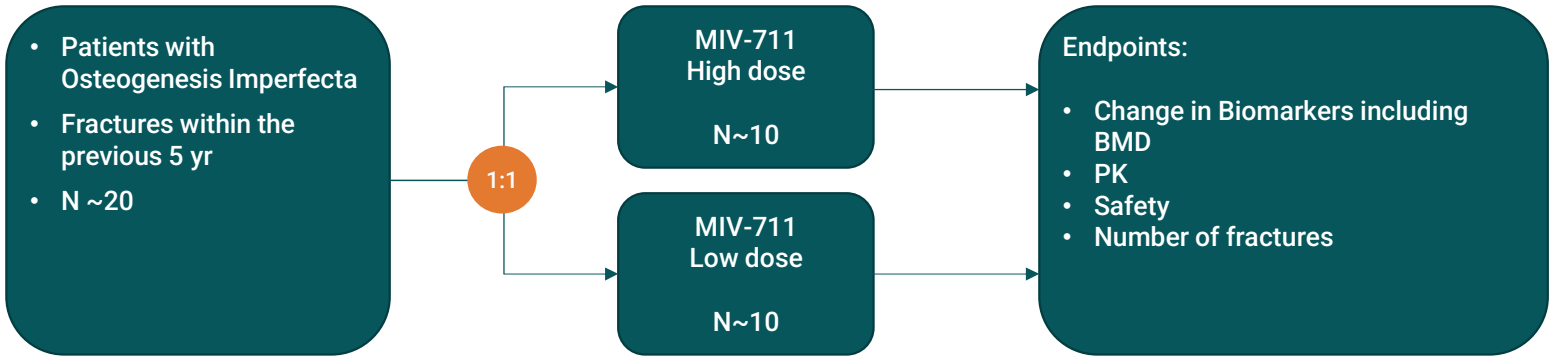
Preparation for the upstart of the randomized phase 2 PoC study with MIV-711 in OI well underway



Phase 2 POC study in Osteogenesis Imperfecta

- Treatment for 12 months randomized in 2 dose arms
- Substantial interest from investigator in participating in the study and 5 sites have already been approved
- Fast enrollment expected with patients already in the medical records at the hospital
- Manufacturing of MIV-711 drug according to schedule
- Great commitment and interest from regional/global and local patient organizations

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Osteogenesis Imperfecta - Economics

- Rare but life-threatening disease: 1 in 15,000 births
- Primary market: 58,000 patients in Europe and USA
- Orphan Drug Disease – Premium pricing
- Rare Pediatric Disease Voucher at market approval* – value ~USD200m
- Market worth at least USD2.5bn - Blockbuster sales potential

* Source: Althobaiti et al., "Disentangling the Cost of Orphan Drugs Marketed in the United States," Healthcare 2023, 11, 558, https://pmc.ncbi.nlm.nih.gov/articles/PMC9957503/

** The PRV programme is subject to periodic reauthorisation by the US Congress and its continuation cannot be guaranteed. The valuation assumes the programme remains in place at market approval and is based on recent PRV transaction values.

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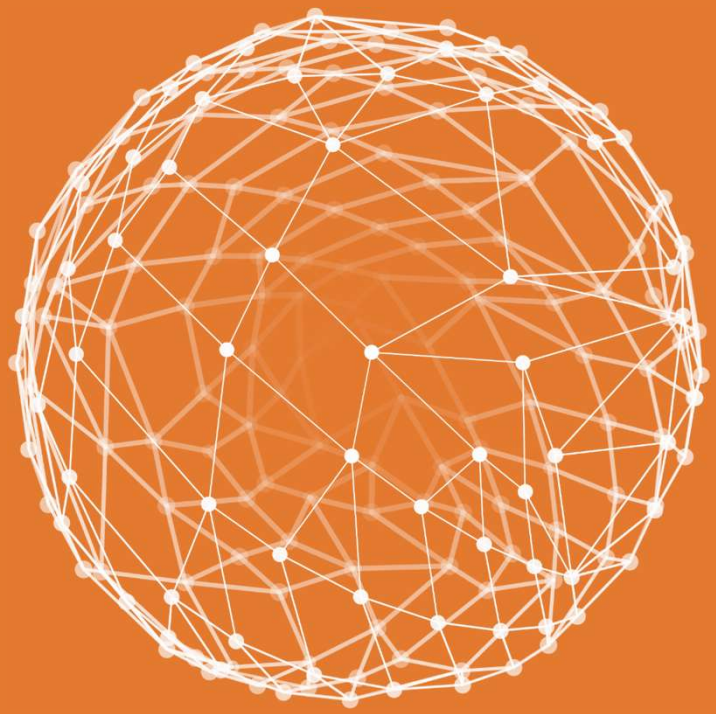
MIV-711 in Perthes Disease — Addressing a significant unmet medical need in children



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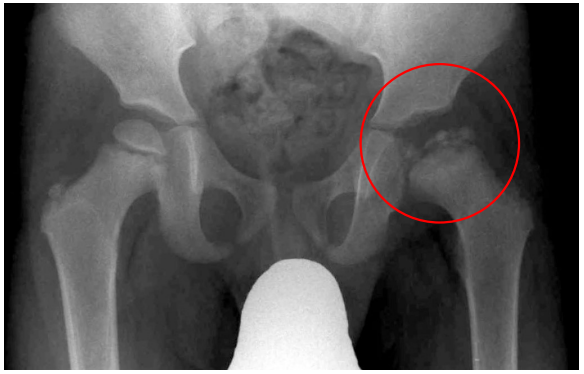
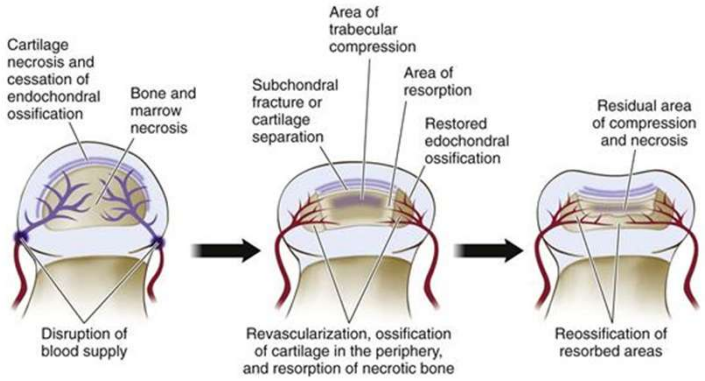
Legg-Calvé-Perthes disease – a rare, life-altering disease in children with no approved drug treatment

- Rare, life-altering disease in children: 1 in 1,500
- Most commonly affects boys aged 4–8 years
- Sudden disruption of blood supply to the femoral head
- Bone necrosis, collapse and deformity of the hip joint
- Bracing and surgery address the acute symptoms only
- Permanent hip damage in the majority of cases
- Development of debilitating osteoarthritis in adulthood and around 20% require total hip replacement





- No approved disease-modifying drug available

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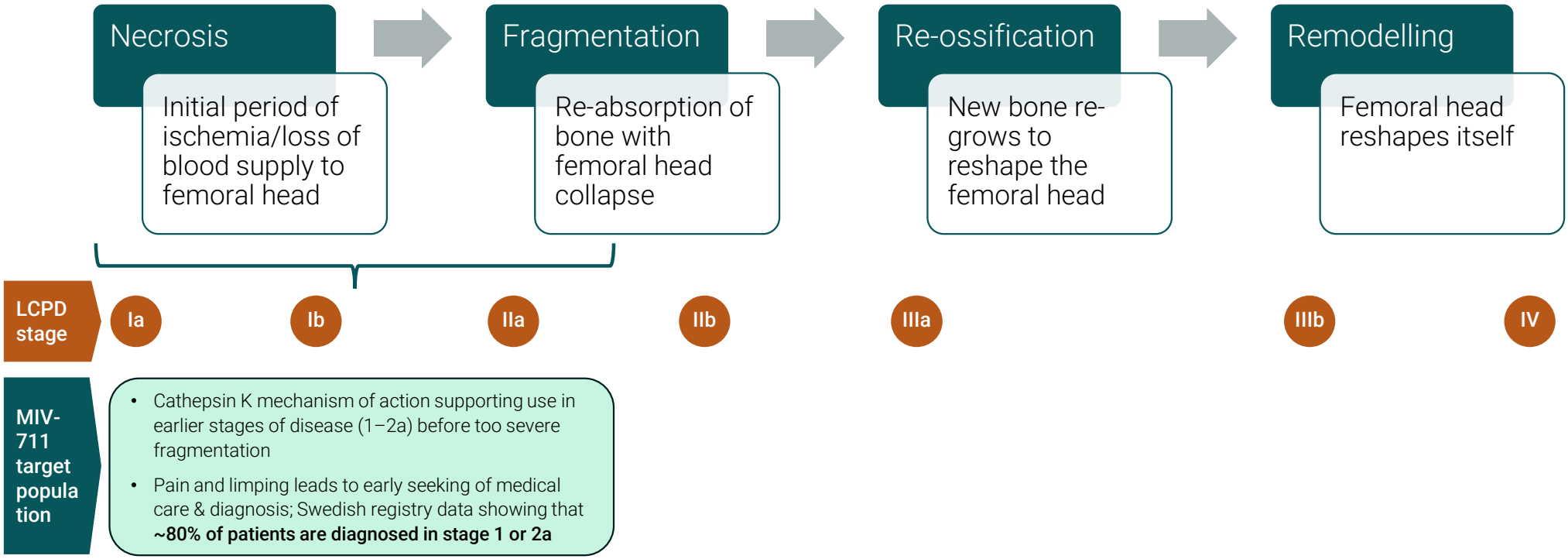
MIV-711 would target 60-80% of the patient population at diagnosis



[1]Kim et al., Bone Joint J. 2022
[2]Larson et al., J Bone Joint Surg Am. 2012

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MIV-711 in Perthes disease – a second rare bone indication built on the same established profile

Rationale for MIV-711 in Perthes disease

- **MIV-711 counteracts deformity of the femoral head in a disease-specific animal model**
- **Same mechanism and established safety profile as in Osteoarthritis – clinical exposure in ~250 subjects in phase 1/2**
- **Orphan Drug Designation and Rare Pediatric Disease Designation for Perthes Disease granted by the FDA**
- **Synergy in pediatric formulation development and dose selection between LCPD and clinical development in OI**

Development plan and status

- Funding secured for phase 2 PoC study, financed by oversubscribed directed share issue in June
- Next steps: pediatric formulation followed by a randomized proof-of-concept study
- Experienced Clinical Operations Lead recruited to drive execution in both Perthes disease and Osteogenesis Imperfecta
- Annual peak sales estimated at ~$1bn five years after marketing approval in the US and Europe

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Perthes disease – significant commercial opportunity with orphan drug premium pricing

- Primary market: 8,800 children annually in Europe and USA
- Patients accessible for treatment: ~80%
- Orphan Drug Disease – Premium pricing
- Rare Pediatric Disease Voucher potential** – ~USD200m
- Estimated annual peak sales: ~USD1bn five years after marketing approval
- Orphan Drug Designation & Rare Pediatric Disease Designation already granted

* Source: Althobaiti et al., "Disentangling the Cost of Orphan Drugs Marketed in the United States," Healthcare 2023, 11, 558, https://pmc.ncbi.nlm.nih.gov/articles/PMC9957503/

** The PRV programme is subject to periodic reauthorisation by the US Congress and its continuation cannot be guaranteed. The valuation assumes the programme remains in place at market approval and is based on recent PRV transaction values.

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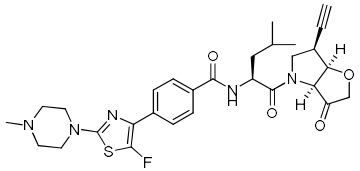
MIV-711 – highly selective cathepsin K inhibitor now in two rare bone indications

3rd generation, highly selective cathepsin K inhibitor



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

- Proven clinical radiographic benefit in ~250 subjects in phase 1/2 Osteoarthritis[1]
- MOA enabling effective inhibition of bone and cartilage degradation

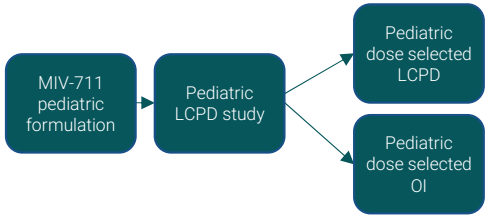
Proven ability to prevent bone degradation & improve bone quality[1,2]

PoC established in animal model demonstrating preventing bone loss and femoral deformity[2]

- MIV-711 has significant clinical exposure and safety data & has shown proven benefit across multiple bone-related diseases

Two first-to-market opportunities in rare bone disease

- No approved treatment options in either indication
- ODD in both indications; RPDD granted in Perthes disease
- Synergy between OI and LCPD program in development of pediatric formulation
- OI >$2.5bn and Perthes ~$1bn in annual market potential



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

PARTNERED PROGRAMS

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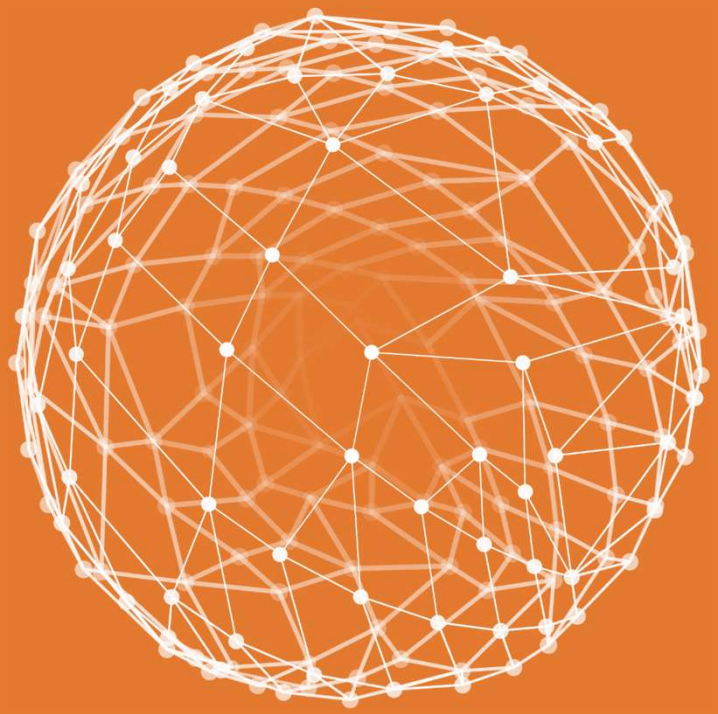
VBX-1000 (MIV-701) – The first, potential disease-modifying treatment of periodontal disease in dogs



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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.

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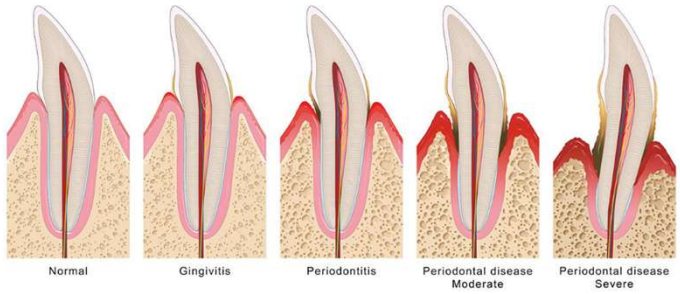
Periodontal disease in dogs (PD)

- 160 million dogs in Europe and USA; >80% of dogs by age 3 has PD
- Periodontal disease is the number one disease in dogs
  - UK study (n=22 300): PD 12.5%, ear infections 7.3%, obesity 7.1%
- Standard of care: Dental cleaning/surgery/tooth extraction
- No drug treatment approved to stop bone resorption
  - Care focuses on control of plaque / inflammation only



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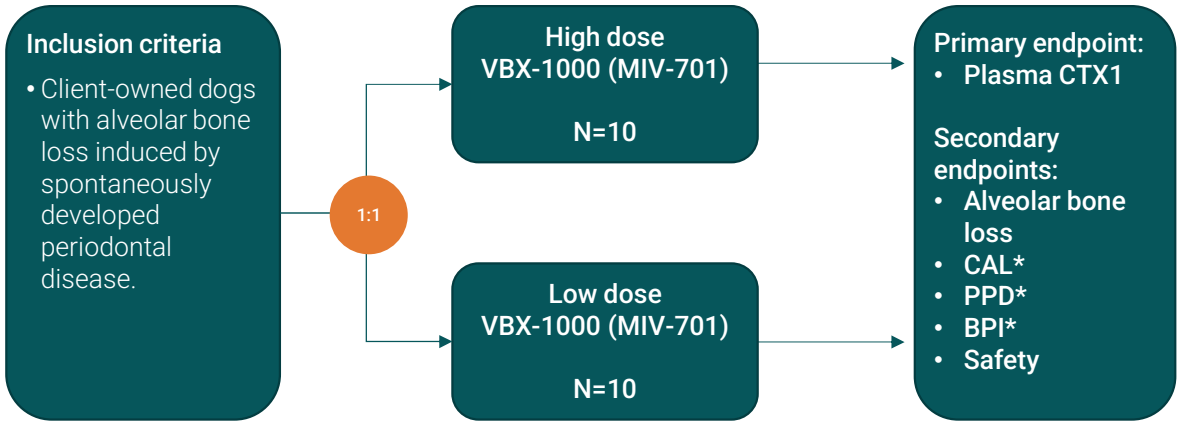
Landmark POC Study in periodontal disease (PD) in dogs showing significant & clinically relevant improvements



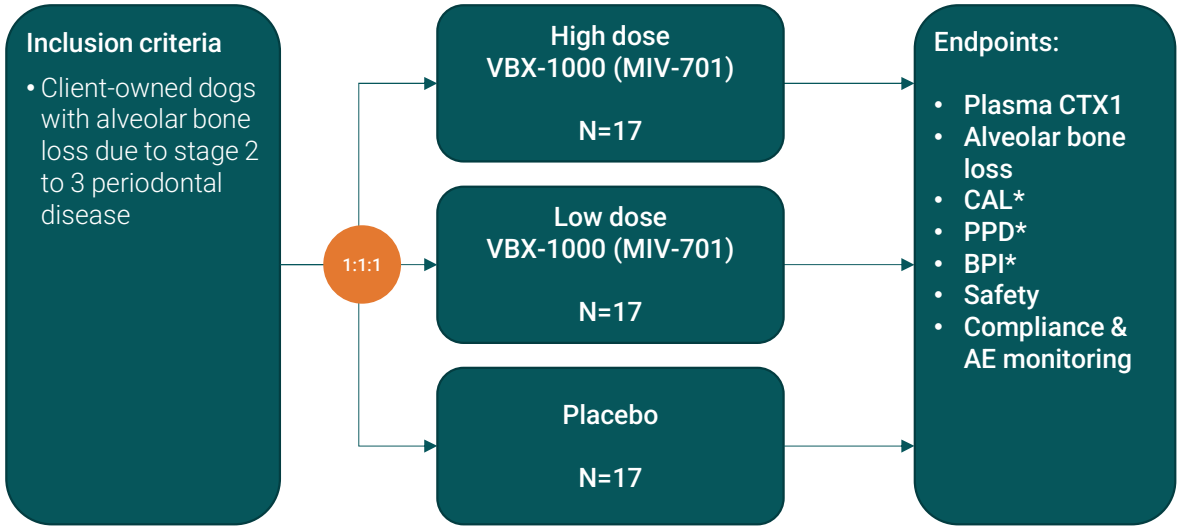
Efficacy assessment on primary and secondary end-point measurements = Day 90 vs baseline

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

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

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:

- 29 dogs dosed to date (June 30)
- 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

Periodontal disease in dogs - Economics

- Vetbiolix as collaboration partner assumes all development costs
- Income split/Royalty agreement
- Drug pricing: Low due to private pay (not covered by insurance)
- Peak royalty potential: around SEK700m

- No approved disease-modifying drug available

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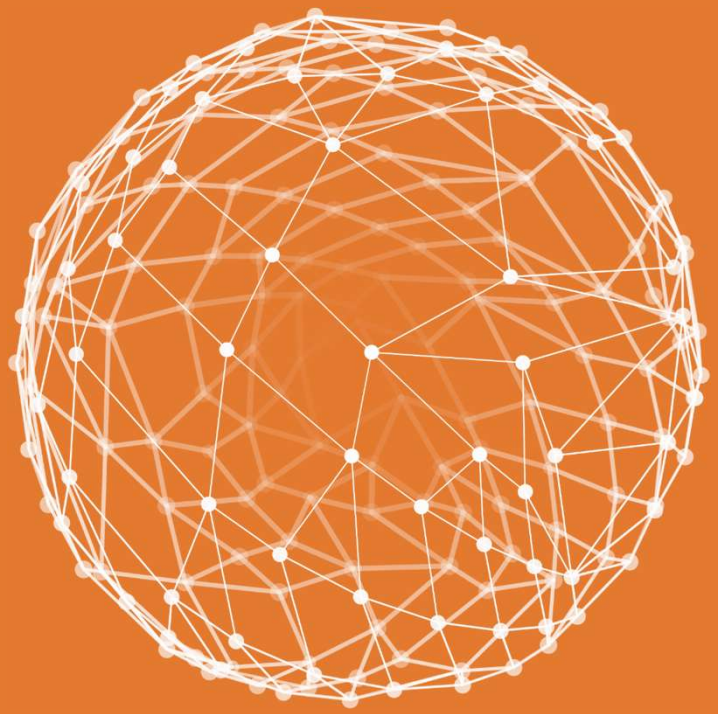
Remetinostat – Phase 3 ready topical HDAC-inhibitor for non-melanoma skin cancer



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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)

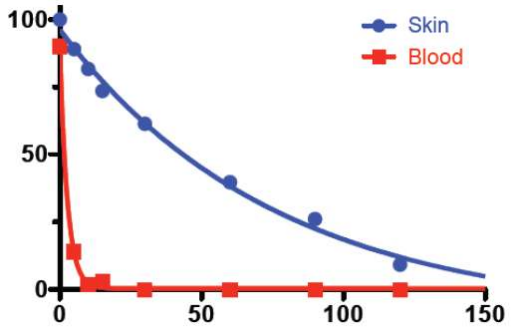
- Open label Phase II study (25 patients, Stanford ISS) results showed 70% ORR

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)

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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.

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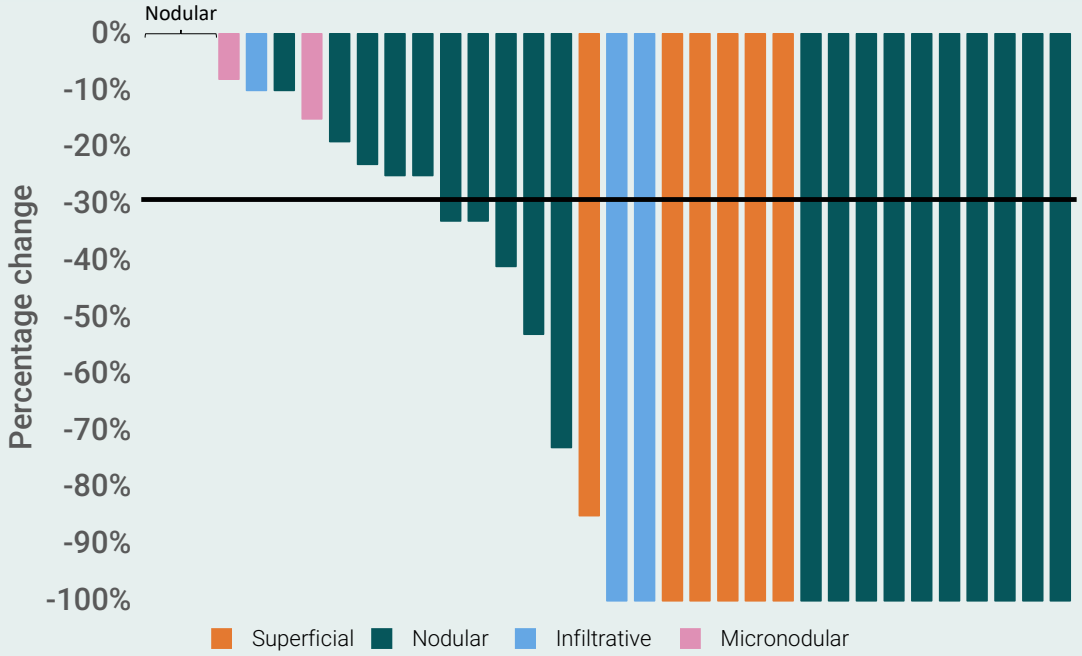
Remetinostat is an effective topical treatment for reducing BCC disease burden in a clinically significant manner

Encouraging efficacy across all subtypes of BCC lesions



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

Overall response rate

Complete pathological resolution

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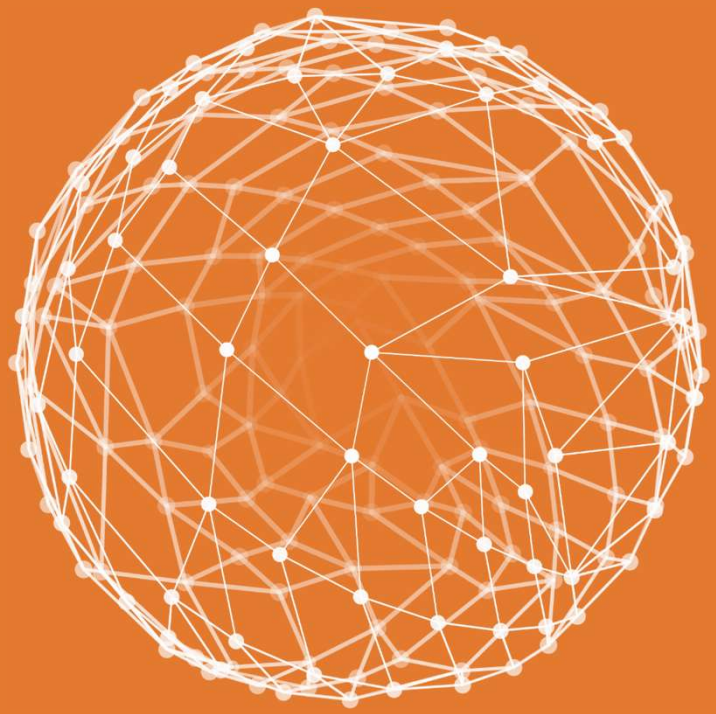
[1]J. Kilgour et al, Clinical Cancer Research published online 6 August 2021

MET-X – AMR resistance bypass drug candidate



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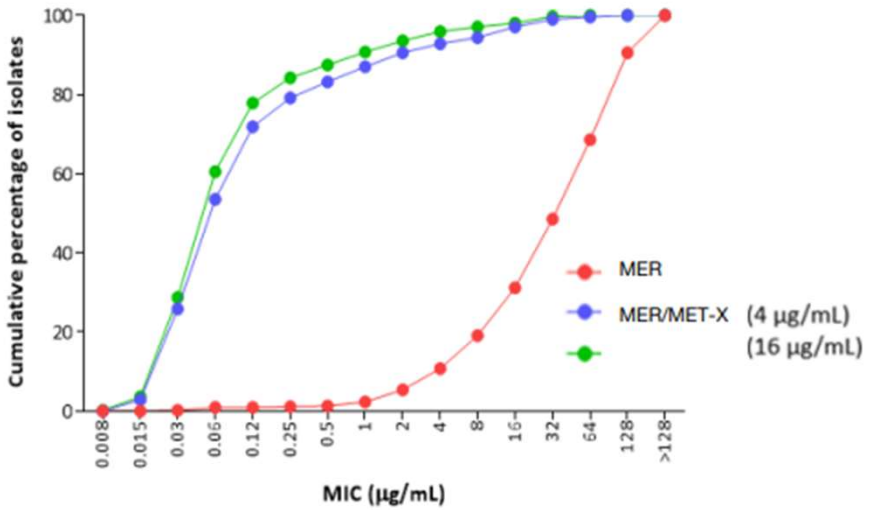
MET-X (MBLI) – FDA QIDP Designation received & sub-licensing agreement signed to enter clinical development

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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Strengthened financial position

Oversubscribed SEK 140 million directed share issue funds all three phase 2 studies;

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Oversubscribed SEK 140 million directed share issue secures funding for all three phase 2 studies

The directed share issue

- **Approximately SEK 140 million gross (SEK 134 million net), resolved by the Board in June**
- **Oversubscribed, bringing in the new institutional investors Cicero Fonder and Storebrand**
- **Continued strong support from Carl Bennet, Hallberg Management, LINC and Nordea Småbolagsfond Norden**
- **Tranche 2 approved at the Extraordinary General Meeting on July 20, completing the issue**

A strengthened financial position

- Cash and cash equivalents of SEK 253.5 million at the end of the quarter, versus SEK 38.2 million a year earlier
- Funding in place for all three phase 2 studies – FLEX-HCC, MIV-711 in OI and MIV-711 in Perthes disease
- Existing cash assessed as sufficient at least into 2030
- Excludes any proceeds from out-licensing of fostrox or MIV-711 and from the agreements with Vetbiolix and Biossil

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Three phase 2 studies – all fully funded after the directed share issue

FLEX-HCC – fostrox + Lenvima in 2nd line liver cancer

- Randomized phase 2, 80 patients, 12 hospitals in South Korea
- First patient dosed May 2026; full enrollment within 12 months
- Topline results expected H2 2027
- No approved 2nd line options – market >$2.5bn by 2030

MIV-711 in Osteogenesis Imperfecta

- Randomized phase 2 PoC, ~20 adult patients, two dose arms
- Study design finalized; first five sites identified, strong external interest
- Approximately 500,000 patients globally with no approved treatment
- ODD granted by FDA – market opportunity >$2.5bn annually

MIV-711 in Perthes disease

- Funding secured for phase 2 PoC study
- Pediatric formulation development initiated, ahead of PoC study
- ODD and Rare Pediatric Disease Designation granted by the FDA
- Peak sales potential ~USD 1bn annually

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Financial summary Q2, 2026

Consolidated Income Statement, summary	Q2		Q1 - Q2		Full year
(SEK m)	2026	2025	2026	2025	2025
Net turnover	1,2	1,5	2,3	2,1	8,5
Other operating income	0,0	0,3	0,2	0,5	0,4
Total income	**1,2**	**1,8**	**2,5**	**2,6**	**8,9**
Other external expenses	-17,9	-17,0	-22,4	-23,2	-41,4
Personnel costs	-4,3	-7,1	-9,8	-14,1	-27,1
Depreciations and write-downs	0,0	-0,7	-0,6	-1,4	-32,5
Other operating expenses	-0,2	-0,2	-0,2	-0,5	-0,5
Operating profit/loss	**-21,2**	**-23,2**	**-30,6**	**-36,5**	**-92,6**
Net financial items	1,1	-0,2	1,1	-0,1	-1,8
Profit/loss after financial items	**-20,1**	**-23,3**	**-29,5**	**-36,6**	**-94,4**
Tax	-	-	-	-	-
Net profit/loss for the period	**-20,1**	**-23,3**	**-29,5**	**-36,6**	**-94,4**

- Net turnover for Q2 was SEK 1.2 million
- Operating loss for Q2 was SEK –21.2 million
  - Cash flow from operating activities for Q2 was SEK -18.3 million
  - Directed share issue in Q2 SEK 140 million
  - Cash balance end of Q2 was SEK 253.5 million
  - Cash assessed as sufficient into 2030

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Value drivers in 2026/2027 – three funded programs and a partnered readout

Value drivers in our own programs

- **FLEX-HCC: continued patient recruitment towards full enrollment within 12 months**
- **MIV-711 in Osteogenesis Imperfecta: site activation and start-up of the phase 2 PoC study**
- **MIV-711 in Perthes disease: pediatric formulation and PoC study preparations**
- **MIV-701: Vetbiolix study in canine periodontitis could generate royalty revenues without material development costs for Medivir**

Upcoming milestones

- Q4 2026: topline results from the Vetbiolix phase 2 study with VBX-1000 (MIV-701) – 29 dogs enrolled at the end of the quarter (June 30)
- Rare Pediatric Disease Designation, completion of OI study feasibility and regulatory approval of study design – preparations progressing well
- 2027: topline results from FLEX-HCC
- Continued strengthening of IP across territories, US combination patent extending protection to 2041

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Thank You!