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Improving life for cancer patients
through transformative drugs

August 27, 2018

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Today’s presenters

CHRISTINE LIND
President and CEO

- Joined Medivir 2015, CEO since 2017
- Extensive experience from strategic advisory and business development for biotech and pharma

RICHARD BETHELL
Chief Scientific Officer

- Joined Medivir 2013
- 28 years drug discovery and development in oncology and infectious disease

CHRISTINA HERDER
EVP, Strategic Business Development

- Joined Medivir 2017
- Experience in Corporate Development, Project & Portfolio Management, and as a biotech CEO

ERIK BJÖRK
Chief Financial Officer

- Joined Medivir 2018
- Global finance leadership roles in consumer and life sciences

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Agenda

1. Medivir in brief
2. Birinapant – science and commercial opportunity
3. MIV-818 – science and commercial opportunity
4. Outlook and financial summary
5. Q&A session

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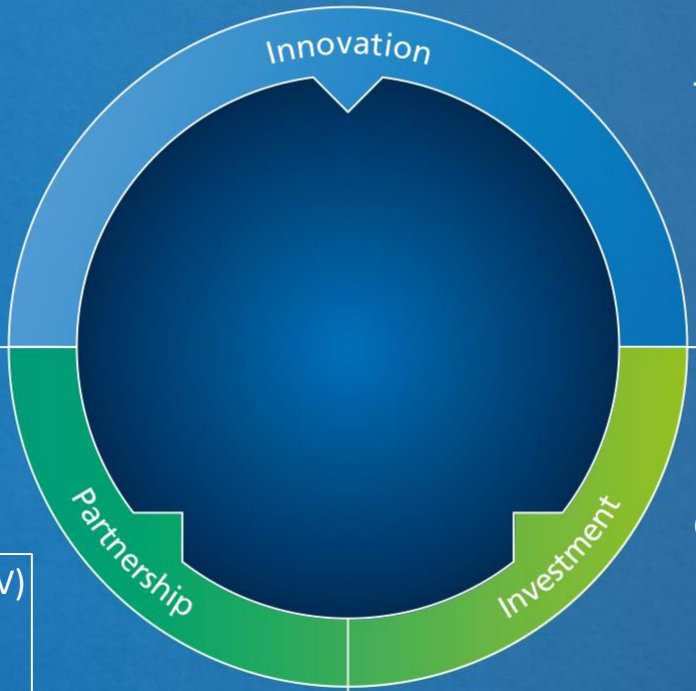
Medivir in Brief

Who is Medivir?

- Using **world-class scientific expertise** to bring new therapies to cancer patients
- **Clinical pipeline** composed of projects with **multi-billion dollar sales** potential as well as **orphan cancer drug candidates**
- Strong commercial focus – delivered more than **20 global partnerships** and **2 products from idea to market**
- Competences **from discovery through regulatory approvals**

Strategy



Discover

Leveraging scientific expertise to build pipeline in oncology

- Protease inhibitors
- Nucleoside prodrugs

Partner

Collaborations enhance the value of programs

- Zoviduo®/ Xerclear
- MIV-802 (HCV)

Develop

Drug development in areas of high unmet patient need

1) Total received revenues from partnerships last 10 years.

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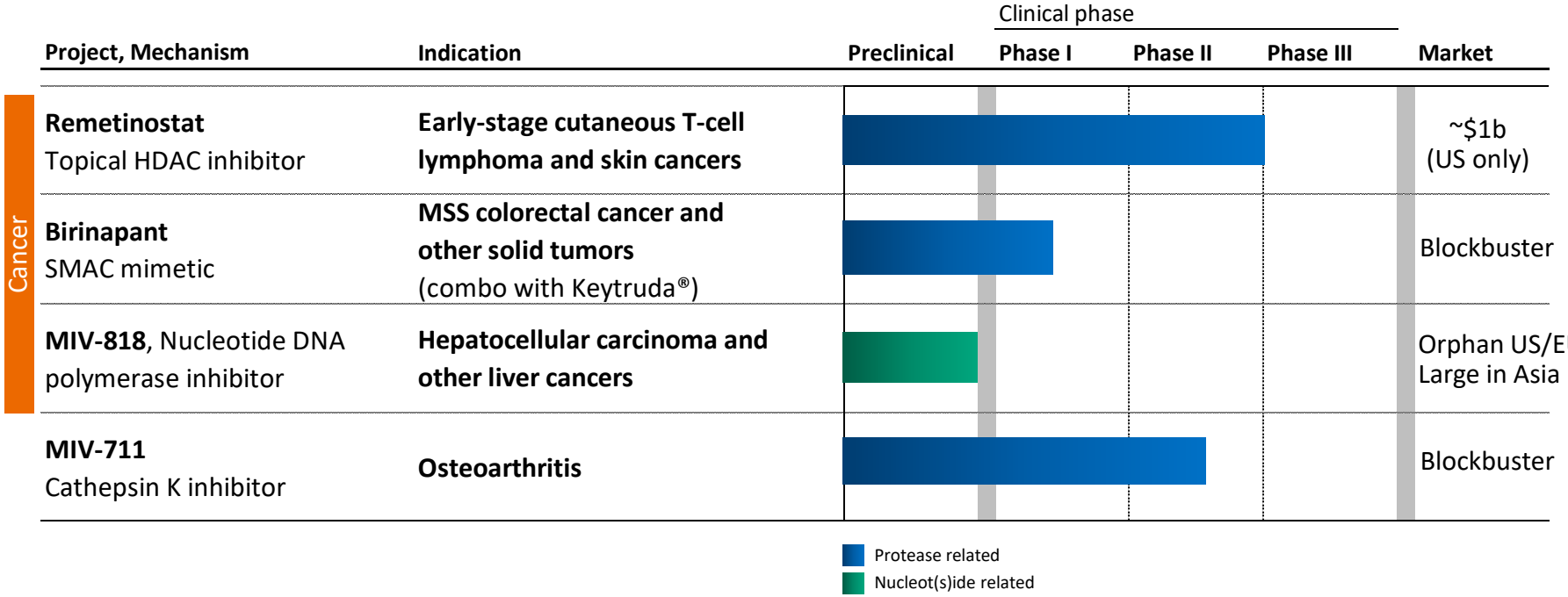
Oncology drug development in areas of high unmet need

Strong and balanced development pipeline based around areas of scientific expertise and focused on cancer



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Key milestones throughout the year

Track record of delivery

JAN – JUL 2018

☑ **Completed** MIV-818 (liver cancer) IND-enabling preclinical studies

MIV-818
EASL | HCC SUMMIT
Geneva, Switzerland
March 2018

MIV-818
AACR American Association for Cancer Research
Chicago, USA
April 2018

MIV-711
2018 WORLD CONGRESS OSTEOARTHRITIS
APRIL 26–28, 2018 LIVERPOOL, UNITED KINGDOM
OARSI

Remetinostat
IID 2018 International Investigative Dermatology
Orlando, USA
May 2018

Birinapant
ASCO American Society of Clinical Oncology
Chicago, USA
June 2018

☑ **Completed** MIV-711 Phase IIa osteoarthritis extension study

Coming events

AUG 2018 →

- ☐ Start of MIV-818 (HCC nuc) Phase I study (2H 2018)
- ☐ Completion of the dose escalation portion of the birinapant Phase I/II study in combination with Keytruda® (2H 2018)
- ☐ Start of Phase III CTCL study remetinostat (2019)

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Birinapant for solid tumors

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Despite immuno-oncology breakthroughs patients have unmet needs

Multi-billion dollar market, and growing for immuno-oncology agents

Revenues of PD-1/PD-L1 inhibitors [1)]

$10bn+

< 1/2

of patients derive meaningful clinical benefit in approved indications

0-5%

ORR in other indications such as MSS colorectal cancer

Combination regimens to enhance benefit in underserved patients

1) Merck (Keytruda), Bristol-Myers Squibb (Opdivo), AstraZeneca (Imfinzi) and Roche (Tecentriq) financial reports, twelve months ended March 31, 2018

Linking targeted therapy with immuno-oncology

Uniquely potent molecule against a novel target

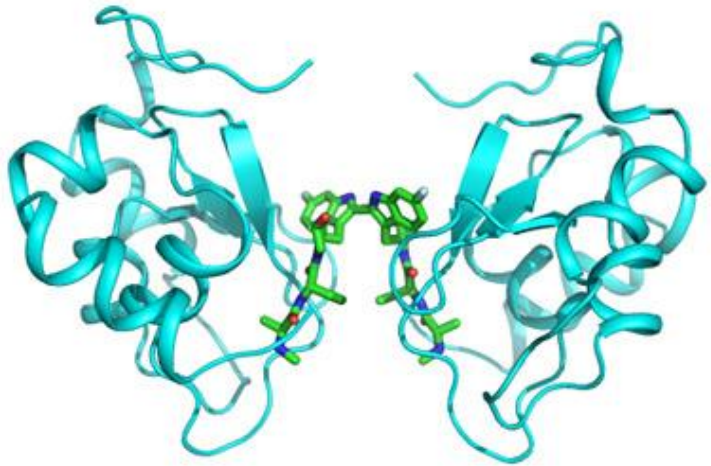
- Bivalent SMAC (second mitochondrial-derived activator of caspases) mimetic
- Upon binding to cIAPs, birinapant triggers their autoubiquitination, leading cIAP degradation by the ubiquitin proteasome system
- Reduction in cIAPs leads to effects on immune cells and tumor cells

Strong rationale for use in combinations

- Efficacy observed in both hematological and solid tumor models
- Synergistic with anti-PD1 and likely other IO pathways
- Phase I/II study in combination with Merck's Keytruda® underway

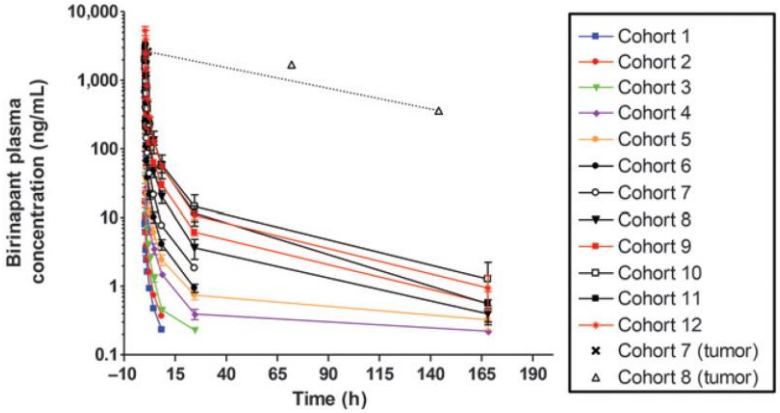
Blockbuster potential and strong patent position

- Expected patent life to ~2034, including extensions

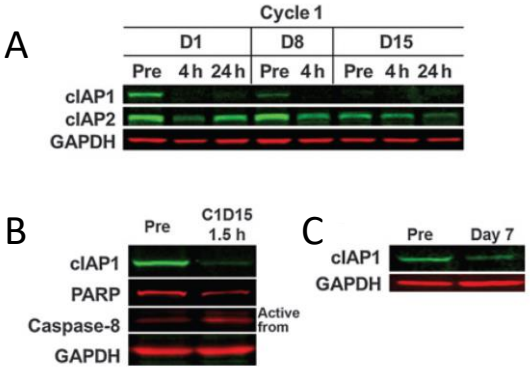


Birinapant: Clinical Pharmacodynamic Effects and PK

Birinapant Plasma PK



Birinapant Clinical Pharmacodynamics



- Birinapant has been dosed to >400 cancer patients, generally well-tolerated up to doses of 22mg/m^2
- Well characterized clinical pharmacokinetics, with elevated levels in tumours compared to plasma
- Birinapant treatment resulted in a greater than 75% reduction in cIAP1 in PBMC samples at doses of 5.6 mg/m^2 and above

Avaramadi et el. Molecular Cancer Therapeutics (2015), 14(11); 2569–75

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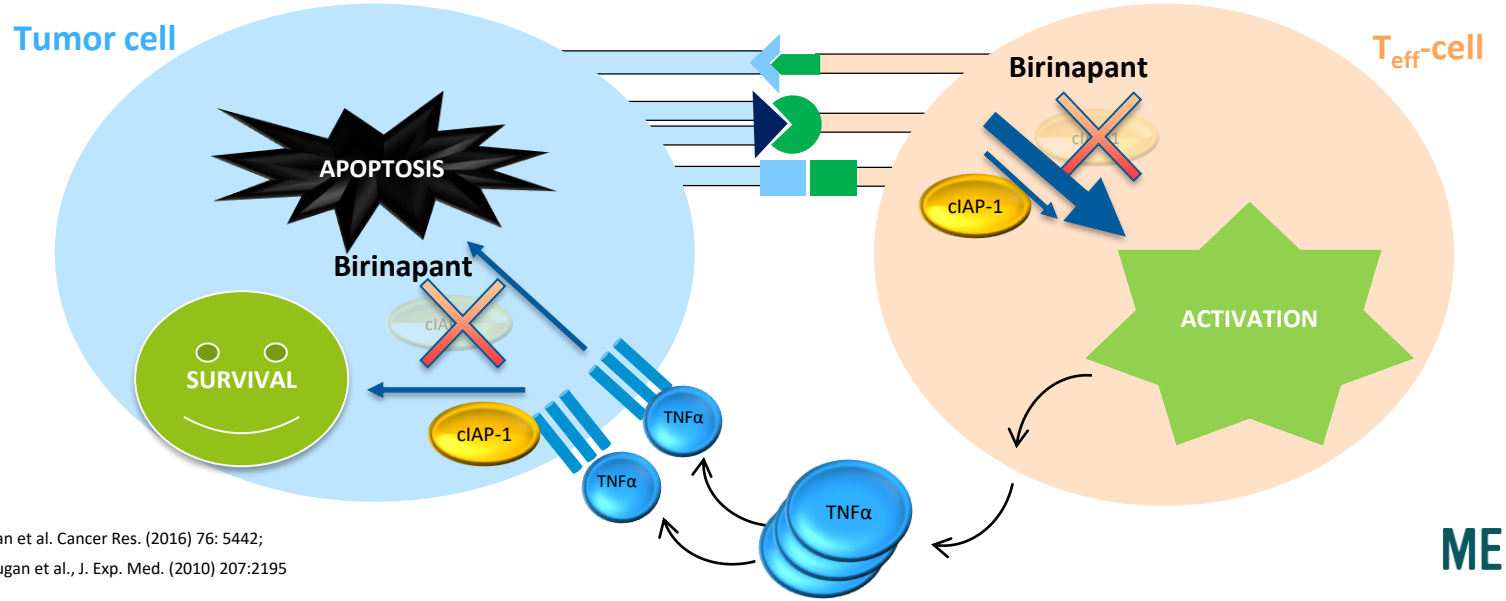
Dual action enhances cancer cell death

Targeting of cIAPs results in dual action on T-cells and tumor cells

- Enhances pro-apoptotic, and suppresses pro-survival, signaling downstream of TNF-α
  - Rationale for combining birinapant with other cancer treatments in which efficacy could be enhanced by pro-apoptotic signaling, especially those known to enhance TNF-α expression, e.g. radiotherapy[1]
- Augments human T cell responses to physiologically relevant stimuli[2]



1. Eytan et al. Cancer Res. (2016) 76: 5442;
2. Dougan et al., J. Exp. Med. (2010) 207:2195

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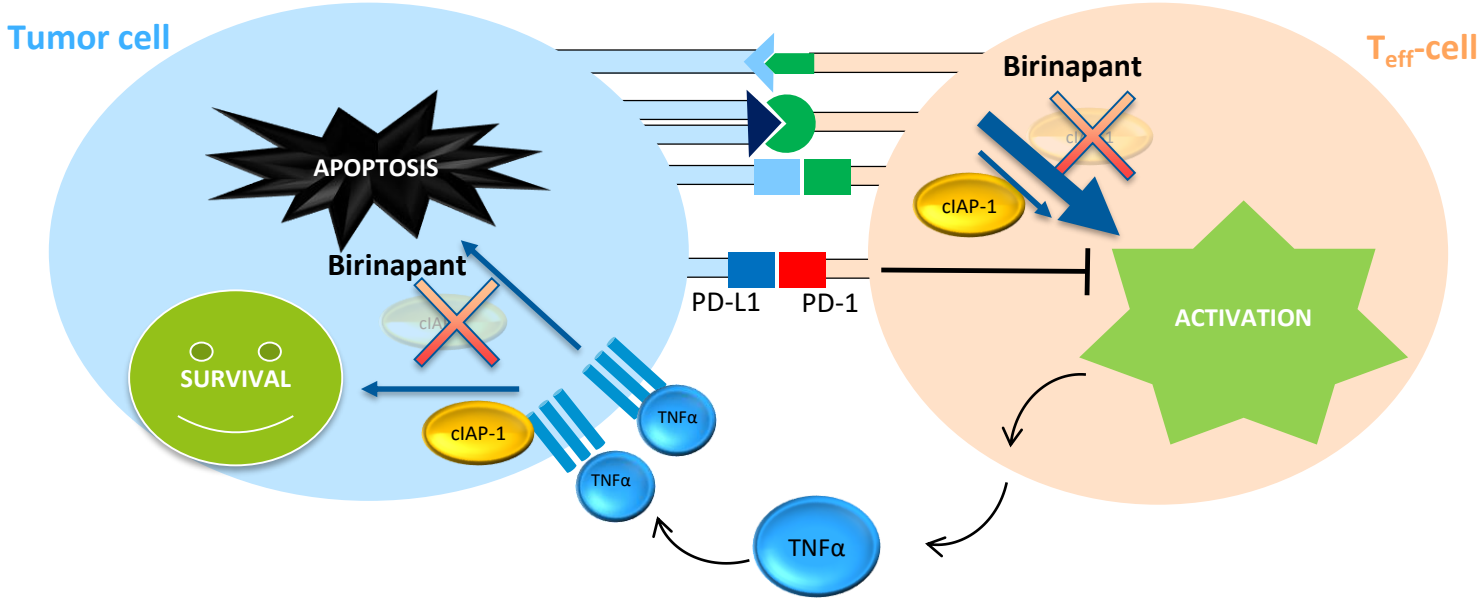
cIAPs and PD-L1 cooperate to protect tumors from the CTLs

Tumor cells acquire resistance to T cell–derived cytokine-mediated antitumor effects to evade the immune response

- PD-L1 expression down-regulates TNF-α release



Kearney et al., Sci. Immunol. 3, eaar3451 (2018);
Kearney et al., Cell Death and Differentiation (2017) 24, 1705–1716

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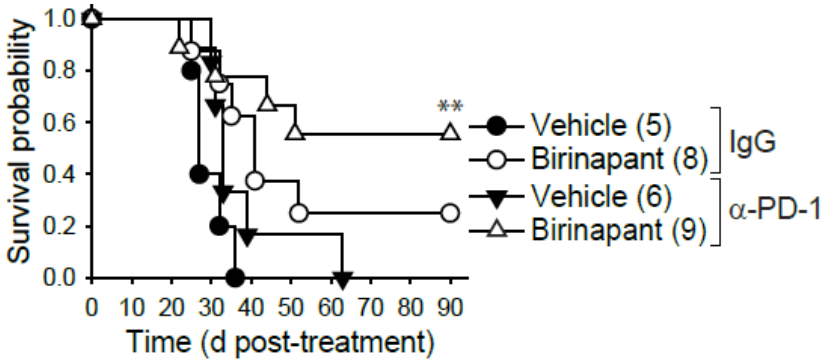
Potential to enhance patient response with immune-oncology therapies

Strong rationale for combination with Keytruda®

- Birinapant/anti-PD1 mAb combo showed enhanced activity in preclinical models[1] compared to either agent alone

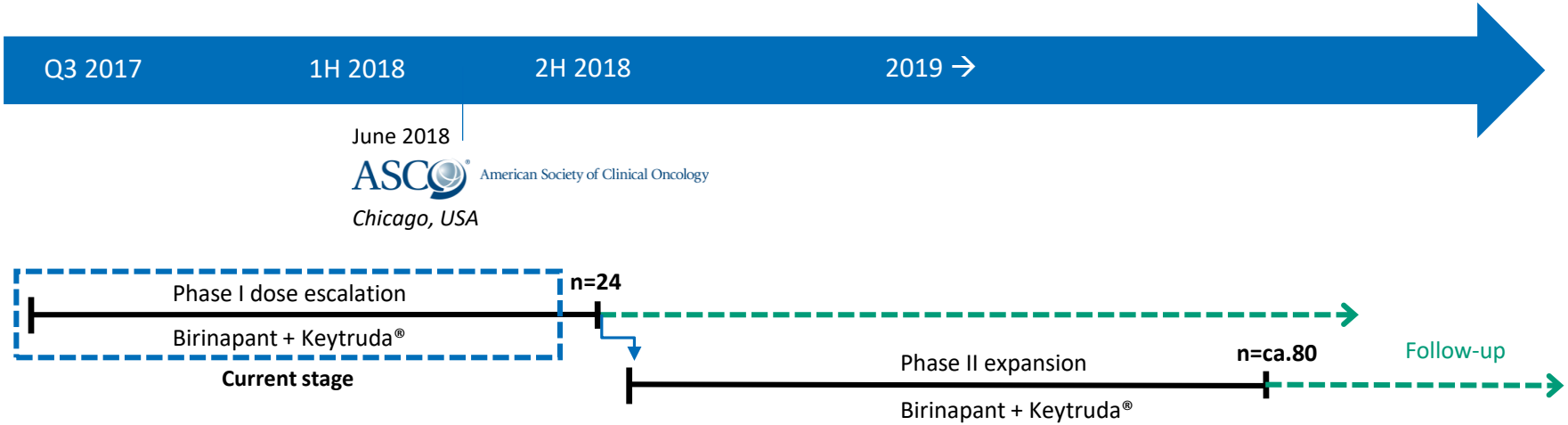


Phase I/II study underway in collaboration with MERCK

- Development collaboration for the Phase I/II study in solid tumors
- Keytruda® provided at no cost
- Joint Development Committee to oversee the study, bringing Merck's immuno-oncology expertise
- Medivir retains full global rights to birinapant and data

1. Solid tumor model: Beug et al., Nature Communications (2017) 8:14278; Multiple myeloma model: Chesi et al., Nature Med. (2016) 22, 1411–1420

Birinapant/Keytruda® combination: Phase I/II Study underway



Phase I: Sequential group dose-escalation to determine the dose-limiting toxicity and recommended Phase II dose, in combination with Keytruda®

Phase II: Safety and tolerability of the recommended dose of birinapant, in combination with Keytruda® in defined disease cohorts:

MSS metastatic Colorectal cancer
Ovarian cancer
Cervical cancer
Other cancer types exploratory cohort

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Birinapant/Keytruda® combination: Dose Expansion Cohorts

Rationale for the selection of the three principal dose expansion cohorts:

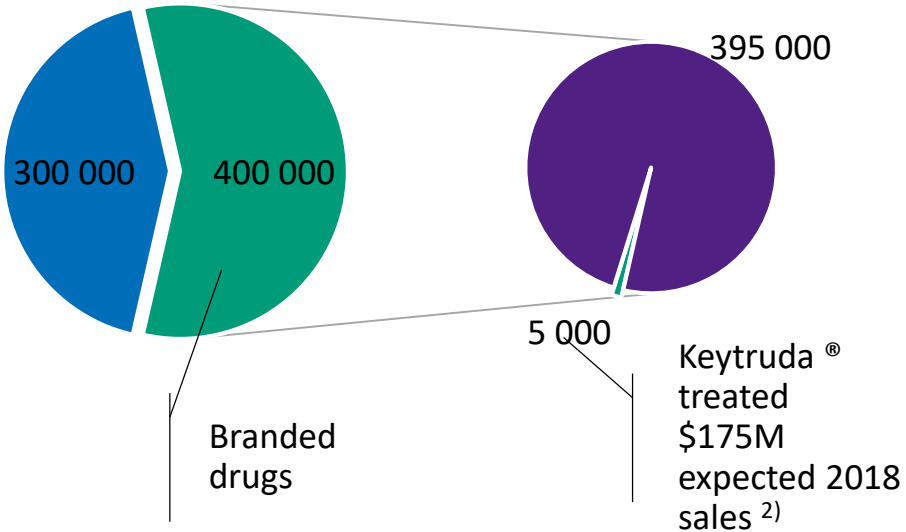
- Very low response rates to Keytruda® monotherapy,

AND either

- Evidence of amplification of cIAPs in those tumor types, or
- Clinical responses to birinapant administered as monotherapy or in combination with chemotherapy, or
- Clinical responses to birinapant and evidence of cIAP amplification

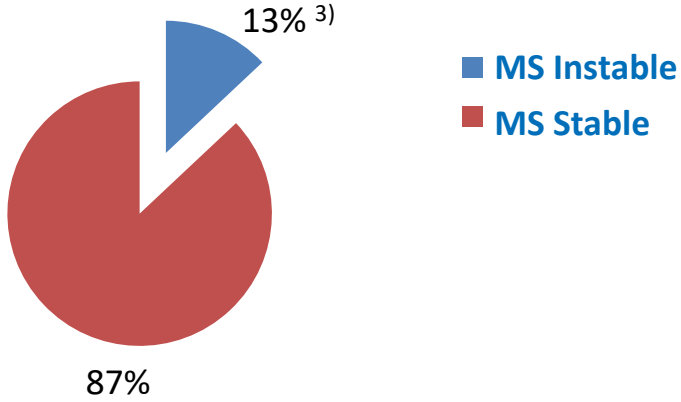
Keytruda® not able to reach the majority of colorectal cancer patients

Significant number of drug treated patients [1)]



PD-1 inhibitors not effective in the largest genotype in colorectal cancer

- There are two genotypes of colorectal tumors:



- Keytruda ® and other PD1 inhibitors only efficacious/indicated in one of them, MSI genotype

1) 7 major markets. Datamonitor Epidemiology 2016
2) Datamonitor Forecast Dec 2017
3) Merck ASCO Event June 6, 2016

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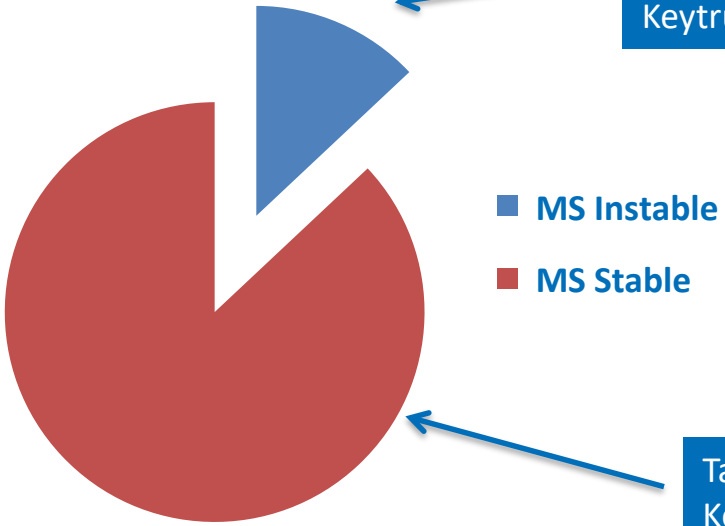
Birinapant may offer access to larger market for Keytruda®

- One of the cohorts in the phase II part of the birinapant/Keytruda® combination study will be patients with MSS-type colorectal cancer



- For 2020, it is projected that Keytruda® will have sales of $1B in MSI-type colorectal cancer [1)]
- If MSS patients could be effectively treated by the combination, the projected sales could be up to 7 times larger

1) Datamonitor Forecast Dec 2017. 7 major markets

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MIV-818 for liver cancers

HCC is 3rd leading cause of cancer-related death worldwide

Hepatocellular Carcinoma

- Orphan disease in Western markets, but one of fastest growing and most deadly cancers in USA
- Increasing incidence of NASH is becoming the driver of HCC is the west, replacing chronic hepatitis C
- High incidence in China and other east Asian countries
- Genetically heterogeneous leading to limited effect of molecularly targeted therapies

Patients with advanced HCC are in need of new treatments:

1st line treatment → **2nd Line Options**

Multikinase Inhibitors
- Sorafenib
- Lenvatinib

- ~3 month survival benefit

(Multi)kinase Inhibitors
- Regorafinib
- *Cabozantanib*
- *Ramucirumab (AFP^{hi} patients only)*

- Incremental survival benefit

PD-1 antagonists
- Nivolumab

- 15-20% ORR
- Survival benefit yet to be defined

Howlader et al. (eds). SEER Cancer Statistics Review, 1975-2011, National Cancer Institute. Bethesda, MD, http://seer.cancer.gov/csr/1975_2011/

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Medivir's Vision for MIV-818

MIV-818 has the potential to become the first liver-targeted orally administered drug to address HCC and other liver cancers

Current and Future Development

- MIV-818 is being developed for advanced HCC patients as a stand alone treatment or in combination with standard of care in 2nd or 3rd line
  - European Phase I/II as stand-alone agent about to start in patients with advanced liver cancer
  - Preclinical data support development in combination with sorafenib and other multikinase inhibitors in advanced HCC
  - Preferential conversion to the active metabolite under hypoxic conditions provides preclinical rationale for future development in combination with TACE and other embolic treatments for intermediate-stage HCC patients
- Development in patients with intra-hepatic cholangiocarcinoma and metastatic liver disease also planned

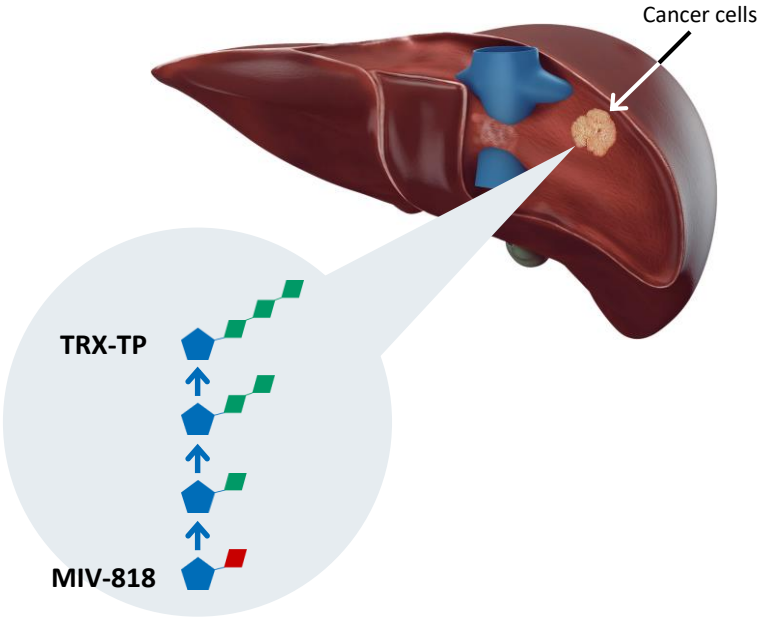
Mechanism of Action

Chain-terminating inhibition of DNA synthesis

- **MIV-818** is an orally administered nucleotide prodrug of the active metabolite **troxacitabine triphosphate** (TRX-TP)
- When incorporated into DNA, TRX-TP causes double strand DNA breaks and cell death
- Troxacitabine progressed to Phase II/III, with clinical responses observed in several cancers, but development halted due to narrow therapeutic window

Liver targeting to deliver high levels of the active metabolite to the liver while minimizing exposure elsewhere

- **MIV-818** has been designed to minimize systemic exposure and limit the toxicity of troxacitabine by primarily targeting liver cells



Prodrug – an inactive molecule that is converted into the active drug within the body

1. Babusis et al., Antimicrob Agents Chemother. (2018) 62:e02587-17 doi: 10.1128/AAC.02587-17

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Potential to improve efficacy and safety for patients with liver cancers

Improve a nucleoside with Medivir prodrug technology

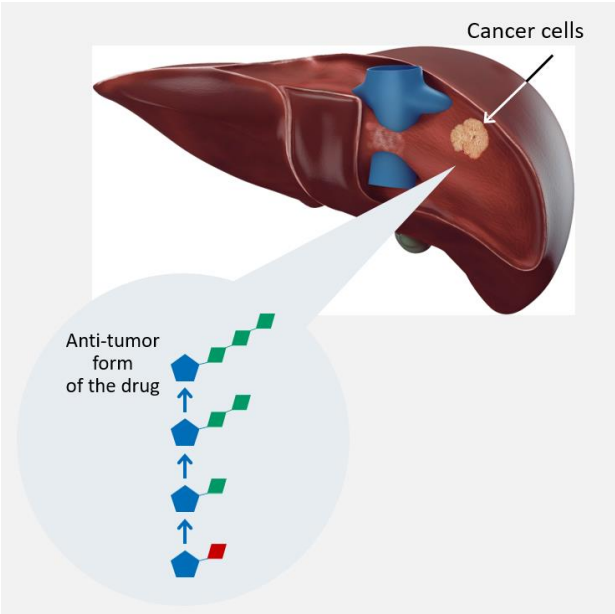
Troxacitabine
(nucleoside)

- **Active** in preclinical cancer models and in clinic
- Failed in clinic due to systemic dose-limiting **toxicities**
- Targets process essential for proliferation, so independent of driver mutations

Medivir prodrug technology →

MIV-818
(liver-targeted nucleotide prodrug)

- **Exhanced activity** 10x more potent against HCC cell lines than parent troxacitabine
- **Selectivity for cancer** Active on HCC cells while sparing non-cancerous hepatocytes
- **Improved delivery to the liver** >100-fold relative to systemic exposure of troxacitabine
- **Synergy with multikinase inhibitors** (e.g. sorafenib)
- **Market exclusivity** with full new chemical entity patent protection



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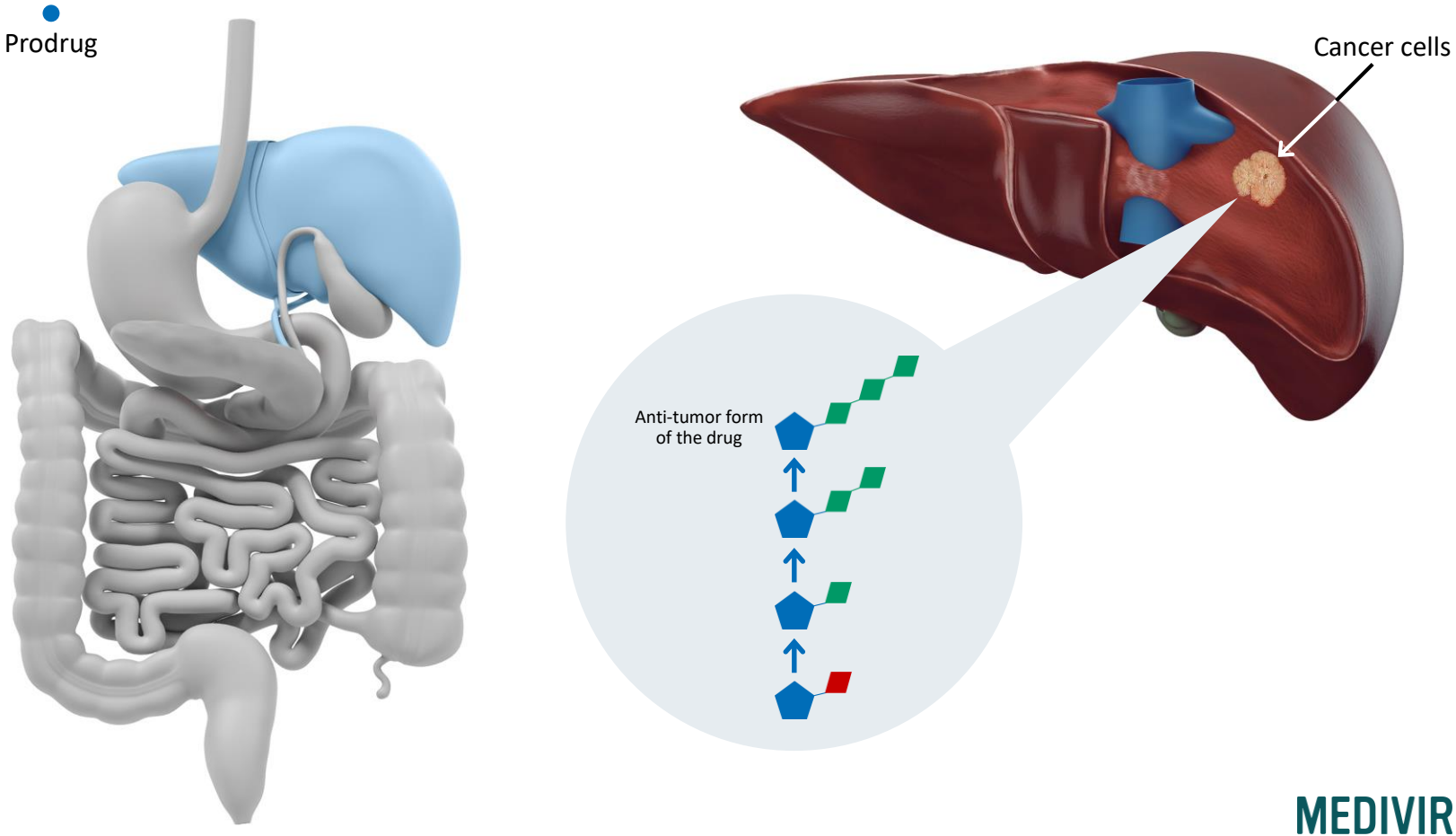
Prodrugging targets the active metabolite of MIV-818 to the liver



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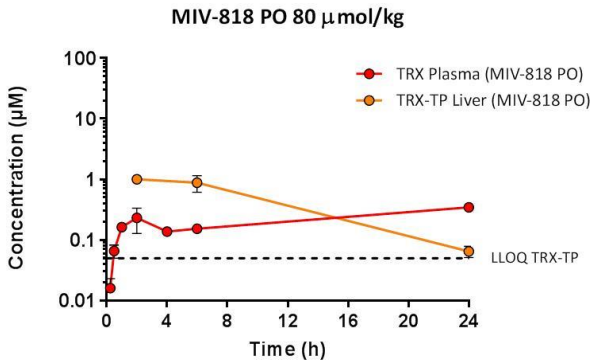
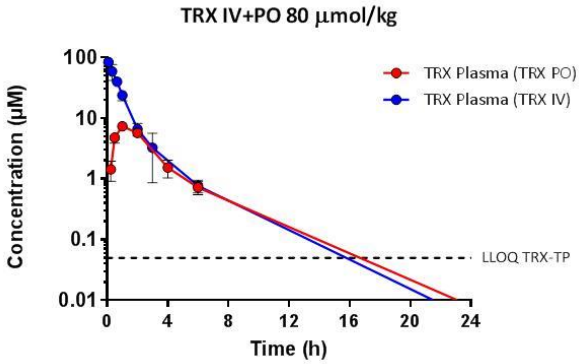
Demonstration of Liver Targeting in rats





Drug	Dose (µmol/kg)	Adm Route	Analyte	Tissue	C_{max} (µM)	$AUC_{0\text{-}24h}$ (µM.h)	TP_{liver} / TRX_{plasma} ratio
MIV-818	80	PO	TRX TRX-TP	Plasma Liver	0.35 1.0	5.4 10	**1.9**
TRX	80	IV	TRX TRX-TP	Plasma Liver	102 <0.05	76 <1.2	**<0.016**
TRX	80	PO	TRX TRX-TP	Plasma Liver	7.4 <0.05	22 <1.2	**<0.055**

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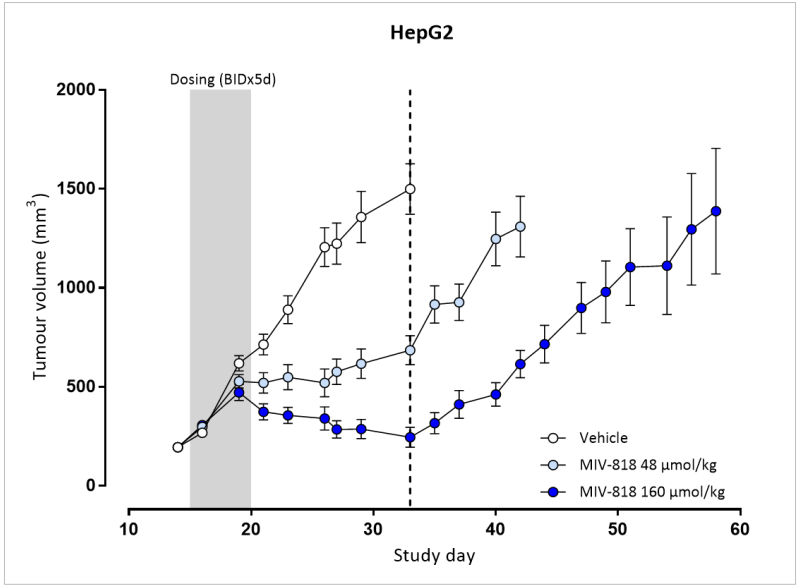
Dose-dependent tumour growth inhibition demonstrated with MIV-818



- Dose-dependent anti-tumor effect seen in HepG2 mouse xenograft model[1]
- 96% tumour growth inhibition at study day 33 after 160 µmol/kg MIV-818 administered orally twice daily for 5 days[1]
- Troxacitabine triphosphate also active in other mouse models of HCC[1,2]

1. Rizoska et.al. EASL HCC Summit (2018), Poster: P02-04, http://www.medivir.se/v5/images/pdf/2018/MIV-818-HCC-Summit-2018-Abstract-P02-04.pdf;
2. Albertella et al. 2017 AACR Annual Meeting, Poster 5101, http://www.medivir.se/v5/images/pdf/2017/AACR-2017-Poster-MIV-818-Albertella-final.pdf

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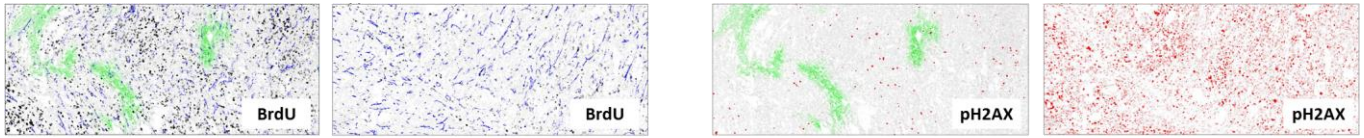
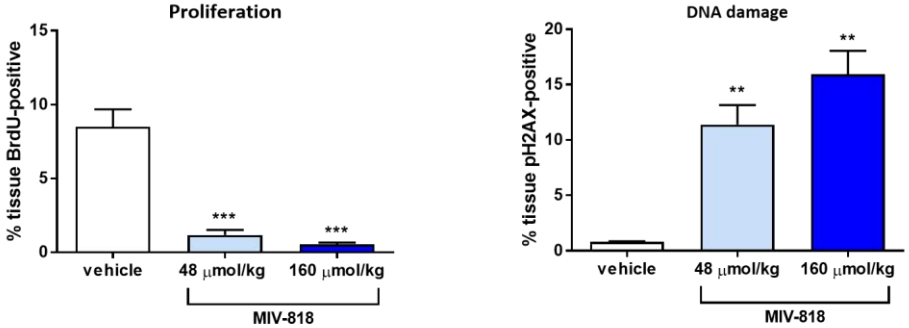
Inhibition of proliferation and induction of DNA damage



Photoimages of sections stained for BrdU (black), pH2AX (red), mouse vasculature (blue) and hypoxia (green) from control (vehicle) and MIV-818 (160 µmol/kg) treated mice



*Data are presented as mean±SEM (n=3). **P<0.01, ***P<0.001, compared to vehicle.*

- MIV-818 shows dose-response effects on proliferation (BrdU) and DNA damage (pH2AX) in the HepG2 tumours after oral dosing at 48 and 160 µmol/kg twice daily for 5 days[1]

1. Rizoska et.al. EASL HCC Summit (2018), Poster: P02-04, http://www.medivir.se/v5/images/pdf/2018/MIV-818-HCC-Summit-2018-Abstract-P02-04.pdf

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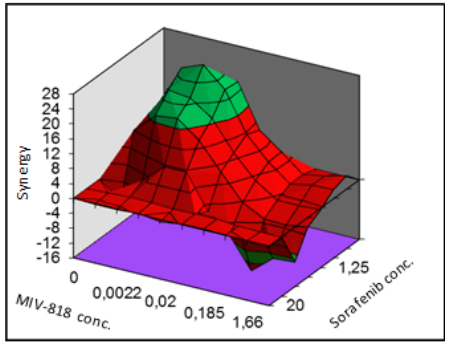
The combination of MIV-818 and sorafenib is synergistic

MIV-818 concentration (µM)

Sorafenib concentration (µM)	0	0,00076	0,0022	0,0068	0,02	0,062	0,185	0,55	1,66	5
20	100,41	100,23	100,23	100,55	99,76	100,41	100,46	100,18	100,09	100,09
10	99,62	99,9	100,18	100,18	99,9	100,13	100,23	99,81	99,71	99,62
5	66,86	59,97	67,37	76,26	80,26	76,54	79,33	75,52	82,03	82,68
2,5	25,54	32,14	38,24	47,64	52,02	57,41	67,98	74,49	78,26	79,61
1,25	10,6	16,46	26,7	36,38	42,99	50,15	64,86	72,58	77,24	78,17
0,62	0	1,48	15,48	29,77	36,8	43,45	61,46	70,21	76,82	77,75
0,31	0	0	0	19,44	31,91	38,47	59,32	68,63	75,38	77,66
0	0	0	0	5,34	17,25	33,96	57,23	70,12	75,38	76,07

Hep3B Percent growth inhibition for each concentration combination (WST-8 assay, Kit8)



Synergy plot (MacSynergy II software)
Hills indicate synergistic interactions

- Synergy demonstrated in Hep3B (data shown above) and other HCC cell lines[1]
- Synergy all observed between MIV-818 and other multikinase inhibitors
- Administration of the combination of MIV-818 and sorafenib leads to superior anti-tumor effect *in vivo* compared to either agent alone[2]

1. Bethell et al. International Liver Conference 2017, Presentation SAT-123, http://www.medivir.se/v5/images/pdf/2017/1704-MIV818-EASL-ILC-April-2017-poster-Albertella.pdf
2. Rizoska et al. 2018 AACR Annual Meeting, Presentation 2930

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MIV-818: Gearing up for Phase I study start

September 2016
ILCA
Vancouver, Canada

February 2017
The Asian Pacific Association for the Study of the Liver (APASL)
Shanghai, China

February 2017
EASL The Home of Hepatology | HCC SUMMIT
Geneva, Switzerland

April 2017
EASL The Home of Hepatology | THE INTERNATIONAL LIVER CONGRESS™ 2017
AACR American Association for Cancer Research
Washington DC, USA

☑ **Completed** MIV-818 (liver cancer) IND-enabling preclinical studies

March 2018
EASL The Home of Hepatology | HCC SUMMIT
Geneva, Switzerland

April 2018
AACR American Association for Cancer Research
Chicago, USA

☐ Planned start of Phase I study (2H 2018)

MEDIVIR

Phase 1/2 Study

Study population

- Patients with advanced hepatocellular cancer (HCC) and other liver cancers

Objectives

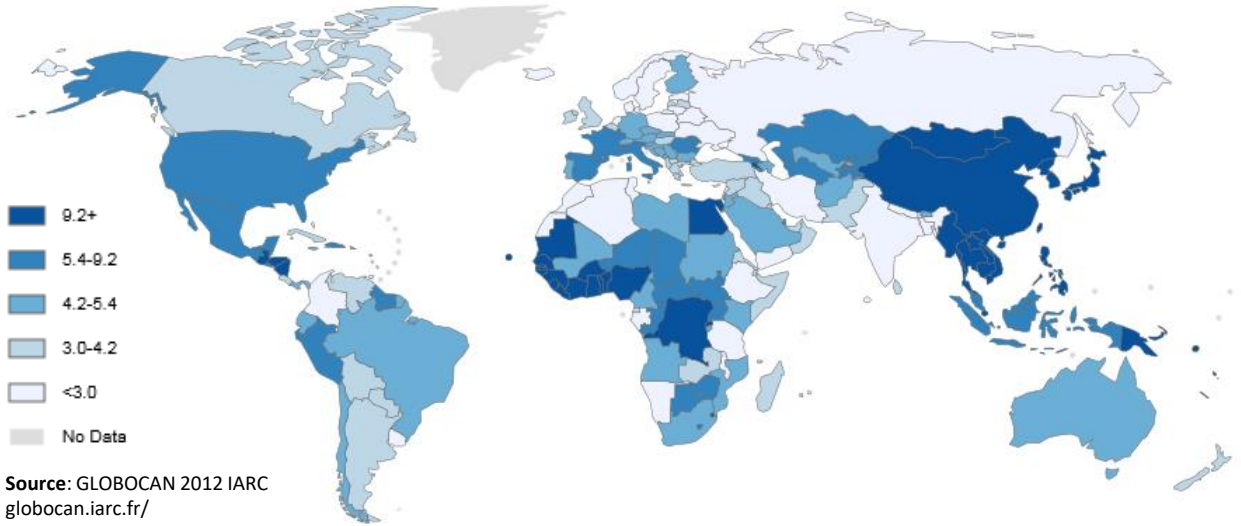
- Phase 1 part
  - Assess safety and tolerability of MIV-818
  - Determine the recommended Phase 2 dose (RP2D)
  - Assessment of clinical response
- Phase 2 part
  - Clinical activity of the RP2D will be assessed by RECIST 1.1
  - Separate dose expansion cohorts in HCC and intrahepatic cholangiocarcinoma
  - Panel of translational biomarkers

Study planned to start 2H 2018

MIV-818 with a unique opportunity in liver cancer

- Global annual incidence of primary liver cancer is 780,000
  - ~80% is hepatocellular carcinoma (HCC)
  - ~15% is intrahepatic bile duct cancer
- HCC is a orphan cancer in Western markets
- One of fastest growing and most deadly cancers in US [1)]

Liver cancer
incidence/100,000



Source: GLOBOCAN 2012 IARC
globocan.iarc.fr/

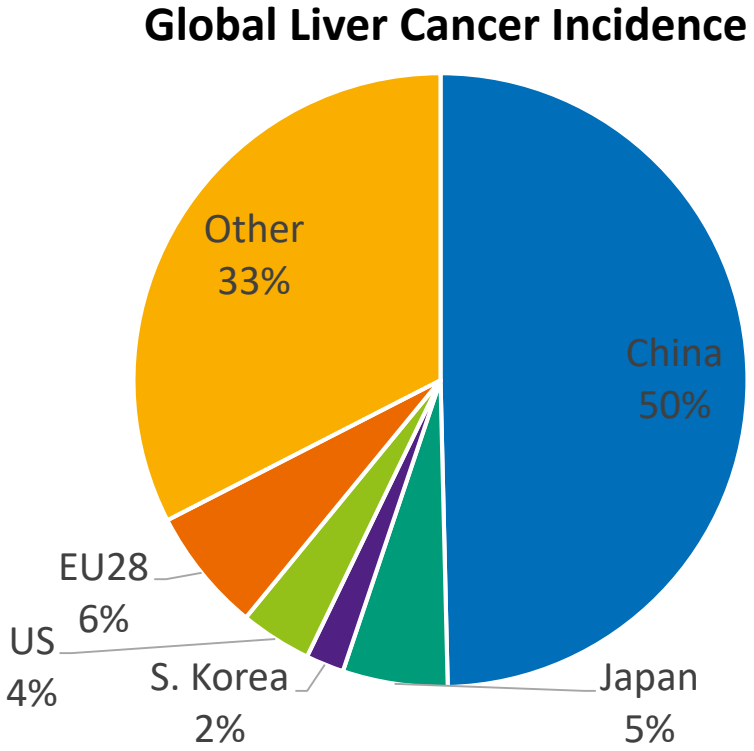
1) Howlader et al. (eds). SEER Cancer Statistics Review, 1975-2011, National Cancer Institute. Bethesda, MD, http://seer.cancer.gov/csr/1975_2011/

MEDIVIR

Commercialization and co-development opportunity in Asia

- Incidence rate of liver cancer is significantly higher in Asia, there is a large market opportunity in this region with approximately 75% of liver cancer incidence occurs in Asia, with 50% in China alone

- Medivir plans to co-develop and commercialize in Asia with a partner



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Outlook and financial summary

Key milestones throughout the year

Track record of delivery

Coming events

JAN – JUL 2018 → **AUG 2018** →

☑ **Completed**
MIV-818 (liver cancer) IND-enabling preclinical studies

MIV-818
EASL | HCC SUMMIT
Geneva, Switzerland
March 2018

MIV-818
AACR American Association for Cancer Research
Chicago, USA
April 2018

MIV-711
2018 WORLD CONGRESS OSTEOARTHRITIS
Liverpool, United Kingdom

Remetinostat
IID 2018 International Investigative Dermatology
Orlando, USA
May 2018

Birinapant
ASCO American Society of Clinical Oncology
Chicago, USA
June 2018

☑ **Completed**
MIV-711 Phase IIa osteoarthritis extension study

- ☐ Completion of the dose escalation portion of the birinapant Phase I/II study in combination with Keytruda® (2H 2018)
- ☐ Start of MIV-818 (HCC nuc) Phase I study (2H 2018)
- ☐ Start of Phase III CTCL study remetinostat (2019)

MEDIVIR

Cash position and shareholder base

CASH POSITION

THE SHARE

Nasdaq Stockholm main board listed (Ticker: MVIR)	One class of shares	Market Cap[1)] >900m SEK (>USD 100m)	International Ownership ~30%	Top 20 shareholders ~51%

1) As of Aug 1, 2018. Stock price 37.35 SEK. 24,287,818 total Class B shares.

MEDIVIR

Sum up and Q&A

Why Medivir?

- Strong pipeline from discovery through clinical stages focused in oncology
- Upcoming catalysts with newsflow in multiple projects
- Near-term opportunities for revenues from partnerships