LEADING THE WAY IN
BREAKTHROUGH
IMMUNE THERAPIES

Faron AGM presentation

23 APRIL 2021

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and
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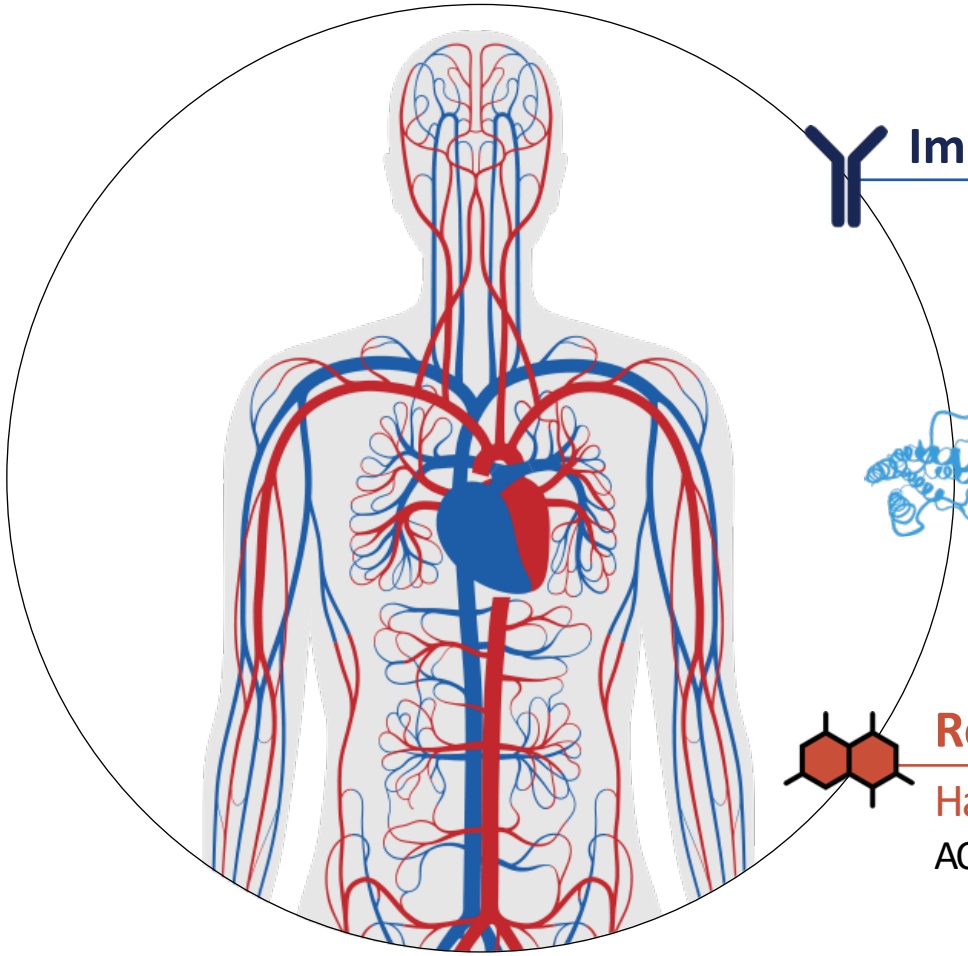
Harnessing the Power of the Immune System

Our approach is focused on activating, suppressing and maintaining the immune system



Immuno-Oncology & Infectious Disease

Clevegen® *(bexmarilimab)*

CLEVER-1 regulates tissue immune status

Organ Protection

Traumakine® (*interferon-beta*)

CD73 controls capillary leakage and escalation of inflammation

Regenerative Medicine

Haematokine®

AOC3-dependent cell replication

Our Pipeline

Building the future of immunotherapy

Therapeutic Space	Program	Indications	Preclinical	Phase I	Phase II	Phase III	Partner
Immuno-Oncology	**Clevegen** *Bexmarilimab* FP-1305-antibody	Solid tumors	MATINS				
		NSCLC[1]	MATINS-05 LUNG				
		Hematological malignancies	MATINAML				
Organ protection	**Traumakine** *Interferon β* FP-1201-lyo	ARDS[2] & COVID-19	REMAP–CAP				REMAP-CAP HARVARD MEDICAL SCHOOL
			HIBISCUS				
		Major Cardiovascular Surgery					
		CAR-T induced CRS[3]					
		Acute Kidney Injury					
		Solid Organ Transplantation					
Regenerative medicine	**Haematokine** *New Chemical Entity* AOC3 inhibitor	Hematological malignancies					

Key 2020 events

Including post period

- ***Bexmarilimab* Phase I/II MATINS study showing early clinical benefits in six hard-to-treat solid cancers**
  - Significant survival benefit observed among responders
  - More frequent and higher dosing to be investigated in MATINS + combination studies planned
  - Increased understanding of immune suppressive nature of Clever-1 in T cell activation suggests broader applicability of *bexmarilimab*

- **IV interferon beta-1a, Traumakine®, to protect organ damage, now also investigated as potential COVID-19 treatment**
  - Ongoing COVID-19 investigations in global REMAP-CAP and upcoming US HIBISCUS study, supported by $6.1 million funding from Coronavirus Aid, Relief, and Economic Security (CARES) Act
  - Application for additional patent protection relating to the induction of CD73 for organ protection
  - Expanded research into additional organ protection indications, alongside 59th Medical Wing of US Air Force and US Army Institute of Surgical Research to explore organ protection in major combat wounds causing systemic inflammation

- **Rights acquired for third pipeline programme – AOC3 protein inhibitor, Haematokine® – for use in regenerative medicine and to treat hematological malignancies**
  - IND-enabling studies ongoing and positive indications of global patent protection

Vuoden 2020 keskeisiä tapahtumia

Sisältää myös vuoden 2021 tapahtumat ennen tilinpäätösjulkaisua

- ***Bexmarilimab*** **faasi I/II MATINS-tutkimus osoittanut aikaisen vaiheen tehohyödyn vaikeasti hoidettavissa pitkälle edenneissä syövissä**
  - Hoitoon reagoivien potilaiden elinajan odote kasvanut merkittävästi
  - Nykyistä annostelua korkeimpia ja tiheimpiä annostelumuotoja testataan sekä MATINS-potilaissa että uusissa lääkekombinaatioissa
  - Uusi hoitoon vaikuttava havainto on, että syöpäpotilailla on kohonnut liukoinen Clever-1 pitoisuus, joka rajoittaa suoraan puolustusjärjestelmän T-solujen aktivoitumista. Liukoisen Clever-1:n neutralisoiminen laajentaa bexmarilimabin käyttömahdollisuuksia

- **Suonensisäinen interferoni beta-1a, Traumakine®, joka on ensisijaisesti tarkoitettu suojaamaan keskeisiä elimiä kuten keuhkoja, testataan nyt myös COVID-19 potilailla**
  - COVID-19 potilailla meneillään globaalissa REMAP-CAP ja USA:han rajoittuneessa HIBISCUS tutkimuksissa, joista jälkimmäistä USA:n puolustusministeriö tukee 6.1 miljoonalla dollarilla
  - Traumakinen laajentuvaa käyttösuojaa sekä kudosvauriotutkimuksia on jatkettu suunnitelmien mukaan, mm. USA:n ilmavoimien kirurgisen tutkimusyksikön kanssa

- **Yhtiön kolmanneksi hankkeeksi on noussut Haematokine®, luuytimen verisolutuotannon aktivoiminen**
  - Keskeisiä pre-kliinisiä tutkimuksia meneillään, jotka mahdollistaisivat kliinisten tutkimuksien aloittamisen

Key financial and corporate information

Including post period

- Cash balances on 31 December 2020 of **€4.1 million** (2019: €7.1 million)
- Loss for the period for the financial year ended 31 December 2020 was **€16.9 million** (2019: €13.3 million)
- Net assets on 31 December 2020 were **€-1.8 million** (2019: €1.6 million)
- **€14.0 million** gross (€13.0 million net) raised in April 2020 from new and existing shareholders through issuance of total of 3,500,000 new ordinary shares
- Grants, loans and loan guarantees totaling **€7.9 million** received from Business Finland, The European Innovation Council and Finnvera (€2.2 million of funds received during the period, the rest will continue to be received post period)
- **€15.0 million gross** (approximately €14.4 million net) raised post period in February 2021, from new and existing shareholders through an issuance of 3,521,127 new ordinary shares
- Awarded **6.1 million USD** grant post period for the HIBISCUS study by the US Department of Defense (DoD)

Avainluvut ja Pääkohdat

Sisältäen tilikauden jälkeisiä tapahtumia

- Rahavarat 31. joulukuuta 2020: **€4,1 miljoonaa** (2019: €7,1 miljoonaa)
- Tilikauden tappio 31. joulukuuta 2020 oli **€16,9 miljoonaa** (2019: €13,3 miljoonaa)
- Oma pääoma 31. joulukuuta 2020 oli **€-1,8 miljoonaa** (2019: €1,6 miljoonaa)
- Faron keräsi **€14,0 miljoonaa** brutto (€13.0 miljoonaa netto) suunnatulla osakeannilla sekä uusille että nykyomistajille huhtikuussa 2020
- Tukia, lainoja sekä lainatakuita yhteensä **€7,9 miljoonaa** Business Finlandilta, Euroopan Innovation Councililtä sekä Finnveralta, joista 2,2 miljoonaa euroa tuloutettiin tilikauden 2020 aikana
- Faron keräsi **€15,0 miljoonaa** tilikauden jälkeen helmikuussa 2021 laskemalla liikkeelle 3.521.127 uutta osaketta sekä uusille että nykyomistajille
- Yhdysvaltojen Puolustusministeriö (DoD) myönsi tilikauden jälkeen **6,1 miljoonan USD** tuen HIBISCUS ohjelmaan

Macrophage-Guided Immunotherapy

CLEVEGEN
The immune switch

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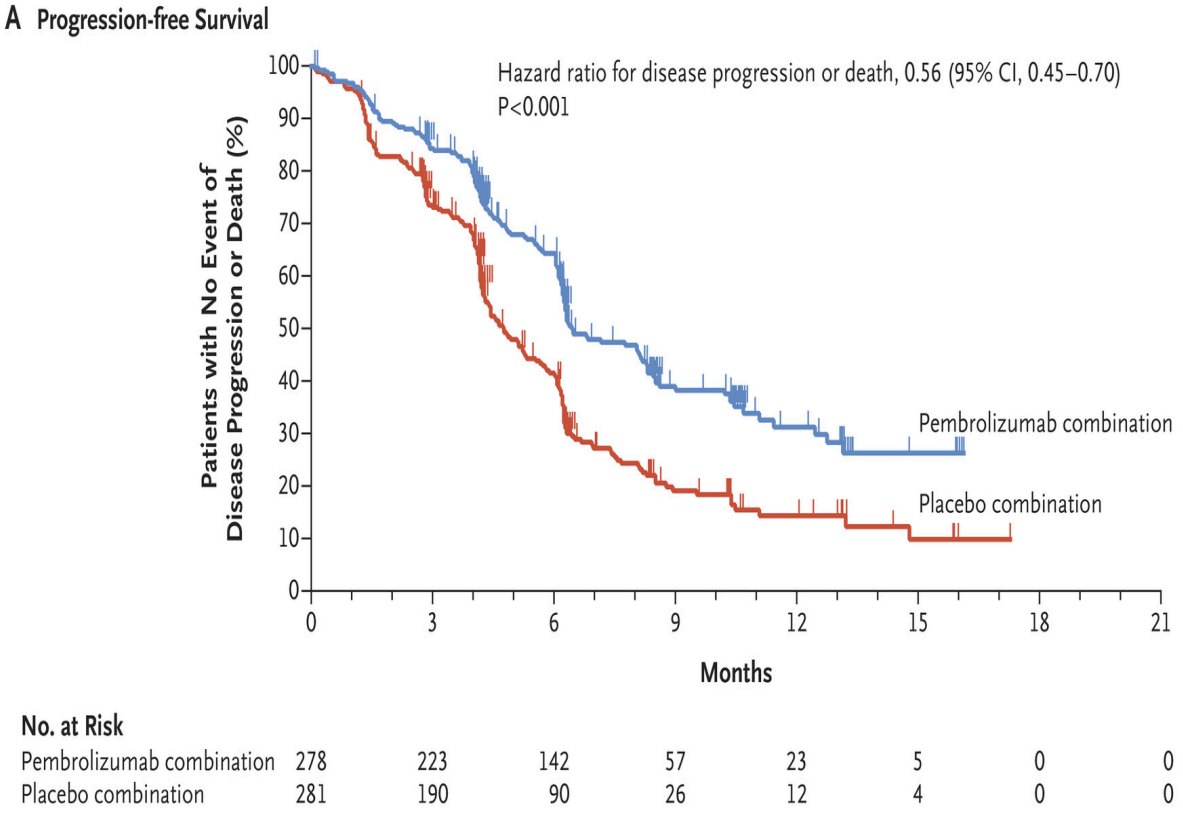
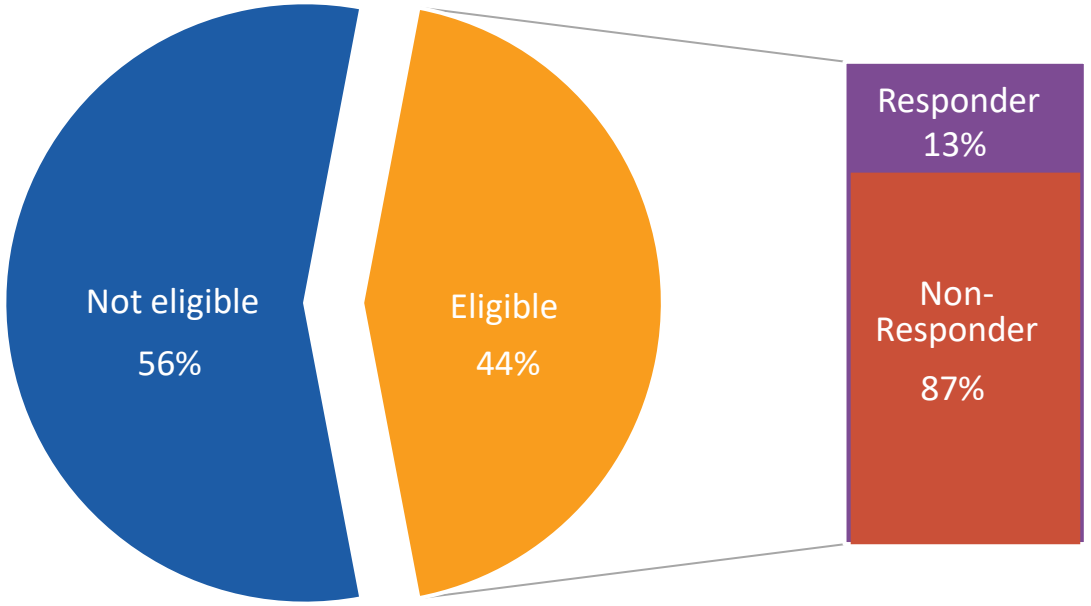
The Revolution of Checkpoint Inhibitors (CPIs) to Treat Cancer

CPI market estimated to become $56.5 billion by 2025





CPIs were the first drugs to significantly impact survival in melanoma and lung cancer, which had been considered un-treatable before this

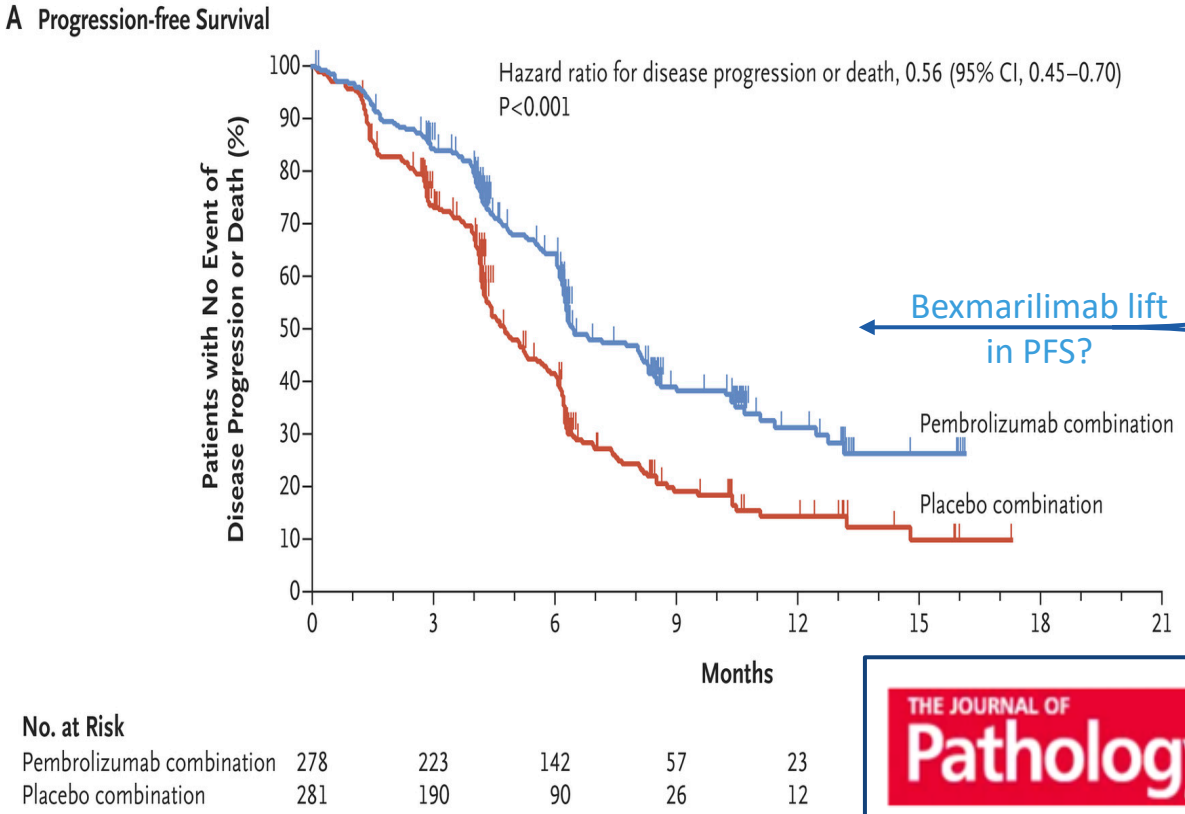
The adoption of CPIs has occurred very quickly due to their success compared to older drugs, despite poor response rates.

Haslam and Prasad (2019) JAMA Netw Open. 2019 May; 2(5): e192535.

Paz-Ares. *et al.* (2018) *NEJM*. 379: 2040-2051

The Desperate Search to Improve Survival Further

Macrophages are considered a key barrier to unlock the next level



Bexmarilimab lift in PFS?

Why Macrophages?

- Macrophages promote tumor growth and metastases
- Macrophages create a highly immunosuppressive environment
- Continued evidence shows that macrophages are key in treatment resistance [2]
- To improve on current strategies, repolarisation of pro-tumoral macrophages has been pointed to as a better strategy[3]
- Targeting tumor associated macrophages (TAMs) has shown promising preclinical results [1,3]

THE JOURNAL OF Pathology

Scavenger Receptor Class A to E Involved in Various Cancers

Sunhyo Ryu[1], Amanda Howland[2], Brendon Song[3], Chakyung Youn[4], and Peter I. Song[5,*]

[1]Boston University School of Medicine, Boston, MA, [2]University of Colorado Denver School of Medicine, Aurora, CO, [3]University of Denver, Denver, CO, USA, [4]Department of Biomedical Science, Research Center for Proteinaceous Materials, Chosun University School of Medicine, Gwangju, Korea, [5]InClinica, Wayne, PA, USA

Original Paper

CD206-positive myeloid cells bind galectin-9 and promote a tumor-supportive microenvironment

Elizabeth Ann L Enninga, Kyriakos Chatzopoulos, John T Butterfield, Shari L Wendy K Nevala, Thomas J Flotte, Svetomir N Markovic

European Journal of Immunology HIGHLIGHTS

REVIEW

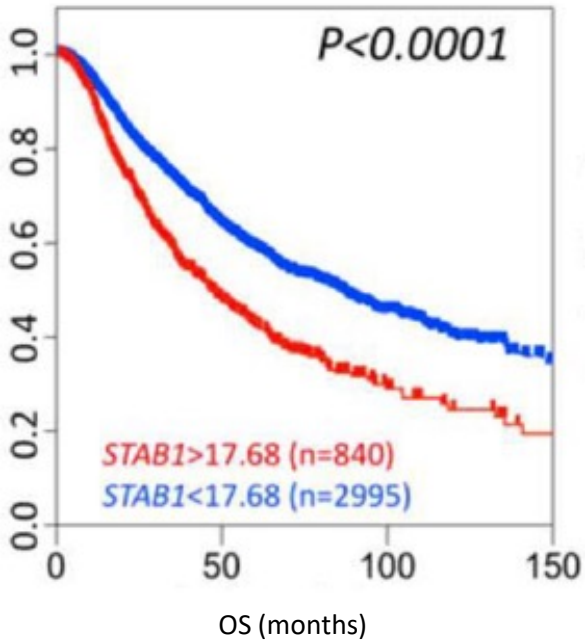
Exploiting scavenger receptors in cancer immunotherapy: Lessons from CD5 and SR-B1

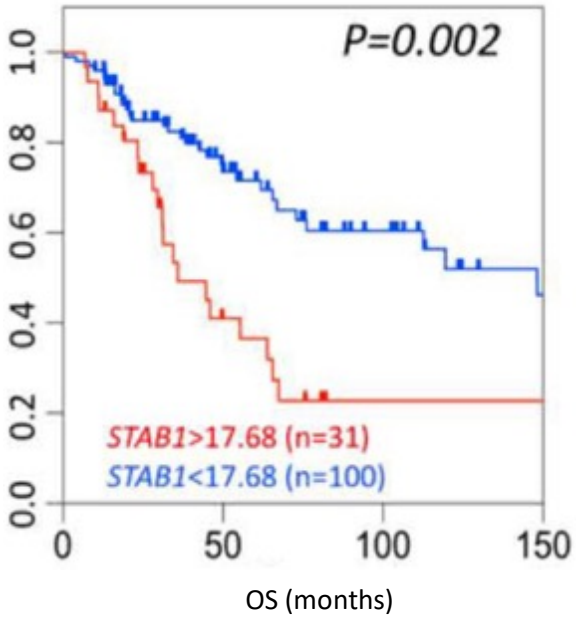
1) Guerriero (2018) Trends in Molecular Medicine. 24 (5) 472-489
2) Jahchan et al., (2019) Front Immunology. 10:1611.
3) Cassetta and Pollard (2018) Nature Reviews: Drug Discovery 17(12) 887-904

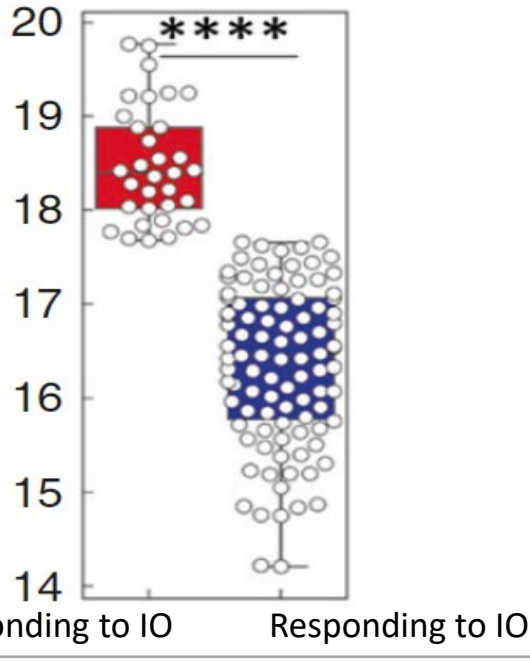
CLEVER-1 Expression is Predictive of Survival, Therapeutic Response, & T-Cell Dysfunction[1]

Kaplan Meier of Clever-1 (STAB1) mRNA expression

TCGA-PANCAN		TCGA
Among all cancer patients	**Among immunotherapy treated patients**	**All immunotherapy treated patients**







CLEVER-1 low

CLEVER-1 high

High CLEVER-1 expression is inversely correlated with overall survival

CLEVER-1 (also known as Stabilin1) is encoded by the gene STAB1

1) Hollmen et al., (2020) BJC. 123. 501-509

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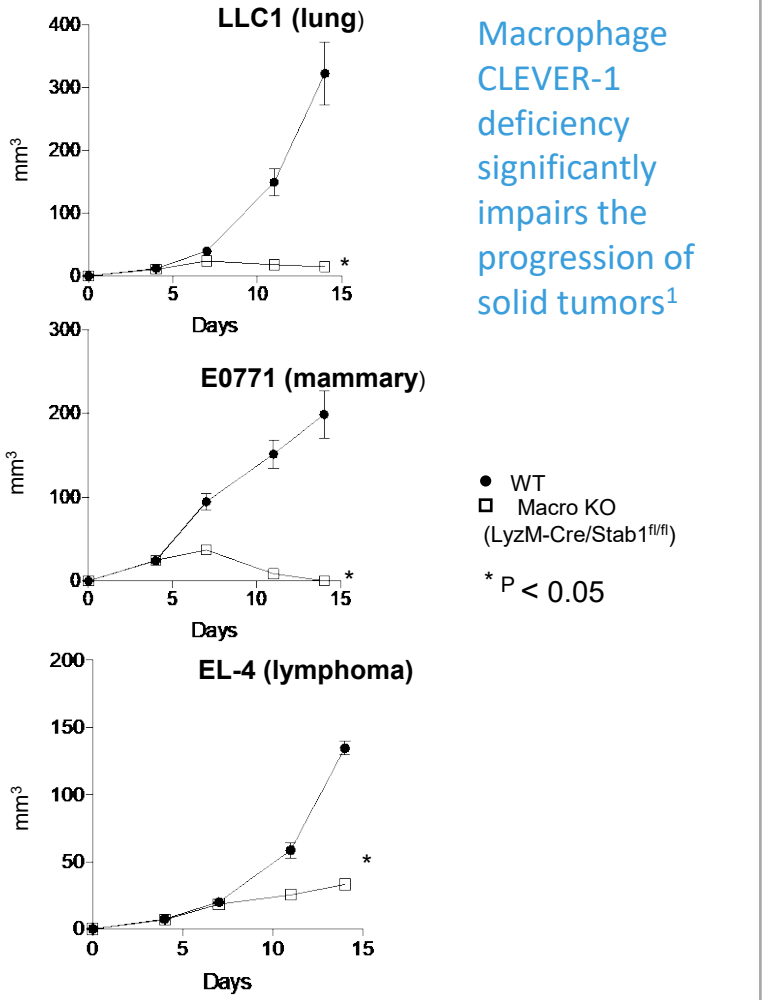
Blocking CLEVER-1 Leads to Tumor Rejection

Single agent activity in checkpoint refractory mice models



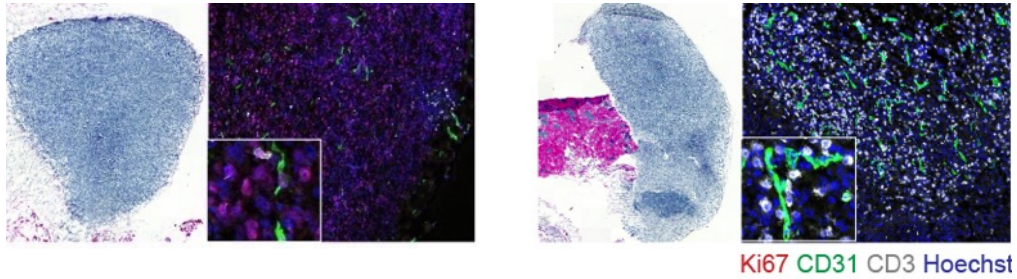
Macrophage CLEVER-1 deficiency significantly impairs the progression of solid tumors[1]

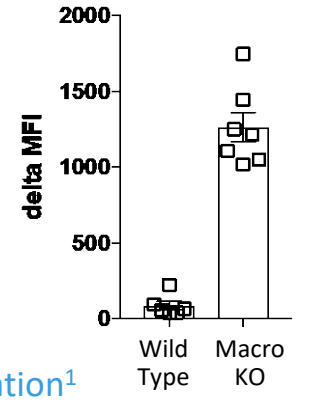
COLD

Wild Type

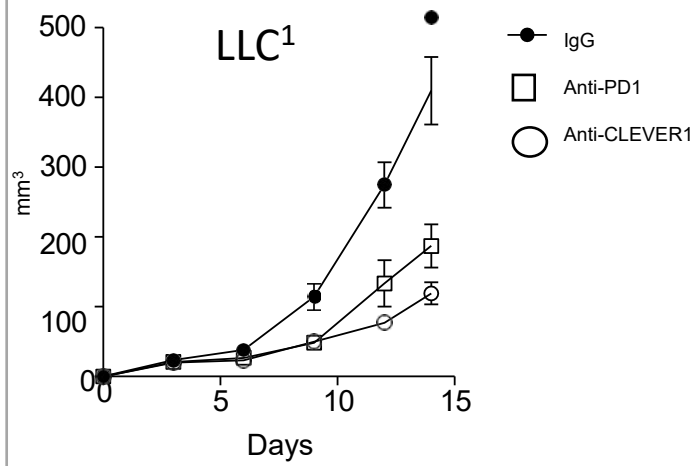
HOT

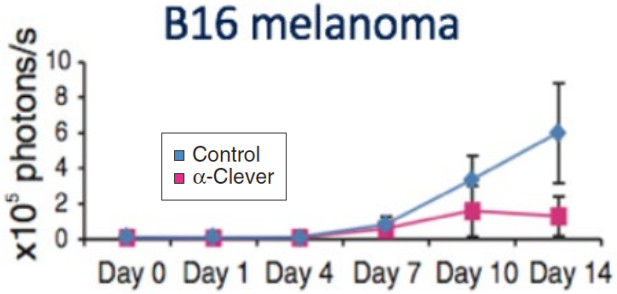
Lyz2-Cre/STAB1$^{fl/fl}$





Blocking of CLEVER-1 leads to significant CD8$^+$ T cell infiltration[1]



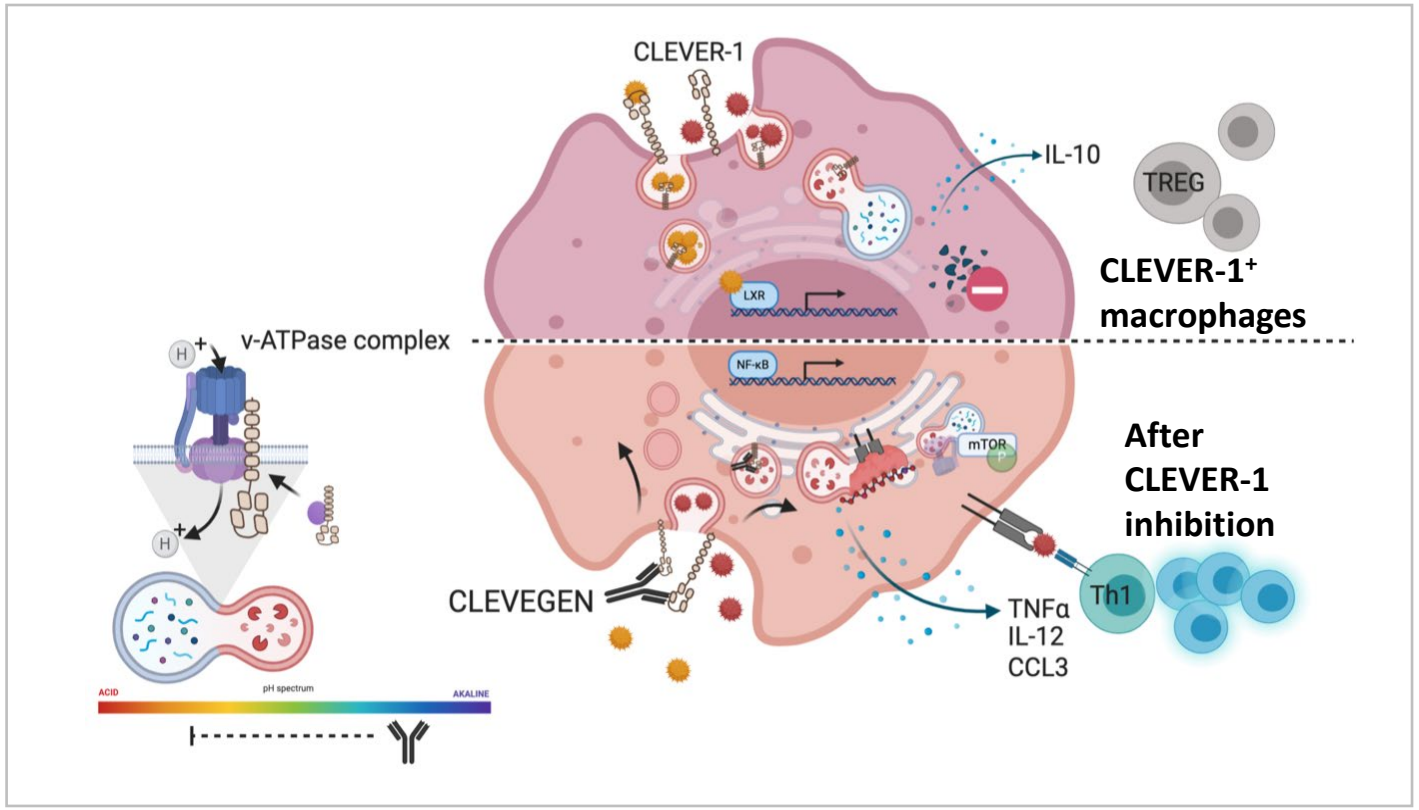


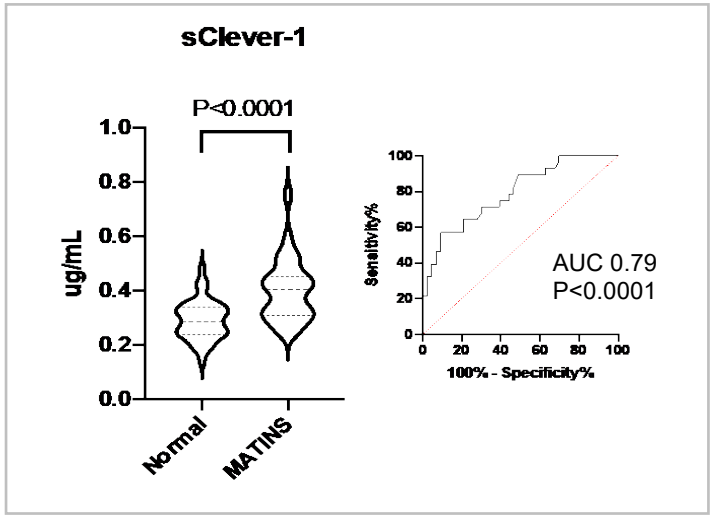
Anti-CLEVER-1 is comparable to anti-PD1 in mouse models of LLC and B16[1,2]

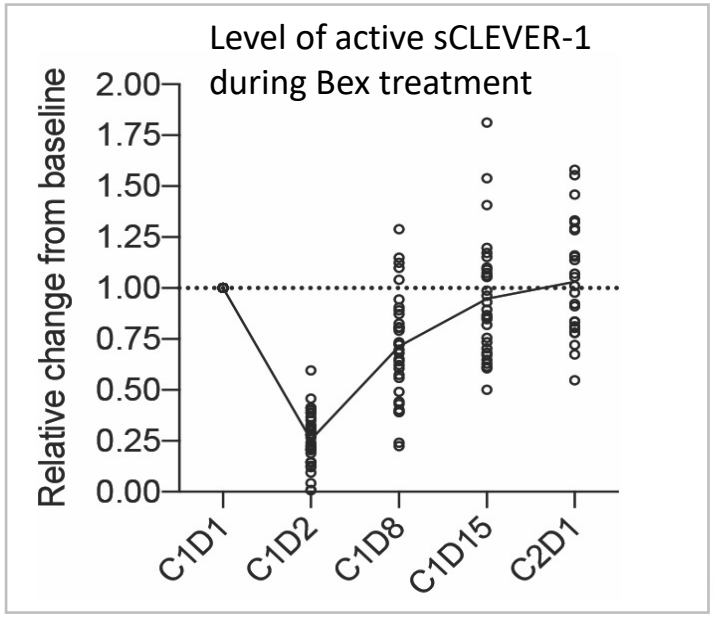
1) Viitala *et al*. (2019) Clin Cancer Res. 25(11) 2) Karikoski *et al*. (2014) Clin. Cancer Res. 20(24)

The New Unlock Agent to Regain Immunity; *bexmarilimab*

Tackling both immuno-suppressive macrophages and soluble protein that inactivates T-cells







- Advanced MATINS cancer patients have high levels of soluble CLEVER-1, which can directly inhibit T-cells and be a tool for cancer to create systemic immune suppression
- Targeting both macrophages and sCLEVER could unleash the power of the immune system, even in immune checkpoint inhibitor refractory patients

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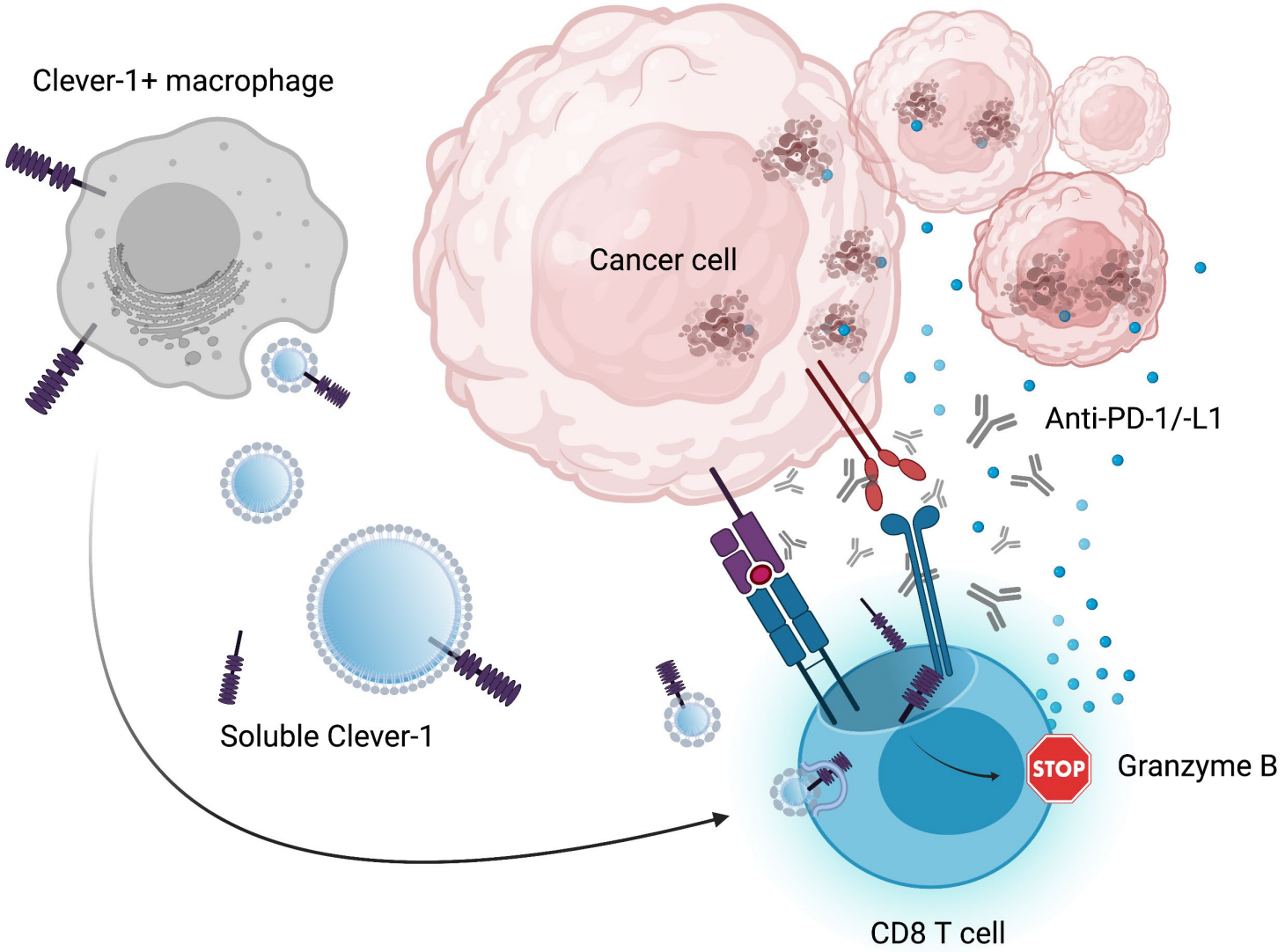
Soluble Clever-1: A Remote Controller of Cytotoxic T-cells

Inhibiting the immunological synapse blocks the benefit of CPI such as anti-PD-1(L1)



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MATINS; Phase I/II Trial Design and Timelines

Adaptive design enabling pivotal data generation

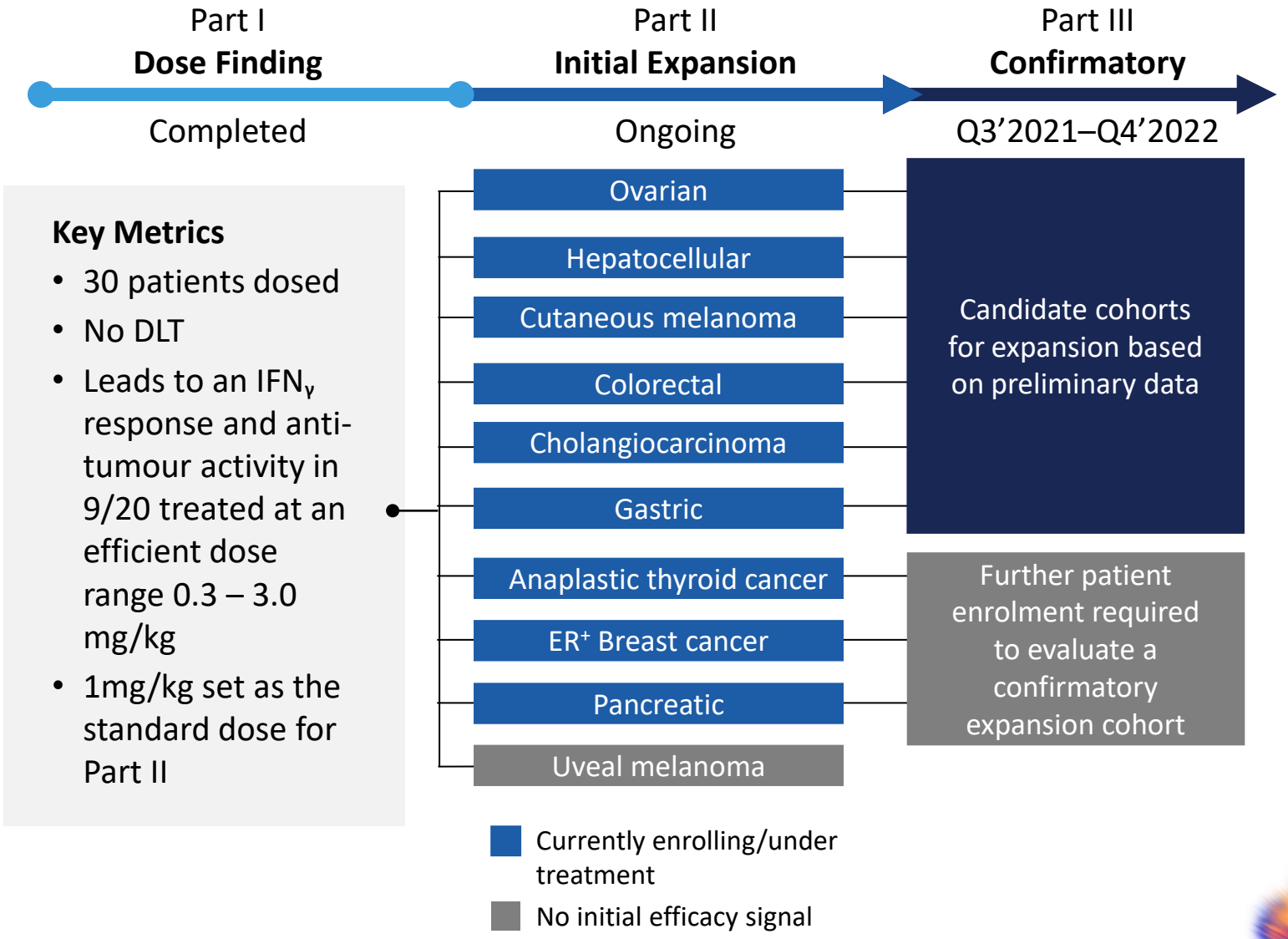
The adaptive design of the MATINS trial

1
Expand into promising indications quickly

2
Effectively optimise patient populations

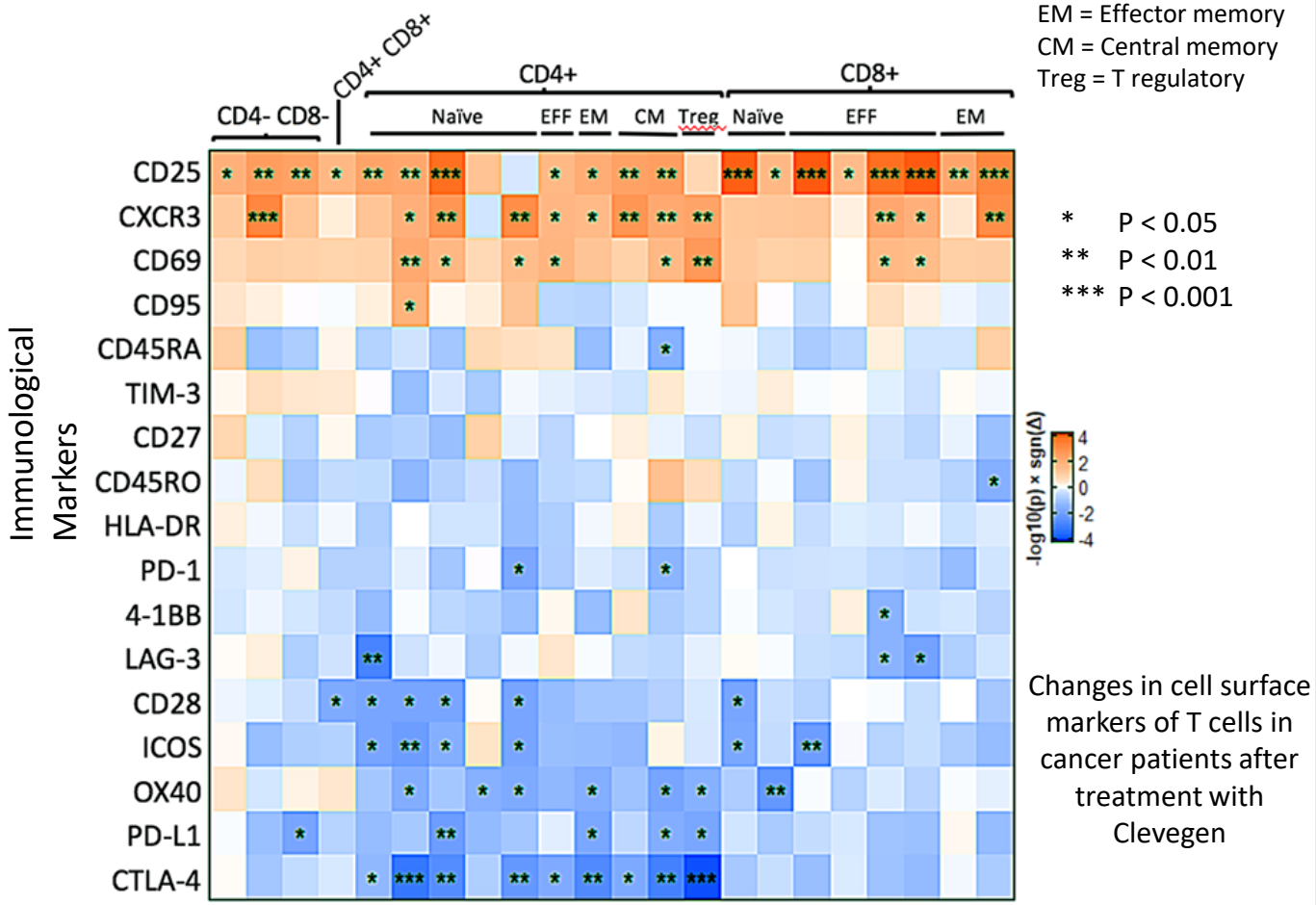
3
Efficiently move through lines of therapy

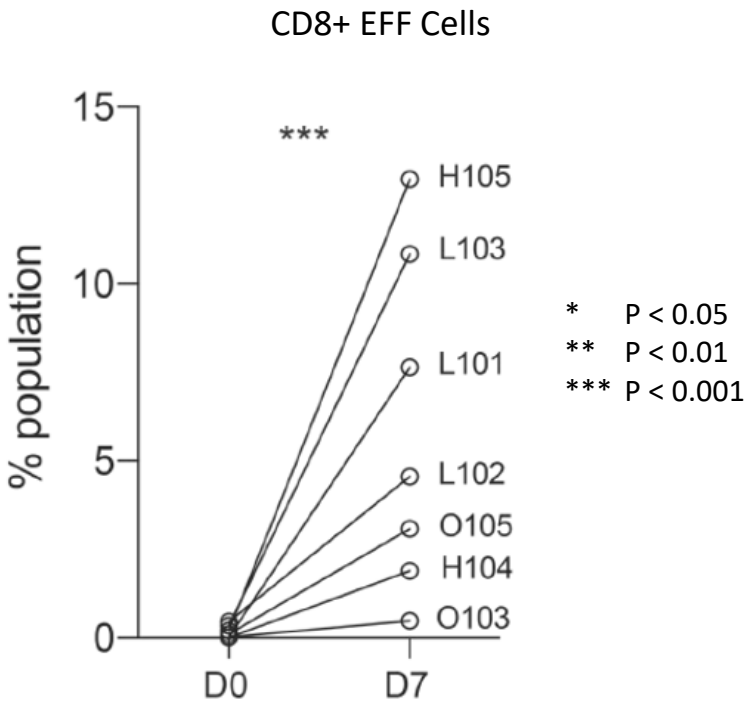
4
Conduct regular meetings with regulatory authorities for marketing approval



CLEVER-1; a Master Regulator of Immunity

Clevegen treatment downregulates all major checkpoints and increases CD8+ effector populations





Clevegen treatment significantly increases the amount CD8+ effector T cells

MATINS Phase I/II; 30-Patient Part I Safety Data Overview

Topline Safety Data

Treatment-related adverse events (n=66 events total)	**Grade 1-2** [n (%)]	**Grade 3-4** [n (%)]
All	19 (28,8%)	1 (1,5%)
Abdominal pain	2 (3,0%)	0
Alanine aminotransferase increased	3 (4,5%)	0
Aspartate aminotransferase increased	3 (4,5%)	0
Blood alkaline phosphatase increased	3 (4,5%)	0
Pyrexia	6 (9,1%)	0
Vomiting	2 (3,0%)	0
Infected seroma	0	1 (1,5%)

Highlights from Part I

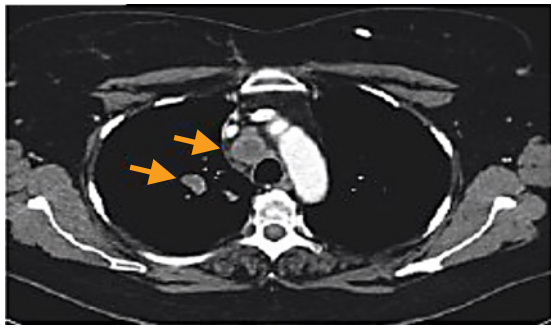
Dose-finding conducted in highly pretreated patients with advanced solid tumors that are refractory to ICIs:

- Clevegen determined to be safe and well tolerated
- 2 Partial Responses, 7 stable disease or mixed responses in target legions
- 36% disease control rate at efficient dose
- Clinical responses are statistically associated with an increase in peripheral natural killer cells and an increase in plasma interferon gamma levels

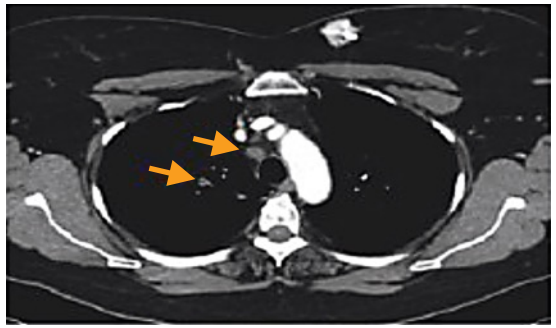
Single Agent Efficacy in Last-Line Checkpoint Refractory Patients (MATINS Part I patients)

MSI-negative CRC

Pre-dose

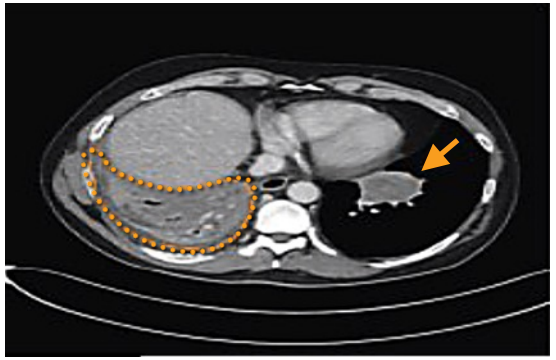


30 weeks

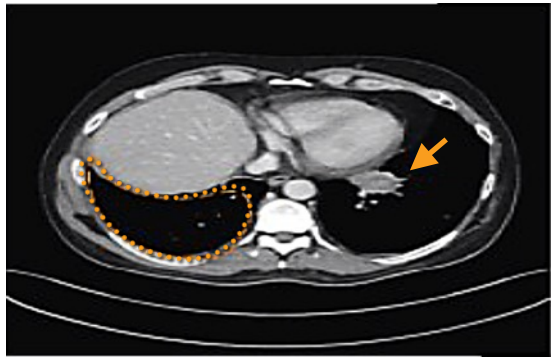


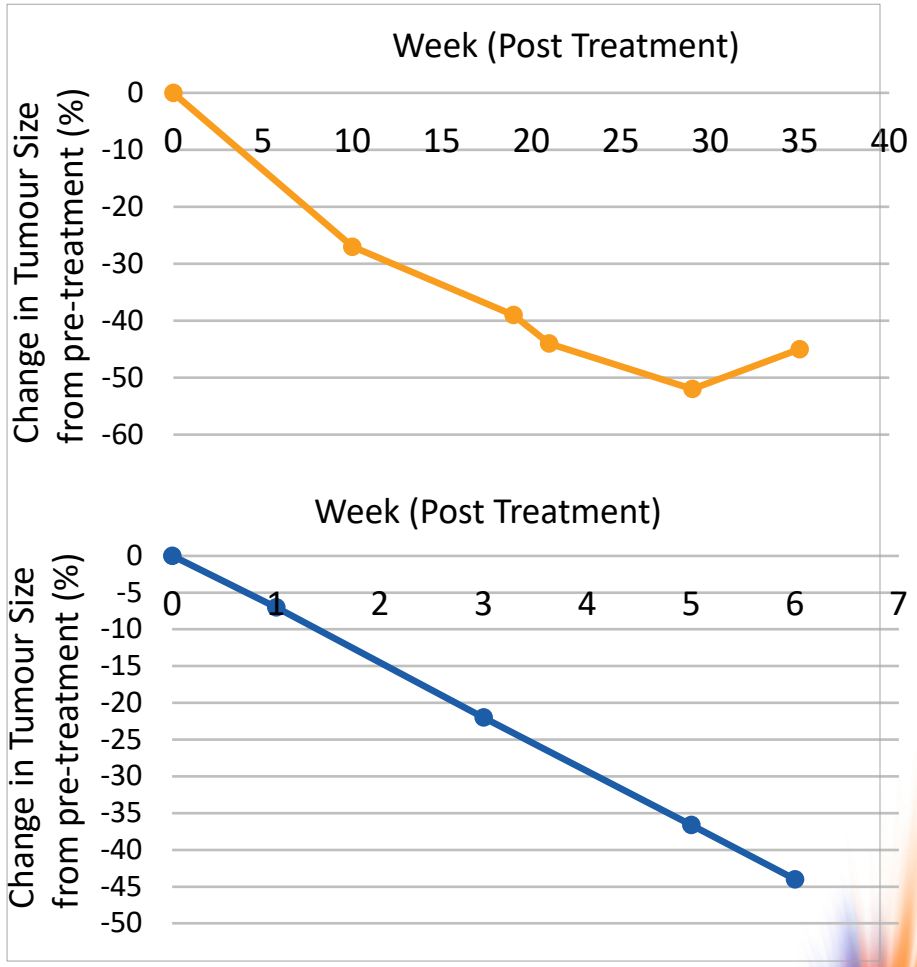
CPI refractory melanoma

Pre-dose



6 weeks





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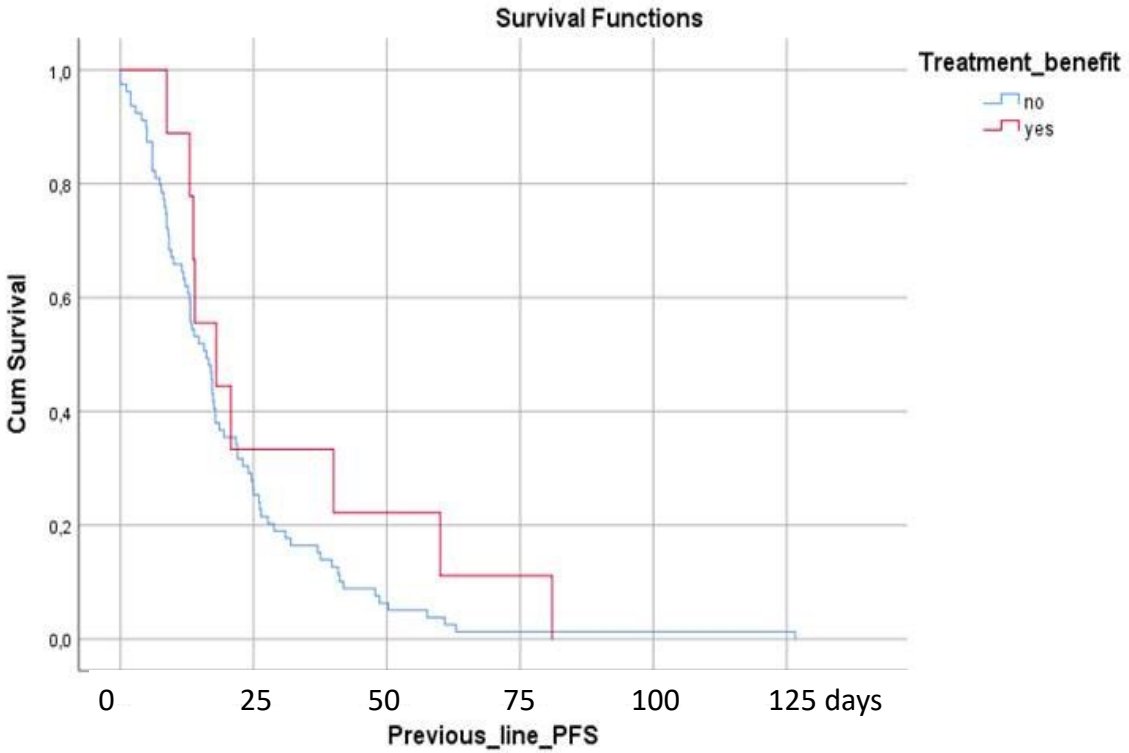
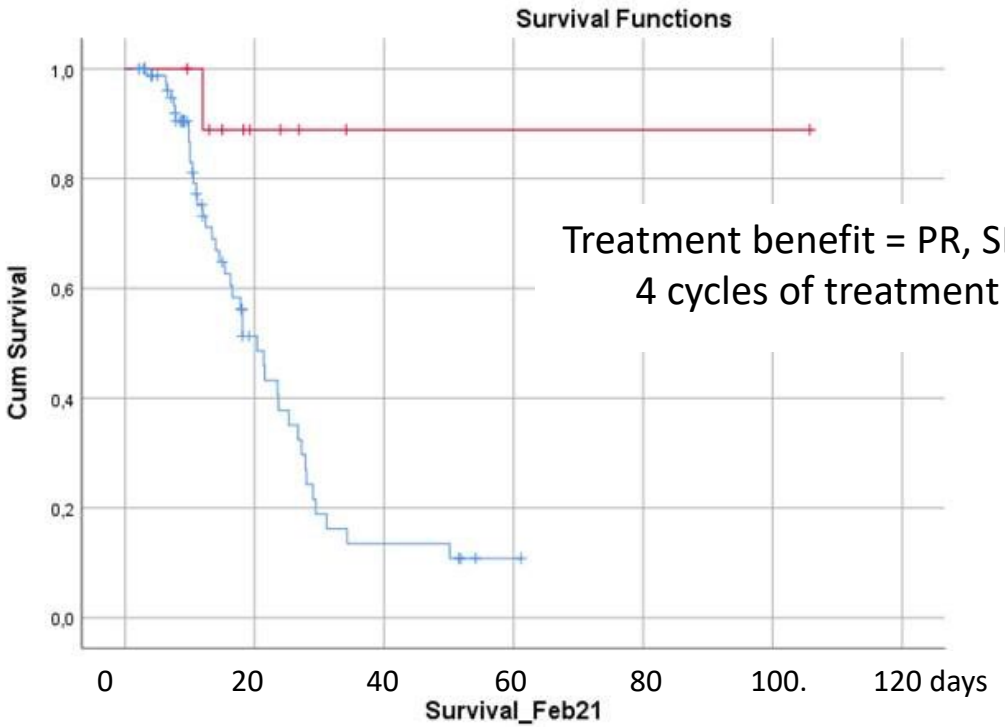
Up to date Survival Data from Part II of the MATINS trial

Significant survival benefit seen in patients that seem to be responsive to *bexmarilimab*

Progression-free survival (PFS) with previous treatment before entering MATINS



Overall survival (OS) of patients with *bexmarilimab* benefit



Treatment benefit = PR, SD and over 4 cycles of treatment (N 10**)**

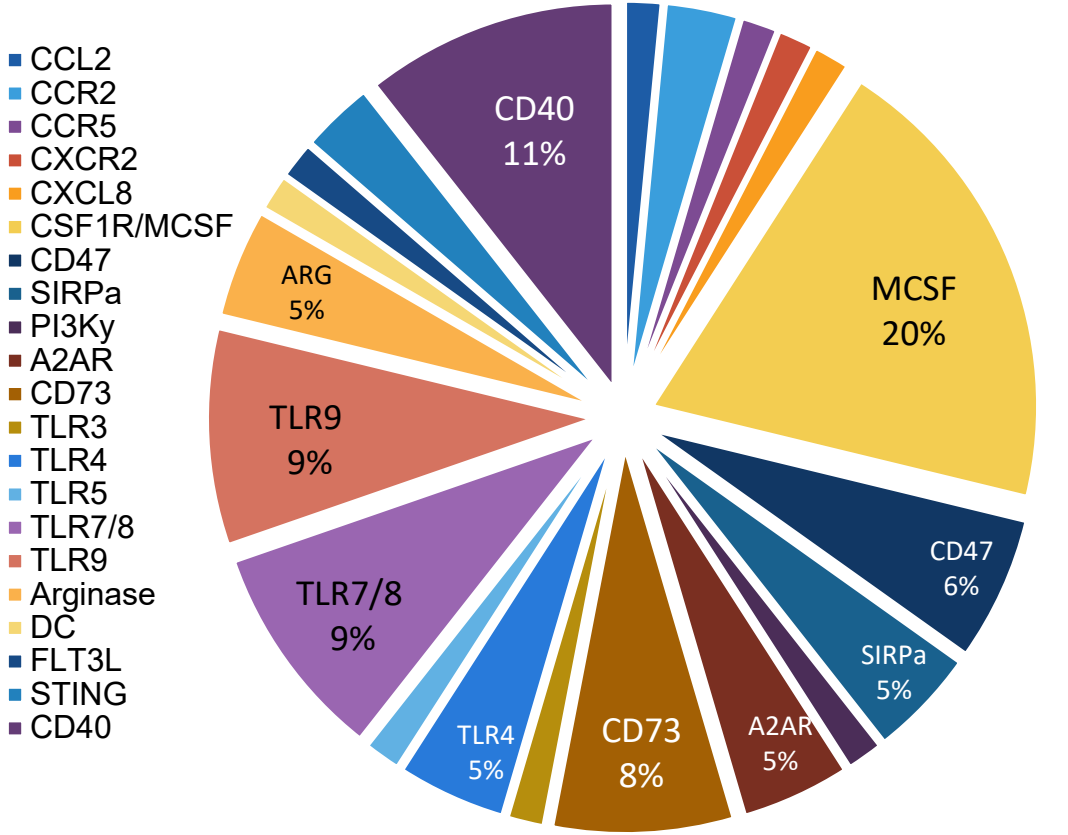
Response rate 14.9% (10/67) across 10 cancers without optimized dosing and patient enrichment

- This includes non-responding cohorts such as pancreatic cancer and uveal melanoma

Next Stages of Development

Targeting Myeloid Cells; Poorly understood area of IO[1]

Ongoing Myeloid Trials which are Modulating Myeloid Cell Function[2] (total=63)



CLEVER-1 is uniquely set to change paradigms

This is a first–in-class target with promising results have been achieved while investigating safety and tolerability.

Bexmarilimab is the first macrophage target that shows signs of single agent activity in checkpoint refractory settings. Furthering the established evidence that shows macrophages are key in treatment resistance[2].

Targeting tumor associated macrophages (TAMs) has shown promising preclinical results [1,3].To improve on current strategies, repolarisation of pro-tumoral macrophages has been pointed to as a better strategy[3]

Parameters we aim to optimize next:

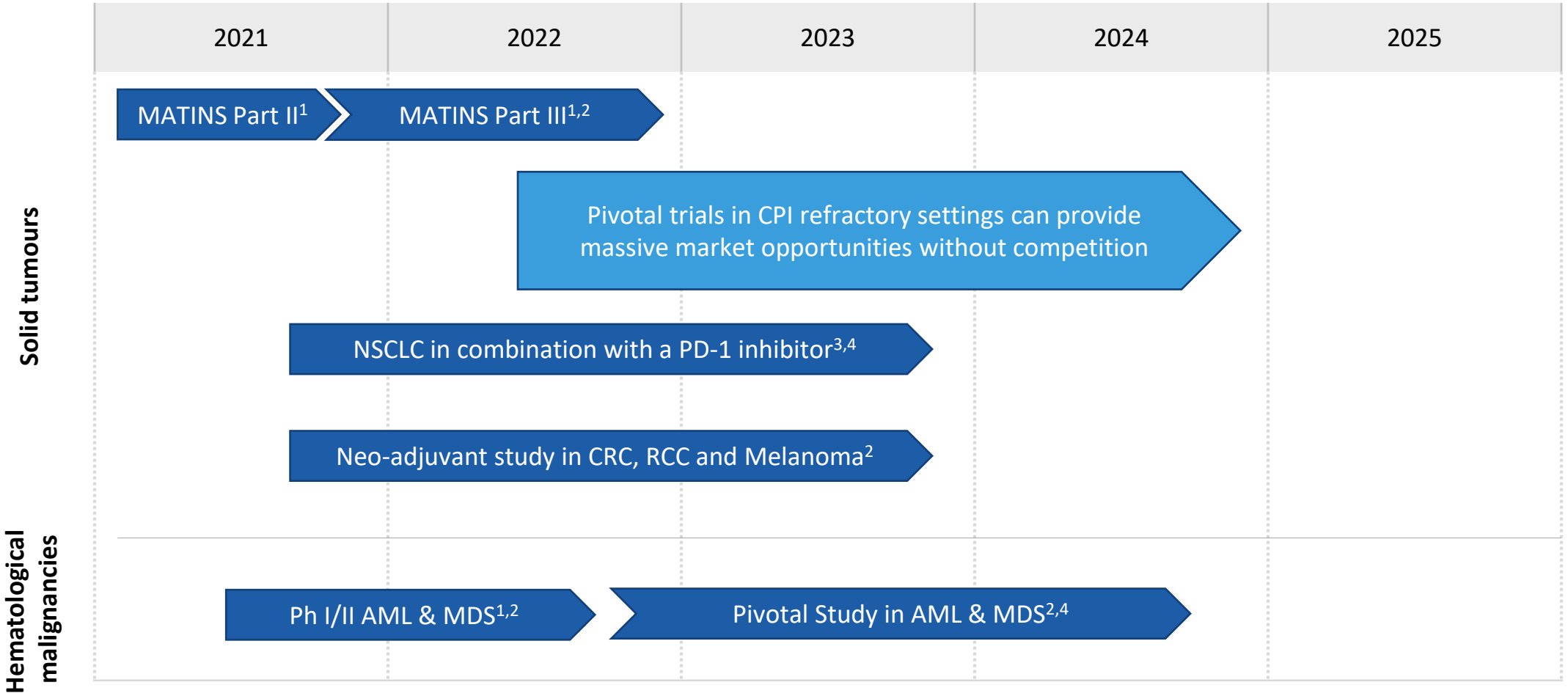
1. **Selection of indications/cancer types that show highest response;**
2. **Optimisation of dosing; both dose size and dosing frequency;**
3. **Starting the treatment earlier; move to earlier lines of treatment.**

1) Guerriero (2018) Trends in Molecular Medicine. 24 (5) 472-489
2) Jahchan et al., (2019) Front Immunology. 10:1611.
3) Cassetta and Pollard (2018) Nature Reviews: Drug Discovery 17(12) 887-904

Trial numbers and the status of said trials are correct at the date of the respective publication listed

Bexmarilimab Clinical Development Plan

Flexible trial designs enables multiple routes to market



1) Open label adaptive trials aiming to become pivotal studies
2) These trials are Faron's future plans and therefore subject to changes depending on multiple factors
3) Investigator sponsored study design
4) RCT in combination with first line standard of care (SOC)

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Clevegen: A Clever Antibody, with a Lot of Potential

CLEVEGEN
The immune switch

- First-in-class asset
- Rapid phenotypical change
- Promising preliminary safety and efficacy from Phase I/II study
- Suitable across multiple therapy areas
- A master immunology regulator

1. **A monotherapy which provides a permanent immune stimulation** in difficult-to-treat cancer types
2. **Positive safety data**; bringing benefits in comparison to currently available checkpoint inhibitors
3. **Opportunity to challenge treatment paradigms** in difficult-to-treat cancers with no option
4. **Large combination potential** with many standard of care agents and immune checkpoint inhibitors

HAEMATOKINE®
Hematopoietic
Stem Cell Expansion

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Targeting Amine Oxidase Copper Containing 3

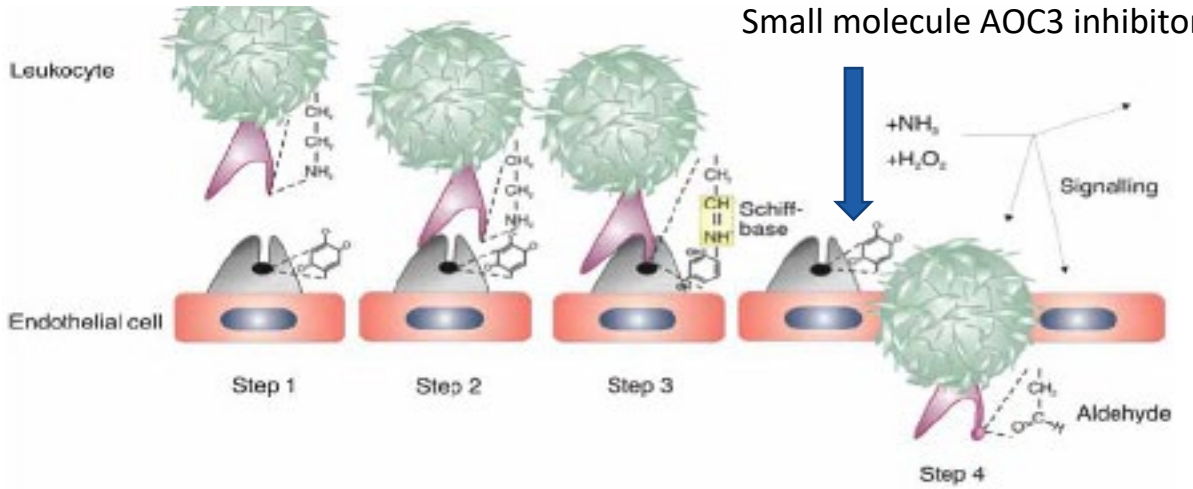
A different approach to a poorly utilised target

Why Target AOC3?

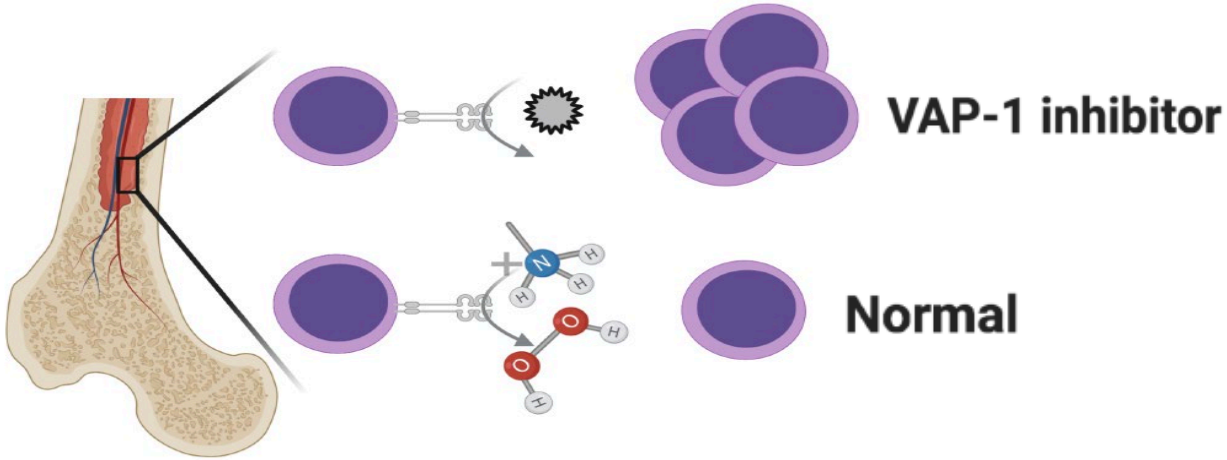
- Faron's founders pioneered the discovery of the AOC3 and have nearly 30 years of knowledge of the target[1]
- Faron is developing a proprietary method for use of AOC3 inhibition for the expansion of hematopoietic stem cells
- Targeting AOC3 historically has been aimed towards chronic inflammatory illnesses such as COPD, Asthma and GI
- The target has been well established in the scientific literature and multiple organisations have an approach inhibiting the target for the above indications

Amine oxidase, copper containing 3 (AOC3) is synonymous with VAP1 and SSAO

Tradition blocking adhesion approach



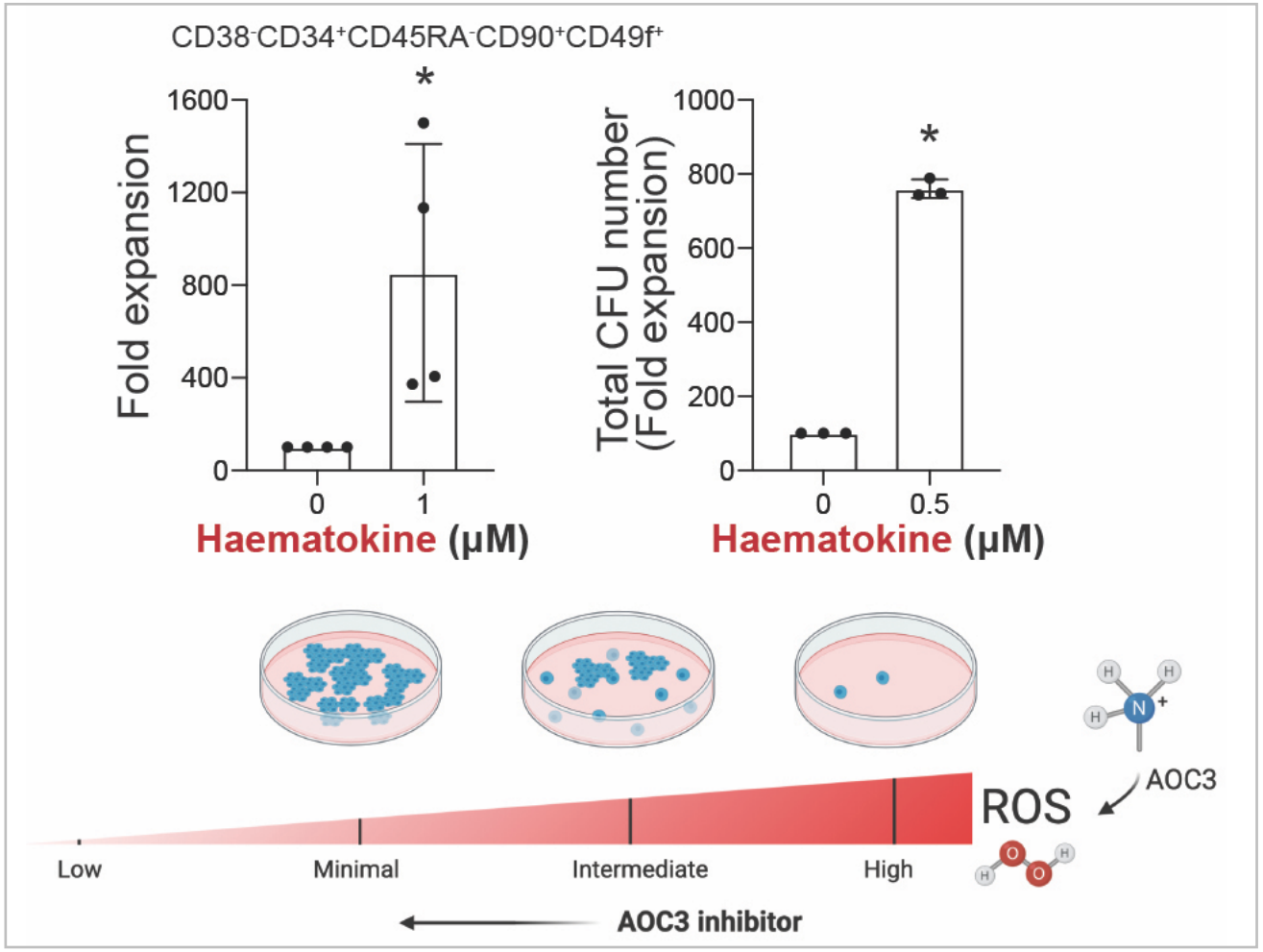
Faron's novel approach; utilising the enzyme



1. Salmi and Jalkanen (1992) Science. 257(5075) 1407-1409

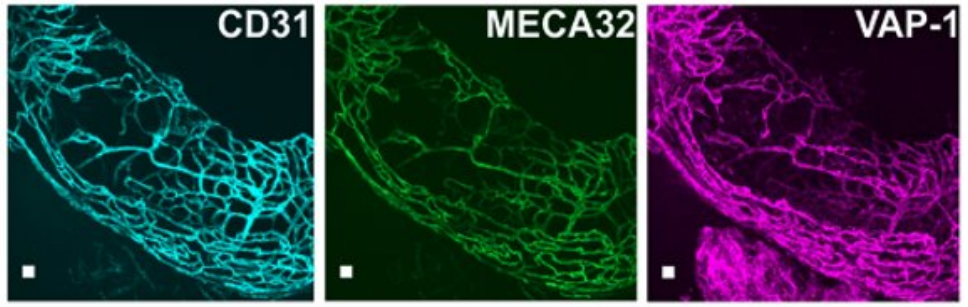
Expansion of Haematopoietic Stem Cell By Regulating AOC3 Activity

AOC3 is highly expressed on bone marrow vasculature



- Blocking enzymatic activity of AOC3 inhibits reactive oxygen species within haemopoietic stem cells leading to more HSC production *ex vivo* in human tissue and *in vivo* in mice after bone marrow depletion

- AOC3 inhibition's effectiveness in this situation has led us to believe it will be effective in a multitude of settings where the expansion of HSC is necessary
  - Ex vivo expansion for cell therapies
  - Enable grafting of an HSC transplant
  - **First effective treatment for graft failure**



Immunofluorescence staining of a mouse femur for VAP-1, MECA-32, and PECAM-1/CD31. VAP-1 is present in all vessels.

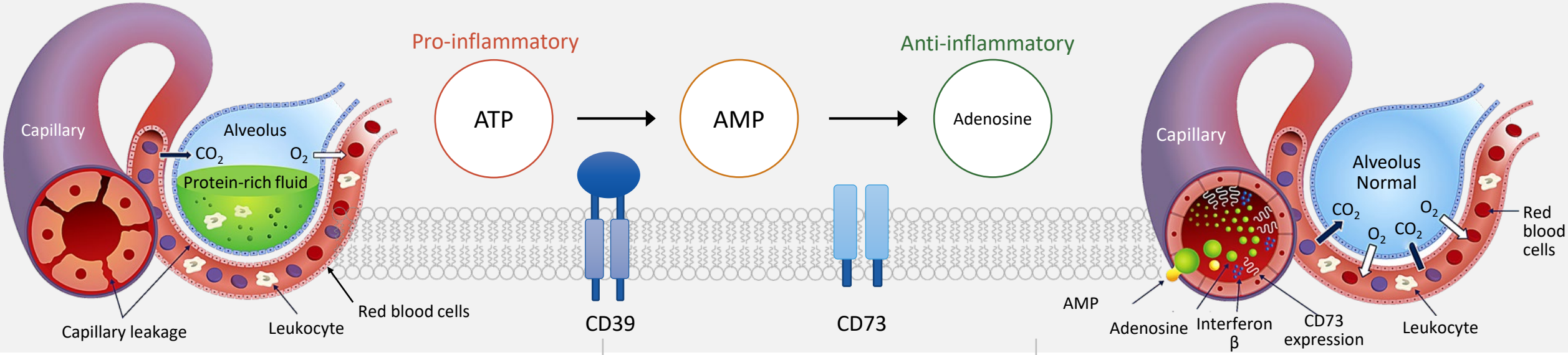
CD73 &
Organ Protection

TRAUMAKINE

FARON

Inducing CD73 for Organ Protection

An effective pharmacotherapy for producing anti-inflammatory, adenosine



Why induce CD73?

- ATP and ADP are fundamental and potent mediators of inflammation and thrombosis
- Adenosine is organ protective[1]
- Interferon is our natural vital response to inflammation especially in severe respiratory viral infections[2]
- Increased adenosine levels and adenosine signalling can decrease mortality[3]

In Organ Protection

- (+) Adenosine is a potent endogenous anti-inflammatory agent
- (+) CD73 is the rate limiting factor of the breakdown of AMP to Adenosine

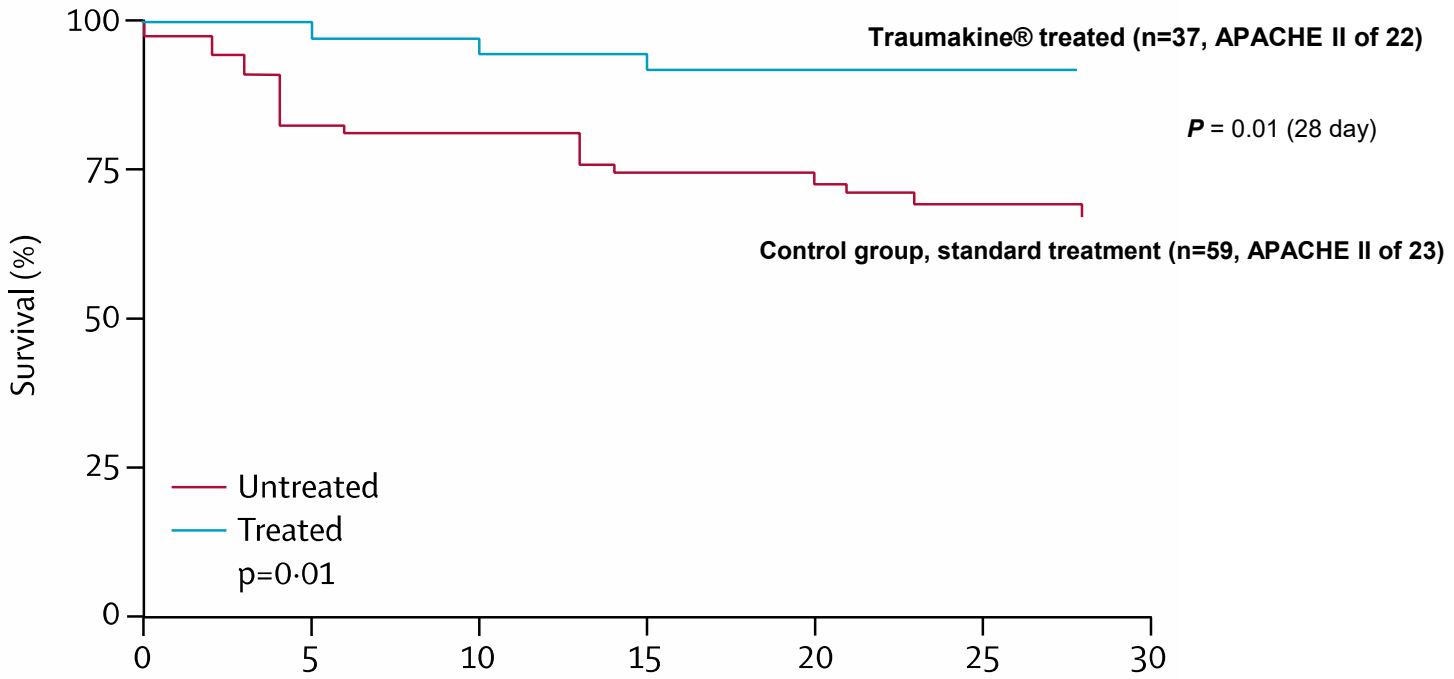
In Inflammation

- (–) Inhibition of Immune Activators
- (–) Decrease in IL-6, IL-8 and TNF-alpha
- (+) Activation of Immunosuppressants (TAMs, MDSC and Treg Cells)

1) Rajakumar et al. (2010) Transplantation. 90(12) 1260-1264
2) Blanco-Melo et al. (2020) Cell 181, 1036-1045
3) Hasko et al (2011) J Immunol 187:4256-4267

ARDS Phase I/II Proof of Concept Trial RESULTS: Significant Reduction in Mortality

Reduction in ICU stay from 28 to 16 days, less need for dialysis between groups



Carried out in 8 ICUs within the UK; of the 37 Patients treated with Traumakine: all were confirmed to have ARDS and of which 30% of the treated patients were diagnosed with sepsis and 41% with pneumonia

Primary Endpoint Achieved

- **Significant Drop in Mortality**[1]
- Traumakine showed 4x mortality benefit compared to placebo

No safety issues

- Short treatment period

Positive secondary endpoints

- Mortality at six months was lower than expected
- Improvement in lung function and functional assessments aligned with improvement in lung function and general dysfunction
- Efficacy improvements are consistent with a reduction in vascular leakage

1) Bellingan et al. (2014) *Lancet Res. Med*. 2: 98-106.

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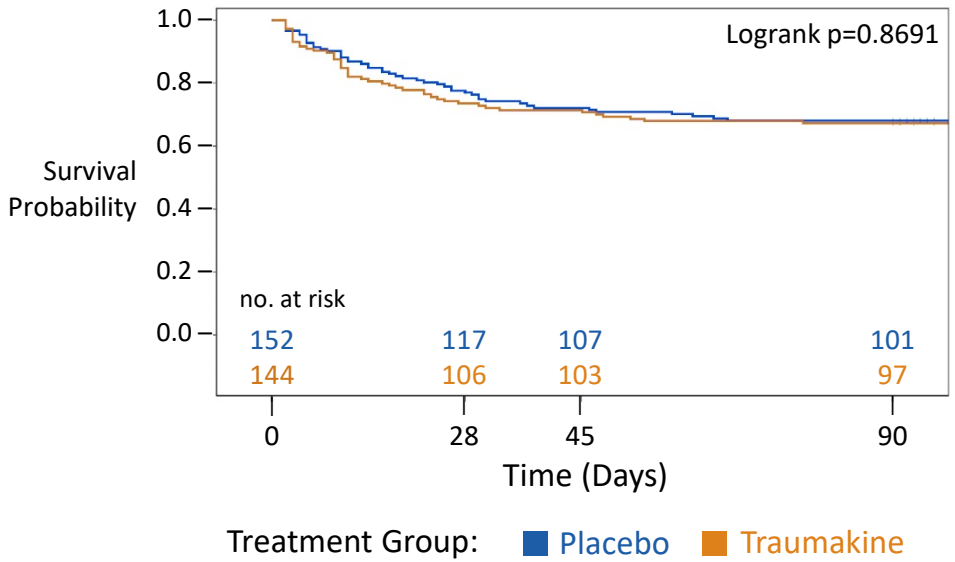
Phase III Trial (INTEREST Study) Key Learnings

Post hoc analysis shows that concomitant steroid use blocks Interferon beta-1a activity and increases mortality risk **by 7x**[4]

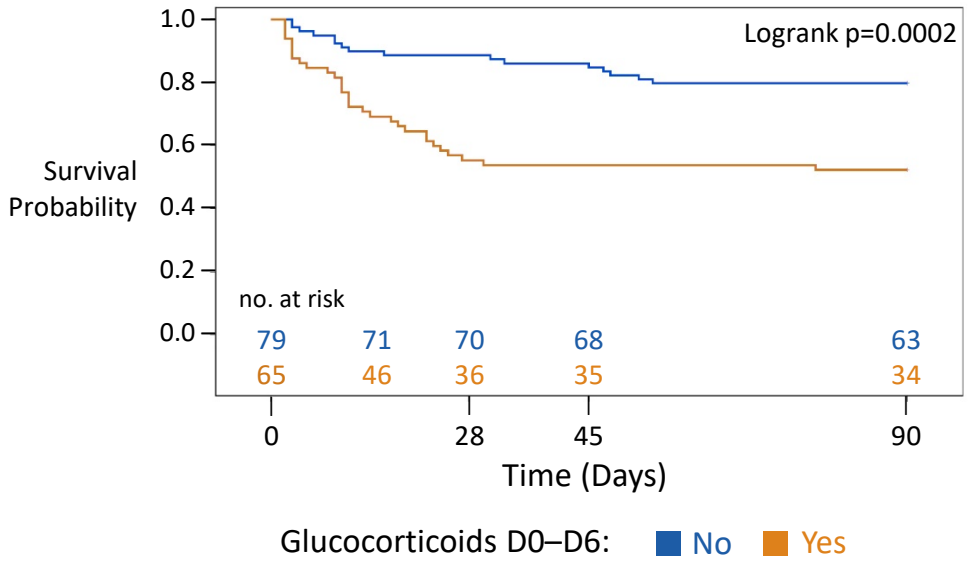
INTEREST Study Result [1,2]

Active vs Placebo
n=301 (296 dosed)



Post-Hoc Analysis of INTEREST [3,4]

IFN beta-1a Treatment Arm
n=144 (active arm)



Missed primary endpoint

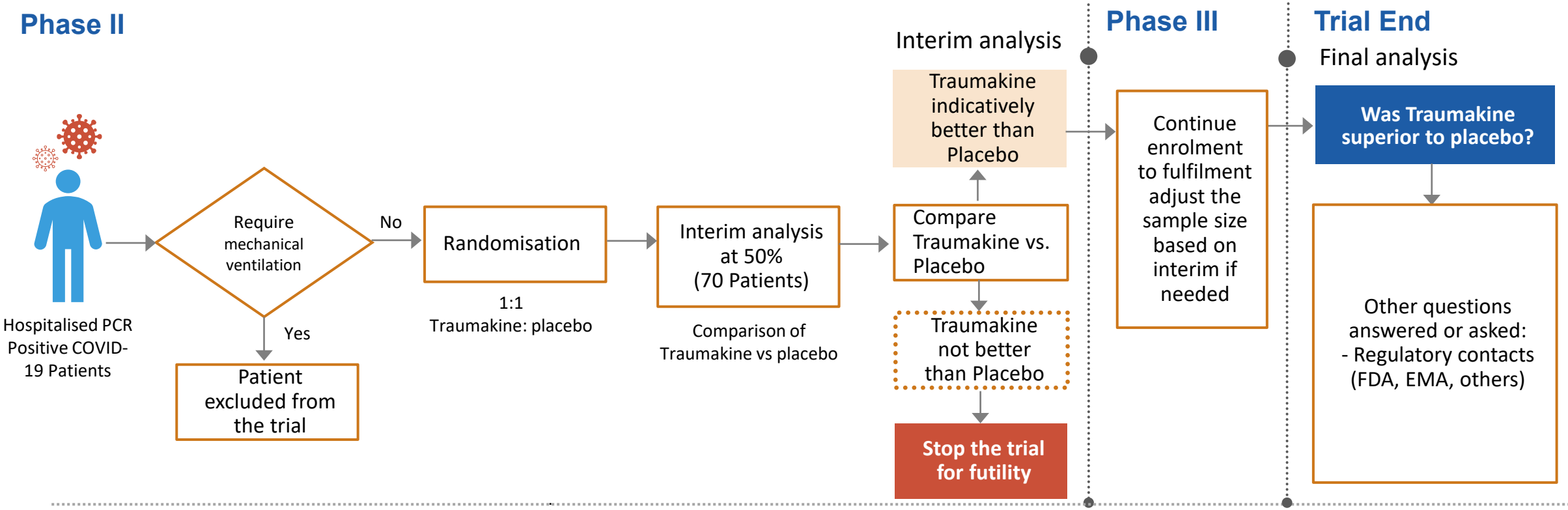
The FDA has granted a new adaptive Phase II/III clinical trial design for Traumakine in the treatment of ARDS based on post-hoc exploratory subgroup analysis determining highest mortality in patients on concomitant treatment of FP-1201 lyo and glucocorticosteroids

Steroids were widely used (60%) and blocked the effect of IFN beta

1) Clinicaltrials.gov. https://clinicaltrials.gov/ct2/show/NCT02622724; 2) Bellingan G, et al. (2017) Trials;18:536
3) Ranieri M, et al. (2020) JAMA; 323(8):725-733 4) Jalkanen J, et al (2020) ICM; https://doi.org/10.1007/s00134-020-06086-3

HIBISCUS: A Phase II/III Supported by the Department of Defense

Supporting hospitalised COVID-19 patients to prevent entrance to the ICU and/or death



Pivotal registration trial in the US:
An RCT, which is awaiting initiation, that aims to prove the superiority of Traumakine against placebo in hospitalised COVID-19 patients who do not require mechanical ventilation

- Primary end-point: Clinical status at D14
- Secondary end-points: Clinical status at D28, Mortality at D28 & D90**
- Size: 140 patients (Interim readout at 50% enrolment)**
- The use of corticosteroids at study entry is an exclusion criteria
- Enrolment estimates: 8-10 months. Final enrolment in Q4 2021**

*Trial protocol is still being developed and these schematic, endpoints and enrolment figures represent Faron's current expectations based on discussions with our Principal investigator, other investigators and Key Opinion Leaders. ** Represents Faron's current expectations

Traumakine – Novel Approach to Organ Protection

Now supported by US Department of Defense

ARDS is a major social and economic burden

>300,000
CASES IN THE U.S. & EU

3 Million
CASES WORLDWIDE[1]

There is no approved innovative drug for ARDS leaving the syndrome with no effective standard of care with mortality or ventilator free day benefits.

30%-40%
MORTALITY[2]

AVERAGE ARDS PATIENT[2]

Indications expected to benefit from Traumakine use

- Acute Respiratory Distress Syndrome (ARDS)
- Viral Pandemics (COVID-19, MERS, SARS, etc)
- Solid Organ Transplant
- Multi-Organ Failure
- Cytokine Storm and Release Syndrome
- Major Cardiovascular Surgery
- Stroke
- Acute Kidney Injury

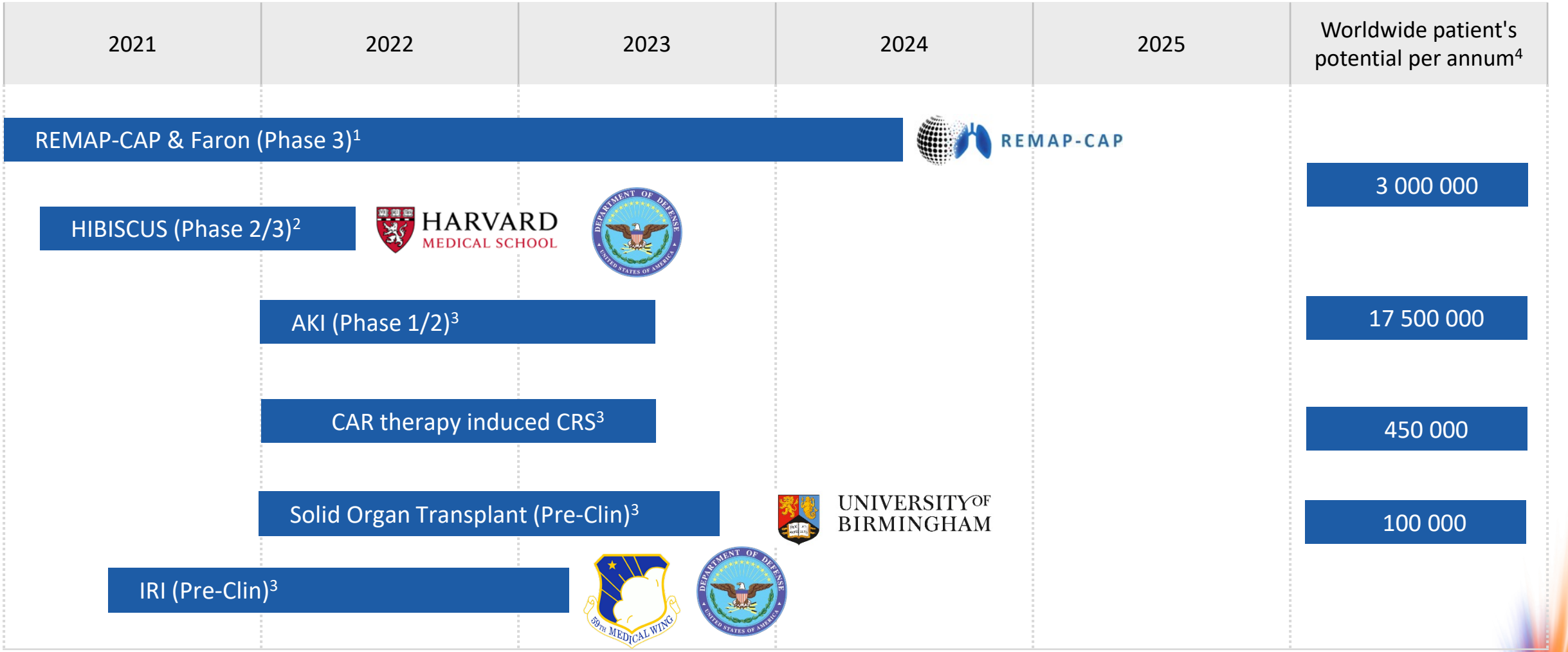
Regulatory achievements in ARDS

MHRA	FDA	EUROPEAN MEDICINES AGENCY
Promising Innovative Medicine	Fast-Track Designation	Orphan Drug Designation

1) Bellani et al. (2016) JAMA, 315(8):788-800 2) Cavalcanti et al. (2017) JAMA, 318(14) 1335-1345
3) Rubenfeld et al. (2005) NEJM, 353: 1685-1693 4) Herridge et al. (2011) NEJM; 364:1293-304

Traumakine; Route to Market

Simultaneous ongoing pivotal trials supported by medical agencies and academic networks



1) The REMAP-CAP Network continues to run their self named trial, is externally funded and therefore has minimal impact on Faron's cash flow.
2) HIBISCUS Study is currently in planning and subject to change. However, it is Faron's future plan to initiate this trial in Q1-2021.
3) These trials are Faron's future plans and therefore subject to changes depending on multiple factors
4) **Patient numbers are approximate:** ARDS from Bellani et al. (2016) JAMA, 315(8):788-800. Major CVD surgery Melly et al (2018) J Thorac Dis; 10(3): 1960-1967. Cases from CAR therapy Induced CRS from McKinsey. AKI from GlobalData Epidemiology. SOT from the WHO

FARON

2021 Set to be a Year of Significant Pipeline Progress

Bexmarilimab

- Top line data from MATINS trial Part II
- Determination of final dosage and frequency
- Selection of first pivotal cohort from MATINS
- Initiation of combination, neoadjuvant and haematological malignancies trials

Haematokine

- Ongoing pre-clinical studies with humanised AOC3 mice and with ex vivo human cells

Traumakine

- Initiation of HIBISCUS Phase II/III study
- Anticipated REMAP-CAP interim read out
- Formation of Scientific Advisory Board for clinical expansions
- Preclinical work on solid organ transplant


Thank you
FARON