SYNACT PHARMA

SynAct Pharma AB (publ)

Transforming the treatment of severe inflammatory diseases

Stockholm, October 2020

Forward-looking statements

The information in this presentation (the "Information") contains forward-looking statements related to Synact Pharma AB (the "Company"). All statements other than statements of historical facts included in the Information are forward-looking statements. Forward-looking statements give the Company's current expectations and projections relating to its financial condition, results of operations, plans, objectives, future performance and business. These statements may include, without limitation, any statements preceded by, followed by or including words such as "target," "believe," "expect," "aim," "intend," "may," "anticipate," "estimate," "plan," "project," "will," "can have," "likely," "should," "would," "could" and other words and terms of similar meaning or the negative thereof. Such forward-looking statements involve known and unknown risks, uncertainties and other important factors beyond the Company's control that could cause the Company's actual results, performance or achievements to be materially different from the expected results, performance or achievements expressed or implied by such forward-looking statements. Such forward-looking statements are based on numerous assumptions regarding the Company's present and future business strategies and the environment in which it will operate in the future.

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SynAct Pharma – Investment Highlights

- Focus on developing a novel treatment concept for inflammatory and autoimmune diseases by targeting the melanocortin system
- High unmet need in inflammatory diseases for therapeutics that can spare the use of immunomodulatory agents due to their poor side effect profile
- Development of lead asset, AP1189, is currently underway in three separate Phase II trials for various indications with inflammatory manifestations, including orphan diseases
- Several near-term value inflection points:
  - 2Q21 – Complete Phase IIa AP1189 data in rheumatoid arthritis
  - 2Q21 – Complete Phase IIa AP1189 data in COVID-19-induced ARDS
  - 2H21 – Complete Phase IIa AP1189 data in nephrotic syndrome
- Management with strong track record in clinical development as well as global business development and a supervisory board with decades of experience in research
- Continually evaluating compelling business development opportunities

Facts and Figures

Founded in 2013

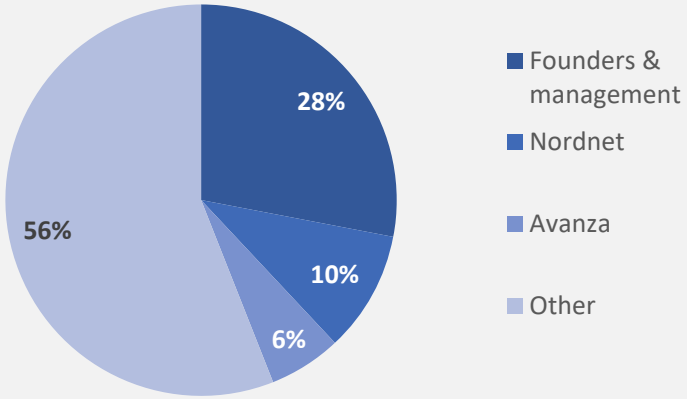
Listed on Spotlight exchange since 2016

Market cap: c. SEK 1.0bn / c. EUR 95m

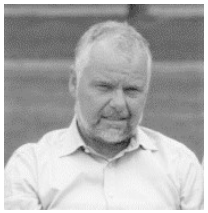
Ticker: (SYNACT:SS)

Cash balance: SEK 4.3m / EUR 410k (as of 30 June 2020), excluding SEK 32.4m / EUR 3.2m raised in July 2020

Ownership structure



SynAct Pharma – Experienced Management



Jeppe Øvli Øvlesen, MBA – CEO

- >20 years of CEO experience
- Founding Board Member of more than 10 biotech/medtech companies
- Co-founder of TXP Pharma
- Former CFO & VP BD of Action Pharma
- Holdings: 1,396,583

Torbjørn Bjerke, MD – Chairman

- >25 years track record from Pharma industry as Head R&D and CEO (private & public)
- Co-founder of Action Pharma, TXP Pharma and Arctic Aurora Life Science
- Member, BoD for DBV Technologies
- Holdings: 763,512

Thomas Jonassen, MD – Co-founder & CSO

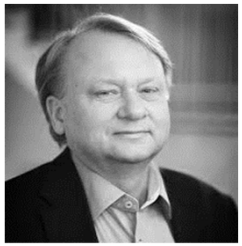
- Member, Board of Directors
- Associate Professor, KU in Denmark
- Visiting Professor, WHRI, UK
- Co-founder of TXP Pharma
- Co-founder and former CSO of Action Pharma
- Holdings: 2,236,971

John Haurum, MD – Board Member

- Former CEO of F-star (UK) with deal flow in excess of €200m
- Co Founder and former CSO of Symphogen
- Member of the board in a number of European biotech companies
- Holdings: 29,840

Henrik Stage, MsC – CFO

- >25 years experience from Biotech and financial industry
- Former CEO and CFO at Santaris Pharma sold to Roche for $450m
- >$150m from Big Pharma deals. Prepared Santaris for US Nasdaq IPO
- Holdings: 511,430



Terje Kelland, MD – Board Member

- Former Executive positions in Novo Nordisk, SOBI, and Pharmacia
- Former Vice VD of Karolinska Dev.
- Background as a professor at Lunds U.
- Holdings: 0

SynAct Pharma – Pipeline Overview

Asset	Indication	Preclinical	Phase I	Phase IIa	Phase IIb	Phase III	Next milestone
AP1189	Rheumatoid Arthritis						2Q21 final readout
	Nephrotic Syndrome						2H21 final readout
	COVID-19-induced acute respiratory distress syndrome						2Q21 final readout
	Psoriatic Arthritis*						
Pharmacology program							

* Phase II development to await data from RA study

Resolution in Inflammation – The New Frontier

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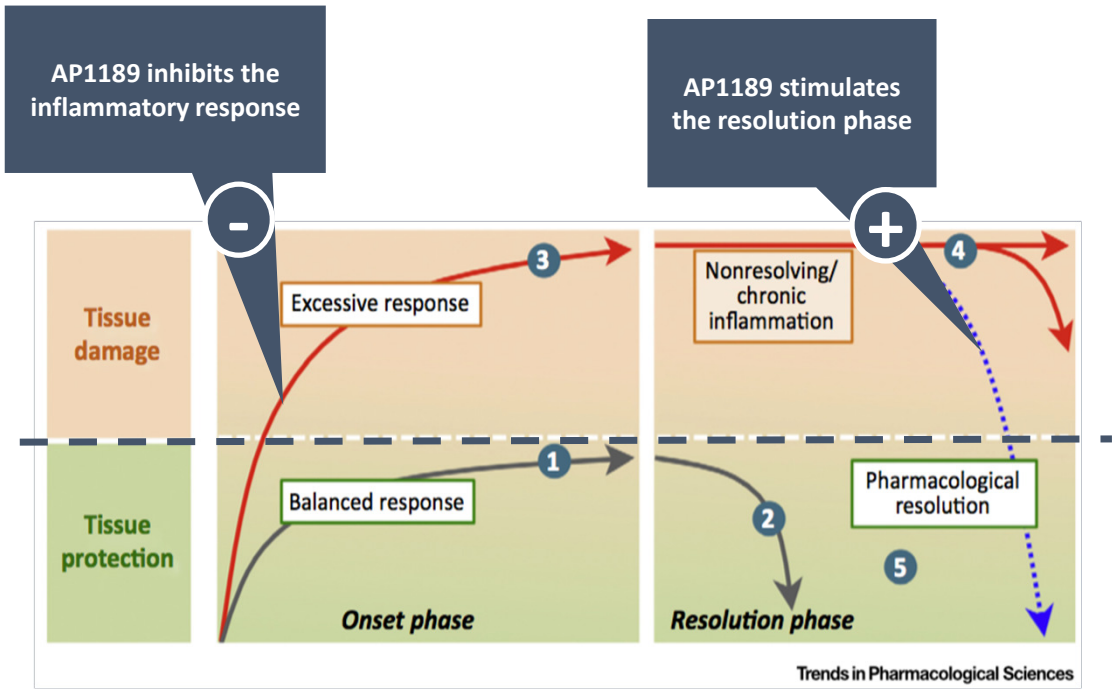

The Inflammatory Response

1. Inflammatory response effectively controlled in extent and time – protects tissues and limits damage
2. Pathways activated to safely terminate the inflammatory response and promote healing
3. Exaggerated response to inflammatory stimuli – can have detrimental consequences and harm tissues
4. Failure to achieve resolution of inflammation can result in chronic inflammatory state
5. Activation of endogenous resolution pathways have potential to restore tissues and function

Resolution therapy holds promise to correct overshooting/ongoing inflammation in many pathological settings

Compared to most current therapeutic approaches Resolution Therapy does not induce immuno-suppression

Lead compound AP1189 could be applied early in inflammatory diseases to restore normal functionality

The Melanocortin System and its Role in Inflammation

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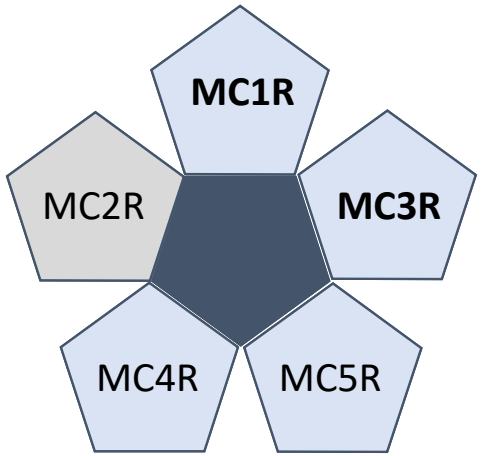
Melanocortin Receptor Family



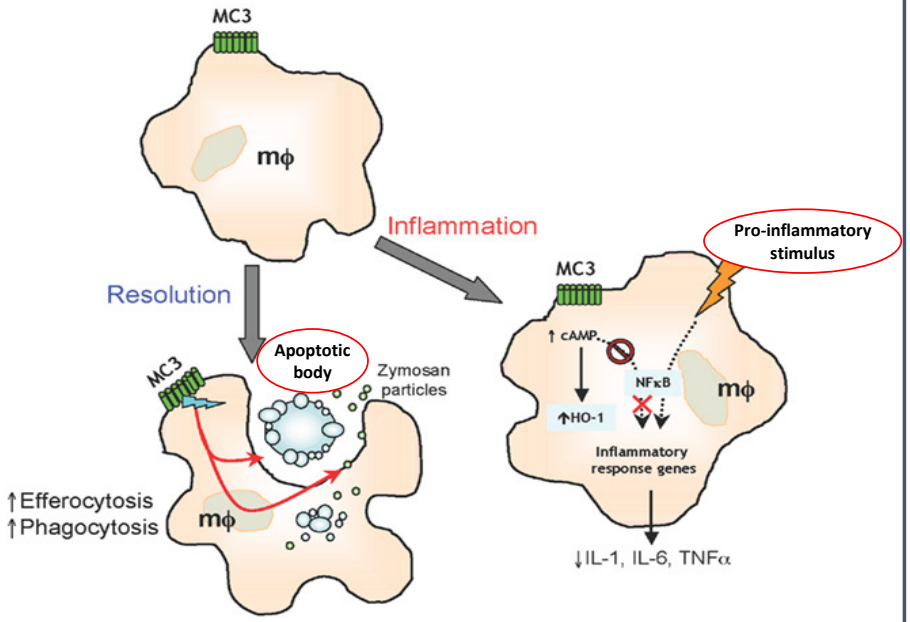
Steroidal dependent effects

Steroidal independent effects

Discovery of the family of 5 melanocortin receptors has provided nuance to the system's role in adrenal stimulation and steroidogenesis

MC1R and MC3R are believed to be the key receptors involved in immunomodulation

Melanocortin is activated in inflammatory diseases



Melanocortins are endogenous peptides released during inflammatory processes

Stimulation of MC1R and MC3R promote naturally occurring resolution processes to fight excessive inflammatory processes

AP1189 – First-in-class biased MCR agonist in Phase II development

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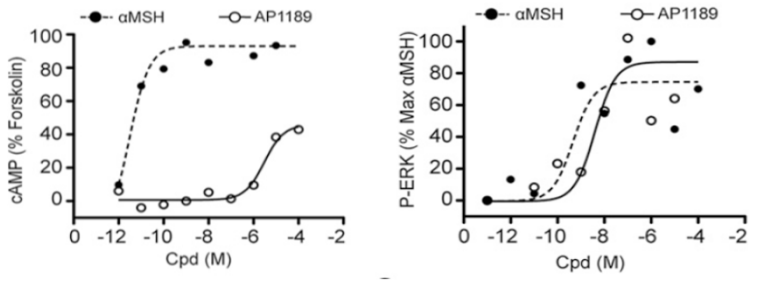
AP1189

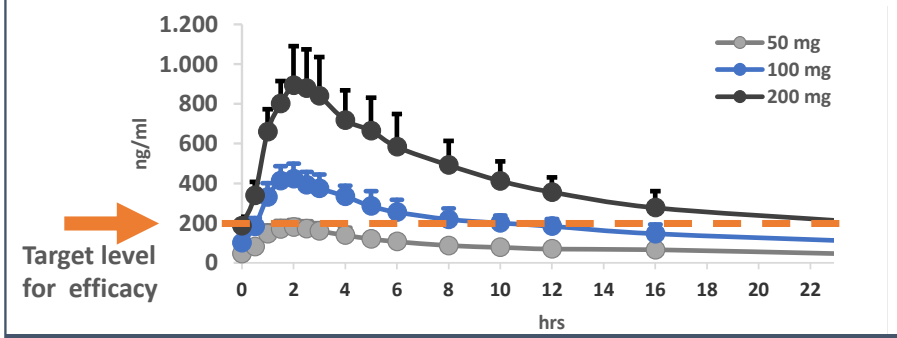
- First-in-class biased melanocortin receptor type 1 and 3 agonist
- The compound stimulates resolution of inflammation
- The compound has shown treatment effects in a number of experimental disease models, including:
  - *Inflammatory Joint diseases (Rheumatoid and Psoriatic Arthritis)*
  - *Inflammatory bowel diseases*
  - *Nephrotic syndrome*
- The compound is intended for once daily oral dosing
- Experience in clinical trials thus far has demonstrated a safe and well-tolerated side effect profile – differentiated from other melanocortin-derived therapies

MC1 receptor activation in transfected cells[1]



AP1189 selectively engages MC1R

PK profile following repeat dosing in man[2]



PK profile demonstrates once daily potential

1. Montero-Melendez et al: J Immunol. 194:3381-8, 2015; 2. Data from Completed Phase 1 study in healthy volunteers

Current melanocortin-derived therapy and AP1189's differentiation

Melanocortin-derived therapies address high unmet needs

- Current melanocortin-derived therapy is unspecific and associated with treatment limiting side effects
- Despite the unwanted side effect profile, the current melanocortin-derived therapy has shown efficacy where no other treatment regimens have
- Novel melanocortin-derived therapy, without treatment limiting side effects, could address a high unmet need in inflammatory and autoimmune diseases

Evidence of efficacy has been generated across many indications

Criteria for melanocortin-derived therapy	SynAct Pharma's AP1189	Acthar Gel & generics
Melanocortin specificity	Melanocortin receptors 1 & 3	None
Safety profile	Well-tolerated in clinical experience so far	Similar to corticosteroids
Potentially steroid sparing?	✓	✗
Presentation	Once a day oral	Intramuscular injection
Source / underlying material	Small molecule	Porcine pituitary extract
Positioning	Potential flexibility in the treatment paradigm	Late-stage patients with severe disease

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AP1189 - Phase II Development in Rheumatoid Arthritis

Positioning as add-on Therapy in Active Joint Disease

Rheumatoid Arthritis

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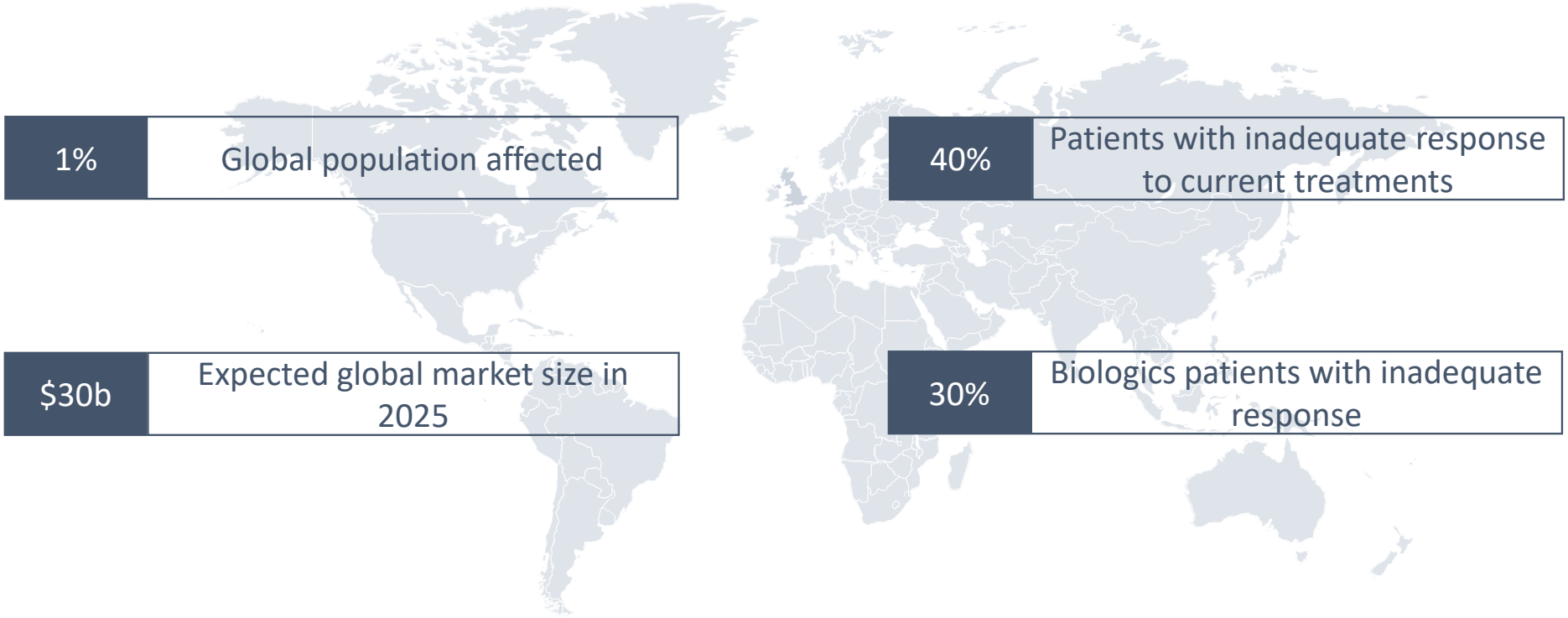
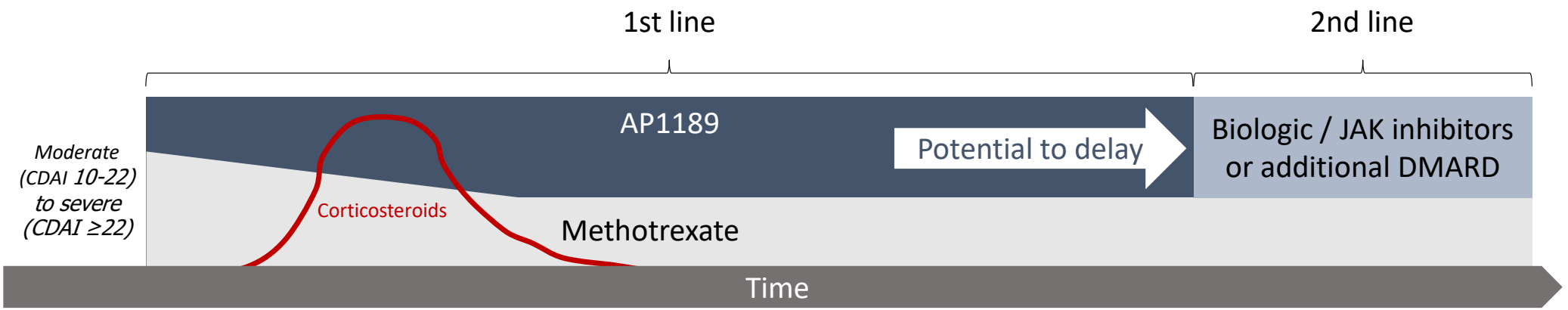


1%	Global population affected
$30b	Expected global market size in 2025
40%	Patients with inadequate response to current treatments
30%	Biologics patients with inadequate response

AP1189 has potential to attenuate RA symptoms , decrease time to resolution, and reduce need for corticosteroid treatment

AP1189's fit in the Rheumatoid Arthritis Treatment Paradigm



Current guidelines recomments to go to 2nd line treatment if methotrexate treatment shows lack lack of effecacy following 6 month treatment

AP1189 promotes inflammatory resolution and has no immunosuppressive effects, making it more suitable for 1st line treatment and providing more pharmacoeconomic benefit compared to biologics and/or JAK inhibitors

Adapted from EULAR 2019 recommendations: Ann Rheum Dis 2020;79:685–699

AP1189 Demonstrates Efficacy in Arthritis Models

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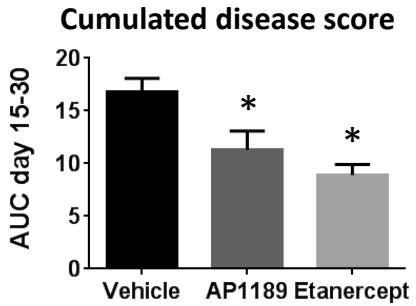
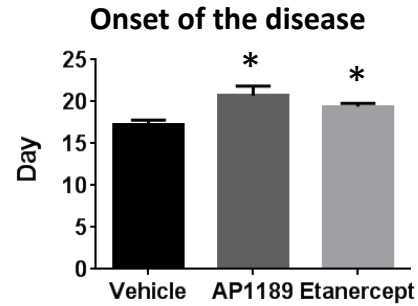
Collagen-induced arthritis

(30mg/kg, p.o.)



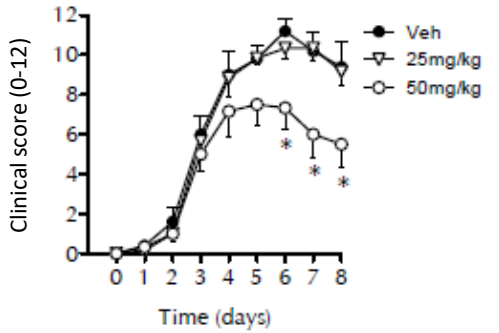


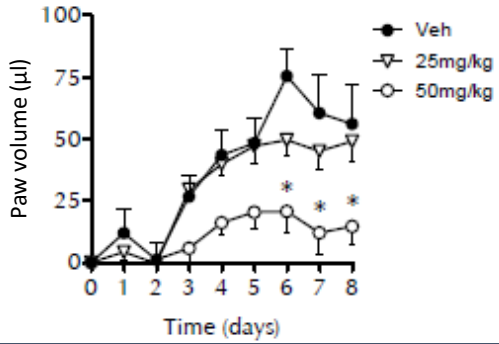
*Unpublished data: n=10, *: p < 0.05 vs Vehicle*

AP1189 provided a significant reduction in disease score as well as delay in onset

K/BxN-induced arthritis

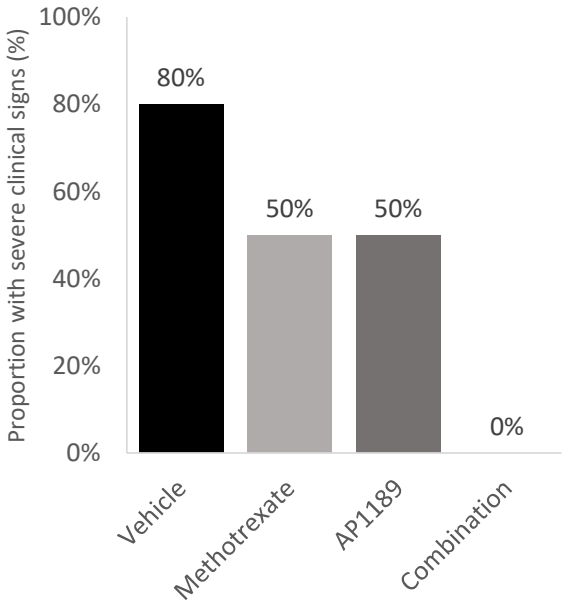
(25/50mg/kg, i.p.)





AP1189 provided a dose-dependent result in K/BxN-induced arthritis

Combination Treatment with Methotrexate



n=6 per group

Incidence of severe clinical signs in K/BxN induced arthritis

Montero-Melendez et al: J Immunol. 194:3381-8, 2015, PCT/EP2019/066578

AP1189 – Phase IIa Trial in Rheumatoid Arthritis

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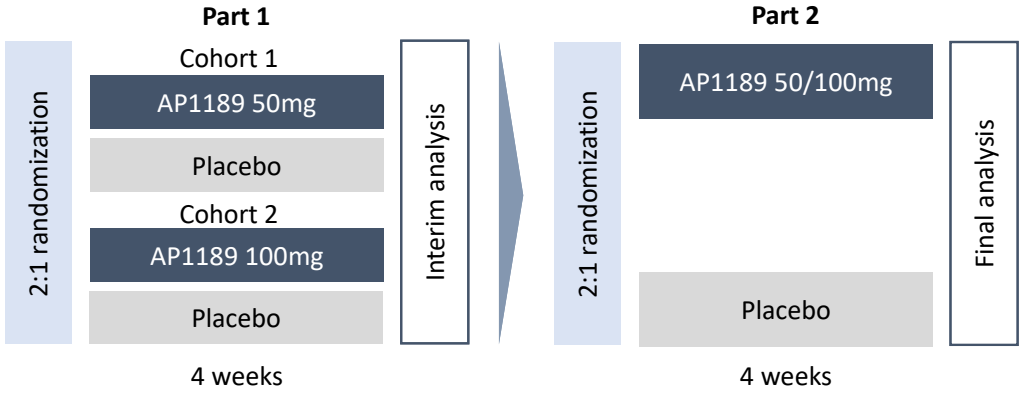
Phase IIa trial design



Key inclusion criteria:

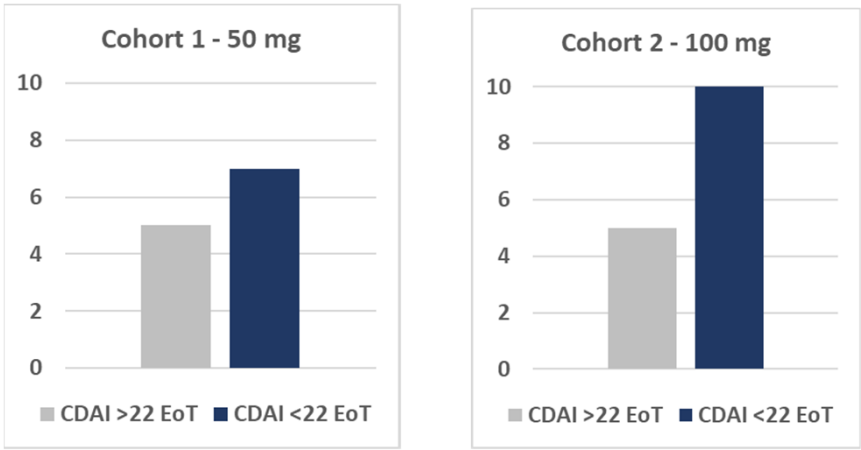
- Severe RA (CDAI score >22)
- Treatment naive
- Candidate for and planning to start methotrexate treatment
- Eligible for sites in Denmark, Sweden, and Norway

Primary endpoint: Reduction in CDAI below 22 relative to placebo

Secondary endpoints:

- Swollen and tender joints
- CDAI score
- DAS28 score
- HAQ-DI score
- FACIT-Fatigue questionnaire
- ACR response

Blinded observations reported to date



- Pts grouped on CDAI at End of Treatment (EoT).
- Analysis on blinded and non-validated data from ongoing study
- Cohort 1 data from 12 pts reported in press release May 5, 2020;
- Cohort 2 data from 15 pts reported in press release October 12, 2020

Key Takeaways

- Both cohorts show a group of patients with notable declines in CDAI score from baseline
  - Cohort 1: 7 out of 12 pts
  - Cohort 2: 10 out of 15 pts
- Blinded safety review (on non-validated data) suggests that AP1189 is safe and well-tolerated

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AP1189 – Phase II Development in Nephrotic Syndrome

Positioning as alternative to immuno-suppressive Therapy

Nephrotic Syndrome

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c.150k	Prevalence across US and EU
Limited	Treatment options for patients
c. 1/3	Patients with inadequate response to current treatments

Unmet Need

- Limited clinical guidelines exist for the proper management of Nephrotic Syndrome
- Management consists of therapeutic treatment with diuretics, ACE inhibitors, anti-infectives, lipid-lowering agents, and immunosuppressants
- High risk of developing Chronic Kidney Disease
- Impacts pediatrics as well as adults
- Potential for Orphan Drug Designation

AP1189 directly stimulates melanocortin receptors on podocytes, which are associated with restoration of full kidney functionality

MC1R-mediated pathways activated in Nephrotic Syndrome

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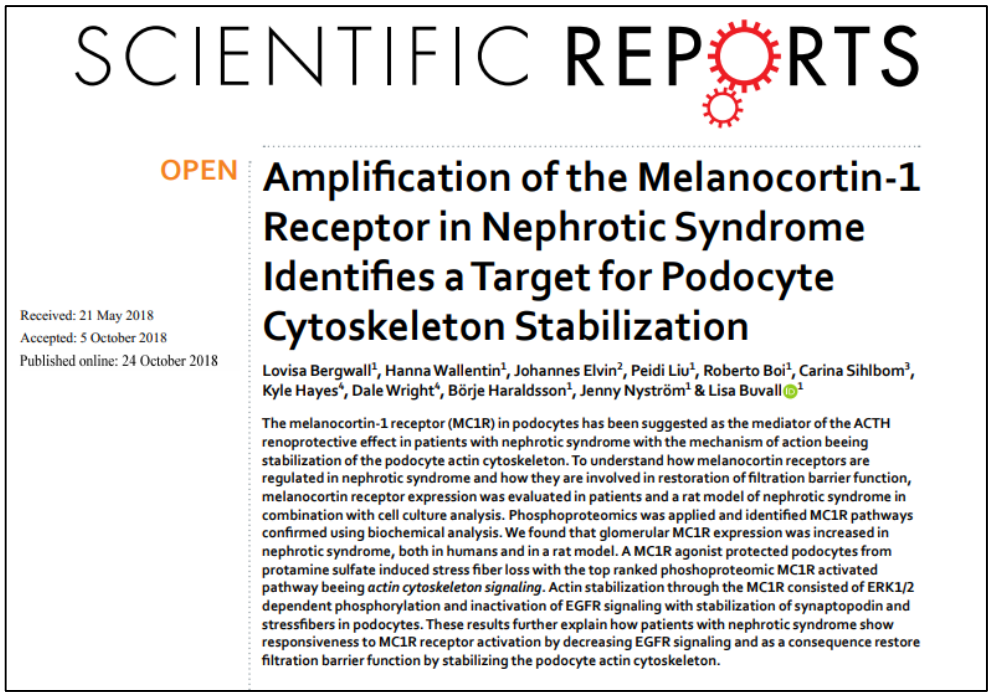


SCIENTIFIC REPORTS

OPEN

Amplification of the Melanocortin-1 Receptor in Nephrotic Syndrome Identifies a Target for Podocyte Cytoskeleton Stabilization

Received: 21 May 2018
Accepted: 5 October 2018
Published online: 24 October 2018

Lovisa Bergwall[1], Hanna Wallentin[1], Johannes Elvin[2], Peidi Liu[1], Roberto Boi[1], Carina Sihlbom[3], Kyle Hayes[4], Dale Wright[4], Börje Haraldsson[1], Jenny Nyström[1] & Lisa Buvall[1]

The melanocortin-1 receptor (MC1R) in podocytes has been suggested as the mediator of the ACTH renoprotective effect in patients with nephrotic syndrome with the mechanism of action beeing stabilization of the podocyte actin cytoskeleton. To understand how melanocortin receptors are regulated in nephrotic syndrome and how they are involved in restoration of filtration barrier function, melanocortin receptor expression was evaluated in patients and a rat model of nephrotic syndrome in combination with cell culture analysis. Phosphoproteomics was applied and identified MC1R pathways confirmed using biochemical analysis. We found that glomerular MC1R expression was increased in nephrotic syndrome, both in humans and in a rat model. A MC1R agonist protected podocytes from protamine sulfate induced stress fiber loss with the top ranked phoshoproteomic MC1R activated pathway beeing *actin cytoskeleton signaling*. Actin stabilization through the MC1R consisted of ERK1/2 dependent phosphorylation and inactivation of EGFR signaling with stabilization of synaptopodin and stressfibers in podocytes. These results further explain how patients with nephrotic syndrome show responsiveness to MC1R receptor activation by decreasing EGFR signaling and as a consequence restore filtration barrier function by stabilizing the podocyte actin cytoskeleton.

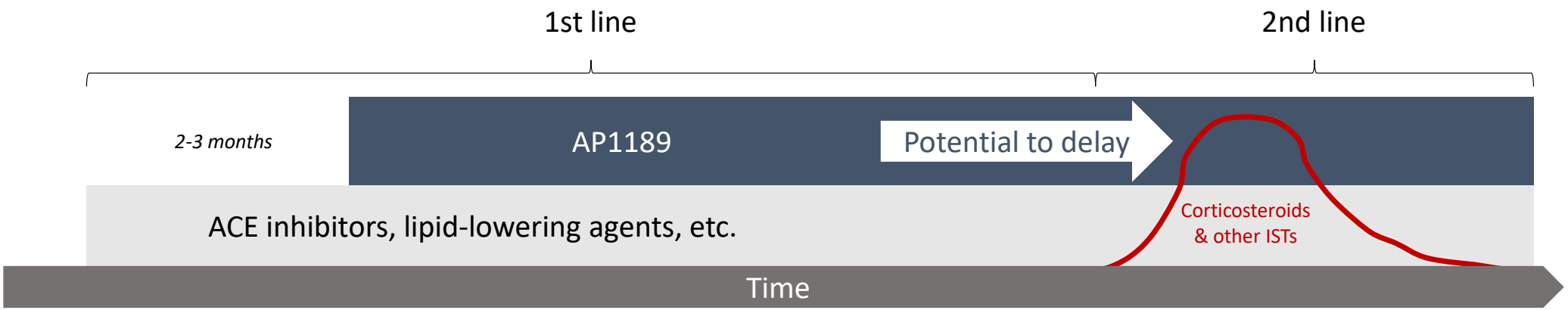
MC1R & Nephrotic Syndrome

- MC1R expression is increased in nephrotic syndrome, both in rat models and in humans
- MC1R agonism induced protection of podocytes from stress fiber loss
- Actin stabilization through the MC1R consisted of ERK1/2 dependent phosphorylation and inactivation of EGFR signaling with stabilization of synaptopodin and stress fibers in podocytes
- Patients with nephrotic syndrome show responsiveness to MC1R receptor activation by decreasing EGFR signaling (though ERK dependent Phosphorylation) and as a consequence restore filtration barrier function by stabilizing the podocyte actin cytoskeleton

AP1189 induces its pharmacological effects though MC1R and MC3R-mediated ERK dependent phosphorylation

Sci Rep. 2018 Oct 24;8(1):15731

AP1189's fit in the Nephrotic Syndrome Treatment Paradigm



Current clinical practice is to go to 2nd line treatment if continued proteinuria is present following 6 months of ACE inhibitor treatment

AP1189 promotes inflammatory resolution and has no immunosuppressive effects, making it more suitable for 1st line treatment and providing more pharmacoeconomic benefit compared to corticosteroids and other immunosuppressive therapies

AP1189 Shows Benefits in Models of Nephritis

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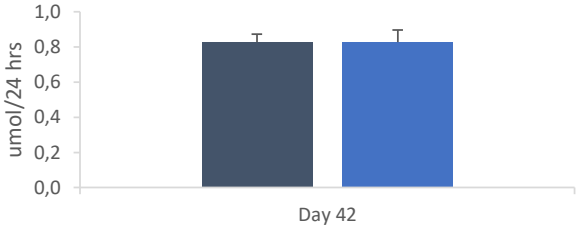
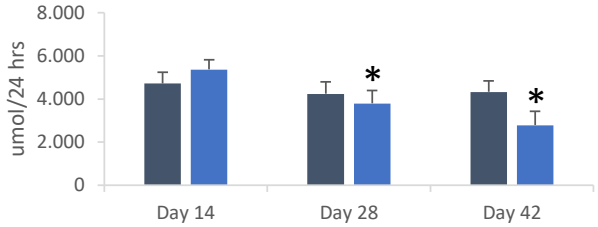
Membranous nephropathy rat model (passive Heymann Nephritis)

Vehicle (n=12)
AP1189 (n=12)

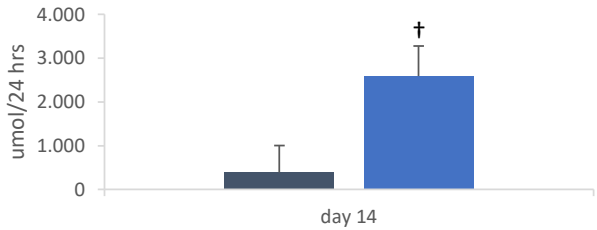
Creatinine Clearance (ml/min/g KW)



Albumin excretion rate



Reduction in AER (day 14-42)

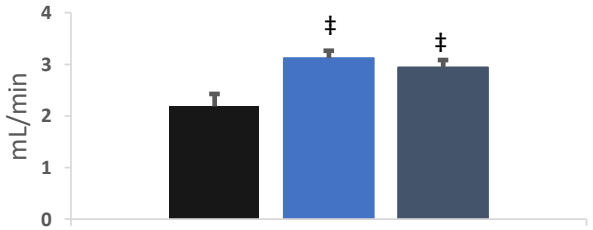


AP1189 showed no effect on creatinine clearance where albumin excretion decreased significantly

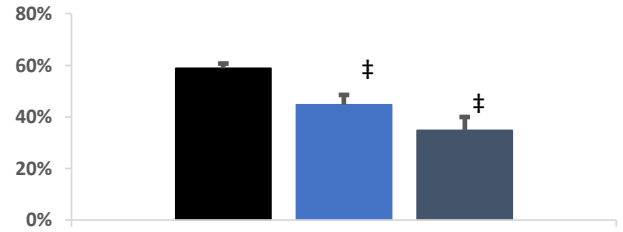
AP1189 vs ACTH in a model of Nephrotic Syndrome

ACTH (n=6)
AP1189 20mg/kg (n=8)
AP1189 40mg/kg (n=8)

Creatinine Clearance



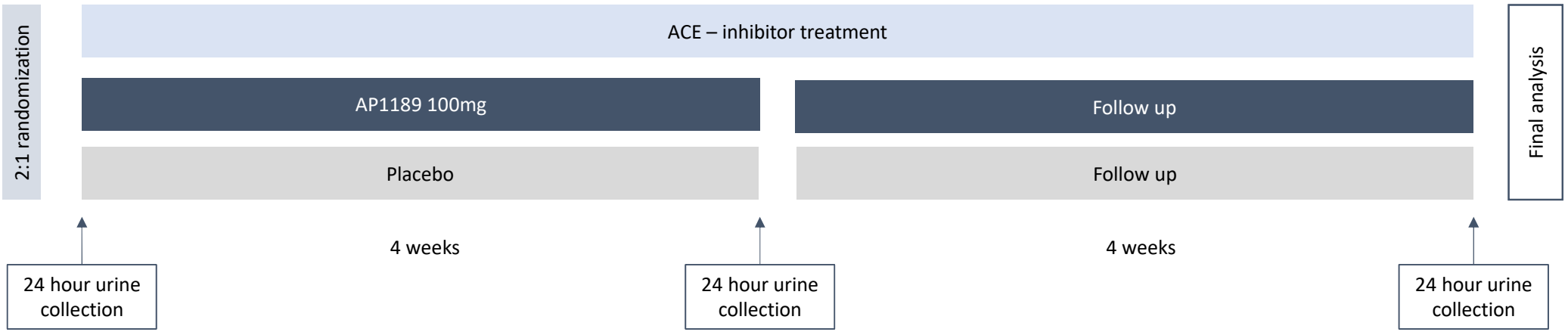
Fractional Albumin Excretion



AP1189 preserved creatinine clearance and dose-dependently reduced the fractional albumin excretion relative to ACTH

*p<0.05 vs. Day 14; † p<0.05 vs. vehicle; ‡ p<0.01 vs. ACTH; data from EP18179319.1 (non-published data); Lindskog et al, J Am Soc Nephrol. 2010; 21:1290-1298

AP1189 – Phase IIa Trial in Nephrotic Syndrome



Key inclusion criteria:

- Moderate-to-severe nephrotic syndrome
- Patients on stable dose of ACE inhibitors
- Patients are experiencing controlled blood pressure yet continued proteinuria

Primary endpoint: Change in 24 hour protein excretion following 4 weeks of treatment relative to baseline compared to placebo

Secondary endpoints include:

- Change in 24 hour albumin excretion following 4 weeks of treatment relative to baseline compared to placebo
- Change in plasma albumin from baseline to the end of the four week treatment period
- The number of subjects who show partial or complete remission in proteinuria on the last day of treatment and four weeks after the last dose is administered

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AP1189 – COVID-19

Patients at risk of developing Acute Respiratory Distress Syndrome (ARDS)

The Melanocortin System and its Role in Inflammation

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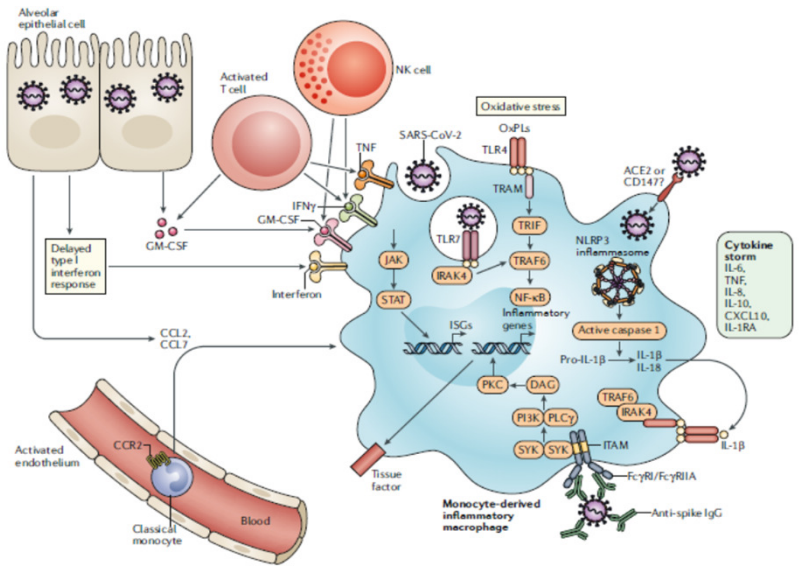
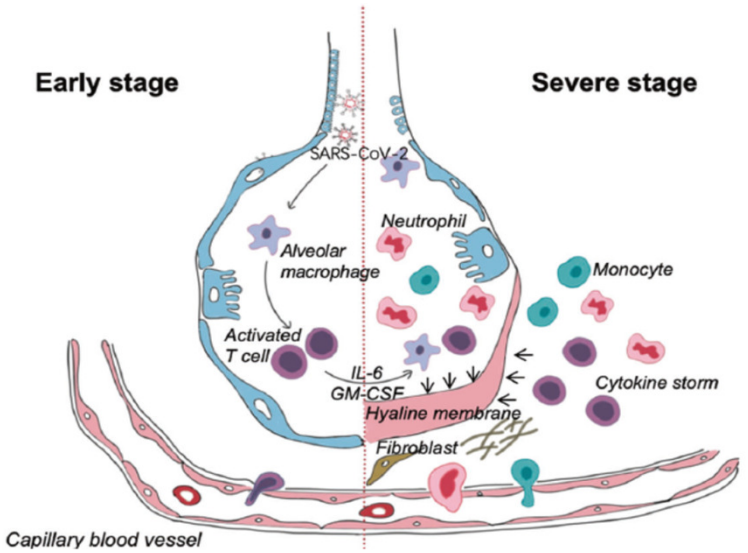
Macrophages play a role in COVID-19 hyper-inflammation



COVID-19 infection is associated with severe pneumonia that if left untreated can develop into ARDS due to an exacerbated inflammatory response

Hyperactivation of monocyte-derived macrophages is a key event leading to development of life-threatening ARDS

AP1189 targets key pathways in hyper-inflammation

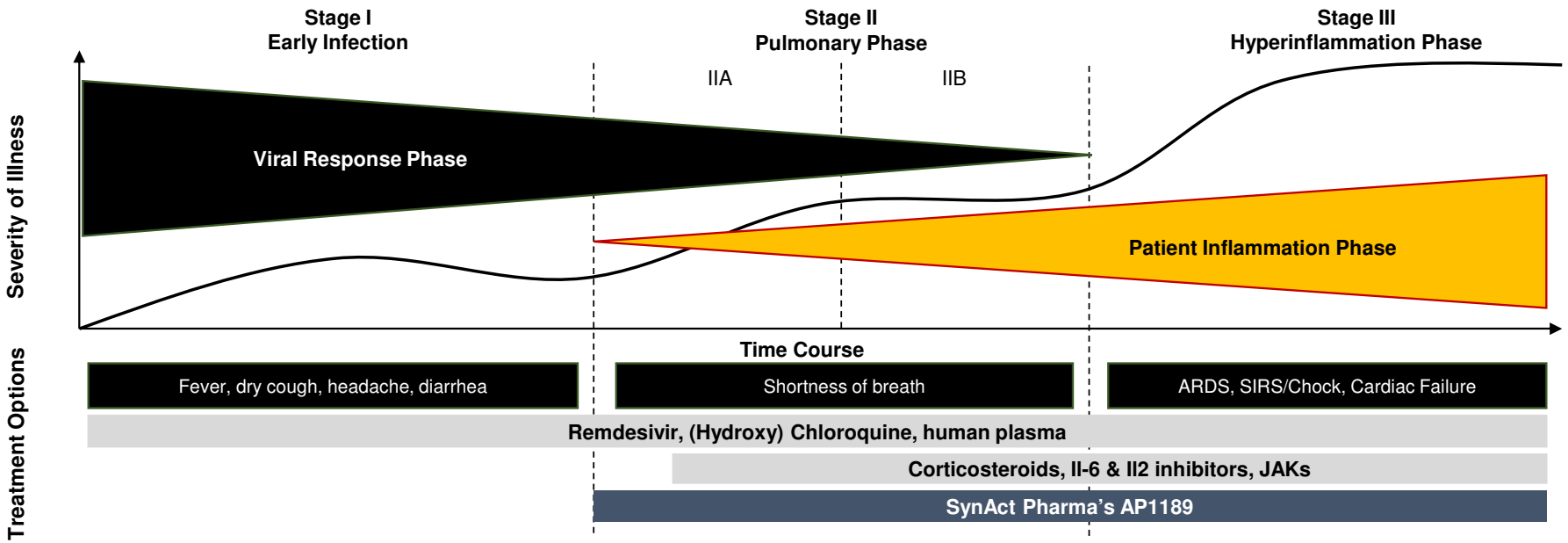


AP1189 induces inflammatory resolution by reducing pro-inflammatory pathways

Shifts the phenotype of macrophages from pro-inflammatory type 1 to a type 2 phenotype with marked pro-resolving capabilities

Nature Reviews Immunology, May 2020; Cell Death & Differentiation, March 2020

COVID-19: Disease development and AP1189's fit in treatment



AP1189's anti-inflammatory properties provide strong rationale to explore its potential utilization in COVID-19 infections

AP1189 as an add-on therapy in COVID-19 infected patients

Key inclusion criteria:

- Positive COVID-19 infection
- Need for supportive respiratory assist[1]

Primary endpoint: Time to respiratory recovery defined as the time from initiation of treatment to the time when the patient's SpO2 is ≥93% determined by pulse oximetry in the patient on ambient air for a minimum of 30 minutes

Key secondary endpoints:

- Rate of ICU admission during the treatment period
- Rate of patients that go to mechanical ventilation at any time during hospitalization
- Rate of patients discharged at Day 14 or before
- Rate of mortality at Day 28
- Length of hospitalization
- Length of stay in the ICU
- Number of supplementary oxygen-free days on Day 28

1. Defined as either SpO2 < 93% without supportive oxygen treatment or PaO2/ FiO2 <300 mmHg despite supportive oxygen treatment

SYNACT PHARMA

Summary

SynAct Pharma Investment Highlights

SYNACT PHARMA

Investment highlights

Focus on developing a novel treatment concept for inflammatory and autoimmune diseases by targeting the melanocortin system

High unmet need in inflammatory diseases for therapeutics that can spare the use of immunomodulatory agents due to their poor side effect profile

Lead asset, AP1189, currently in three separate Phase II trials for various indications with inflammatory manifestations, including orphan diseases

Several near-term value inflection points from Phase II data in rheumatoid arthritis, nephrotic syndrome and COVID-19-induced ARDS

Management with strong track record in clinical development as well as global business development and a supervisory board with decades of experience in research

Continually evaluating compelling business development opportunities

Expected news flow

Q4 2020	▪ Interim Phase IIa data Part 1 RA ▪ Initiation of Phase IIa Part 2 RA
Q1 2021	▪ Reporting of Phase IIa, Part 1 data in COVID-19-induced ARDS
Q2 2021	▪ Phase IIa RA study completion ▪ Phase IIa COVID-19-induced ARDS study completion
H2 2021	▪ End of Phase IIa meeting RA ▪ Completion date Phase IIa in Nephrotic Syndrome
H1 2022	▪ End of Phase IIa meeting Nephrotic Syndrome

SYNACT PHARMA

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