SYNACT PHARMA

SynAct Pharma AB

Resolving Inflammation Through Melanocortin Biology

ABG
19 May 2022

NON- CONFIDENTIAL

Forward Looking Statements

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SynAct Pharma – Highlights

- **SynAct Pharma is focused on the development of novel and first-in-class agonists that target the melanocortin system**
- **There is high unmet need in autoimmune and inflammatory diseases for efficacious and safe therapies – current therapies can present significant risk Vs benefit challenges**
- **Lead drug candidate, AP1189, is currently in Phase II development for various indications with inflammatory manifestations**
- **Several near-term value inflection points for AP1189:**
  - 1H22 – Envisaged up-list to Nasdaq Stockholm Main Market
  - 2022 – Continue clinical development in RA, Phase 2b under IND
  - 2022 – Continue development in Nephrotic Syndrome (redesigned study)
  - 2022 – Discovery results of new compounds targeting MC1R and MC3R
- **Management and Board of Directors possesses a strong track record in global pharmaceutical development, business development and science**

Facts and figures

Founded in 2012

Listed on Spotlight Stock Market with plan to uplist to Nasdaq Stockholm Main Market

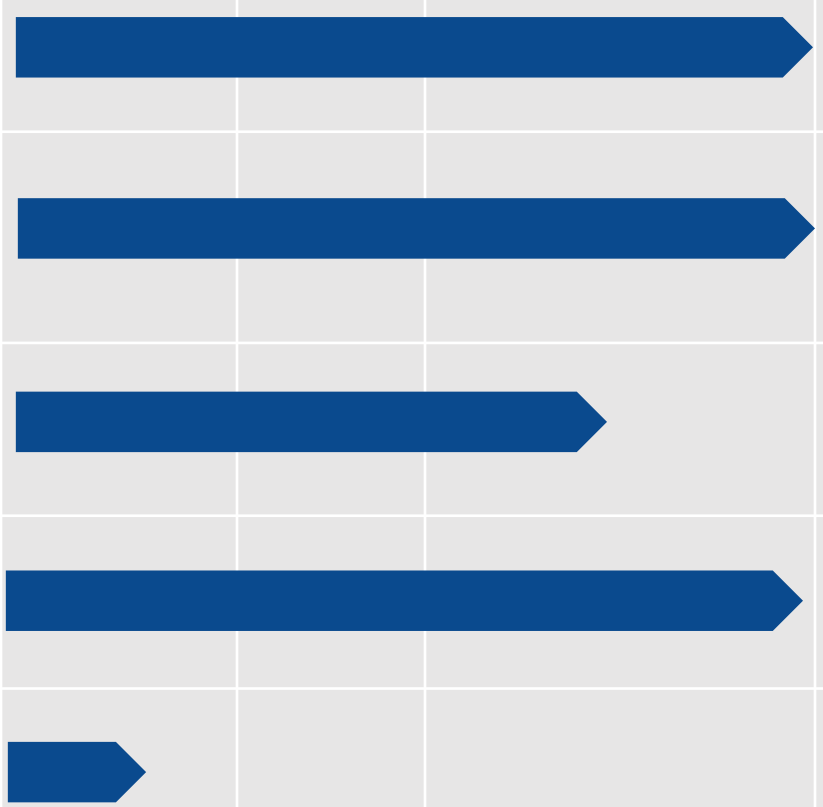
Ticker: (SYNACT:SS)

Market cap: c. SEK 1.4bn/US$140m (May 12, 2022)

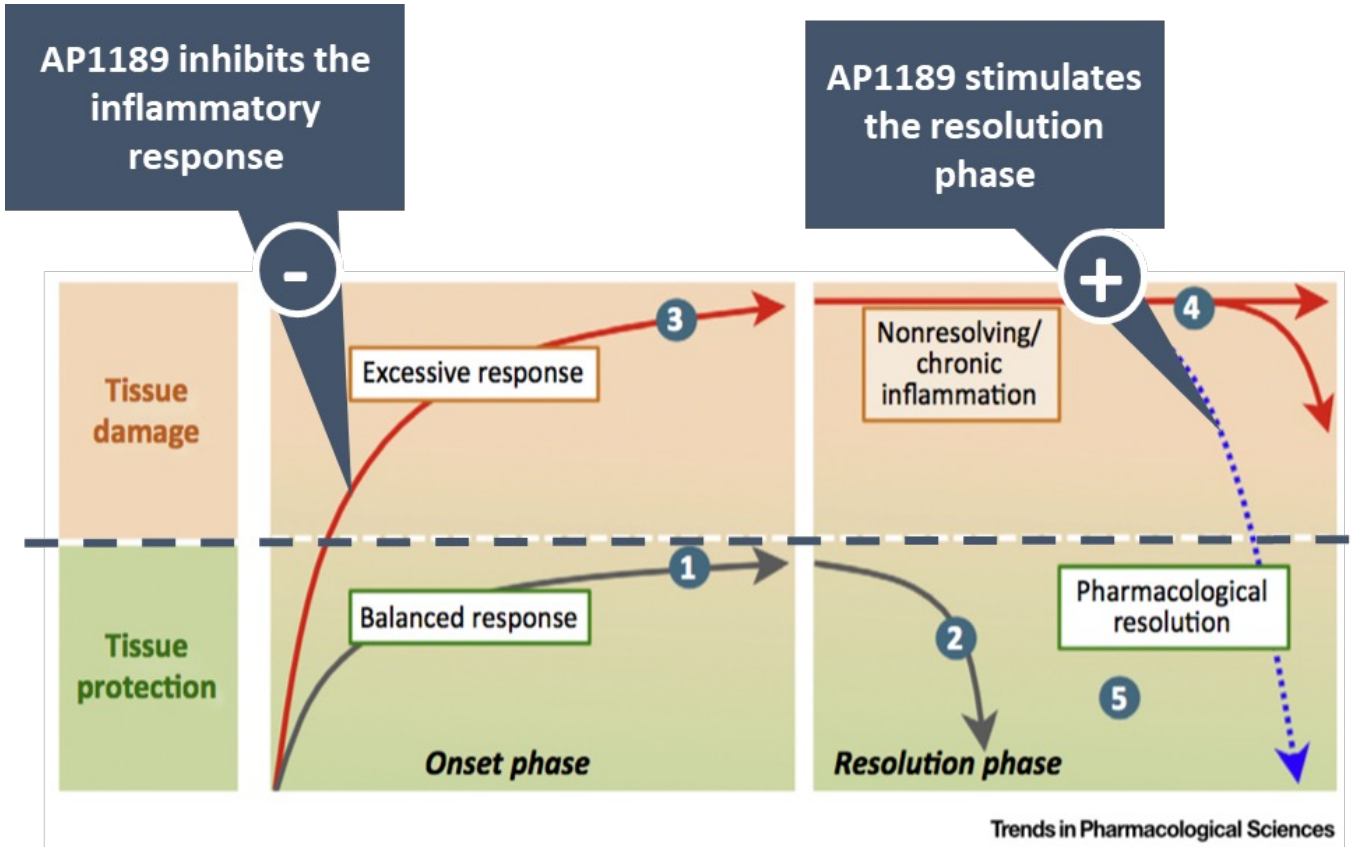
Management holds c. 20% ownership

SynAct Pharma – Pipeline overview

Asset	Indication	Preclinical	Phase I	Phase IIa	Phase IIb	Phase III	Next milestone
AP1189	Rheumatoid Arthritis – First line treatment						Filing CTA Q2 2022 High level data Q3 2023
	Rheumatoid Arthritis – DMARD-IR						Pre-IND meeting Q2 2022 IND filing PH2b H2 2022
	Nephrotic syndrome						Redesigned development program Q2 2022
	Virus- induced respiratory insufficiency						Data in non-COVID disease models H2 2022
Next generation of compounds	Inflammatory diseases						



Inflammation Resolution can correct dysregulated inflammation without inducing immunosuppression

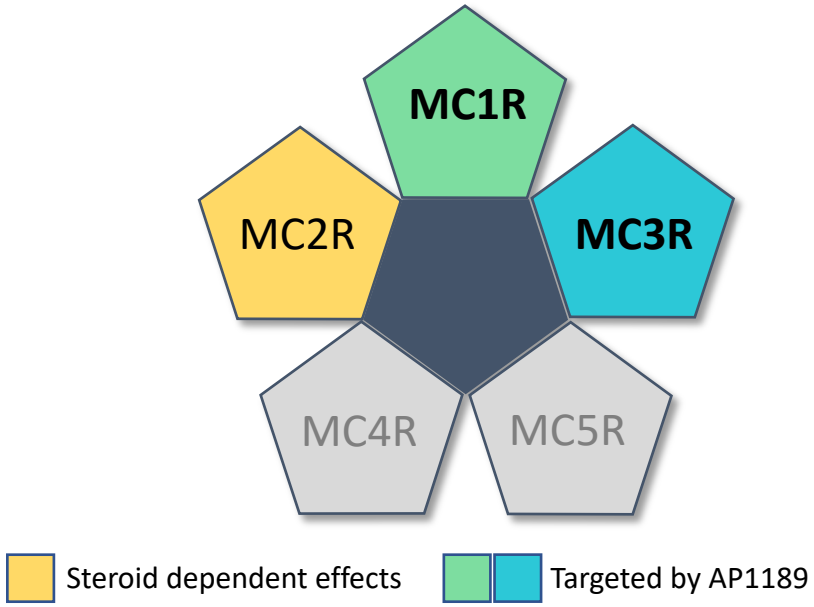


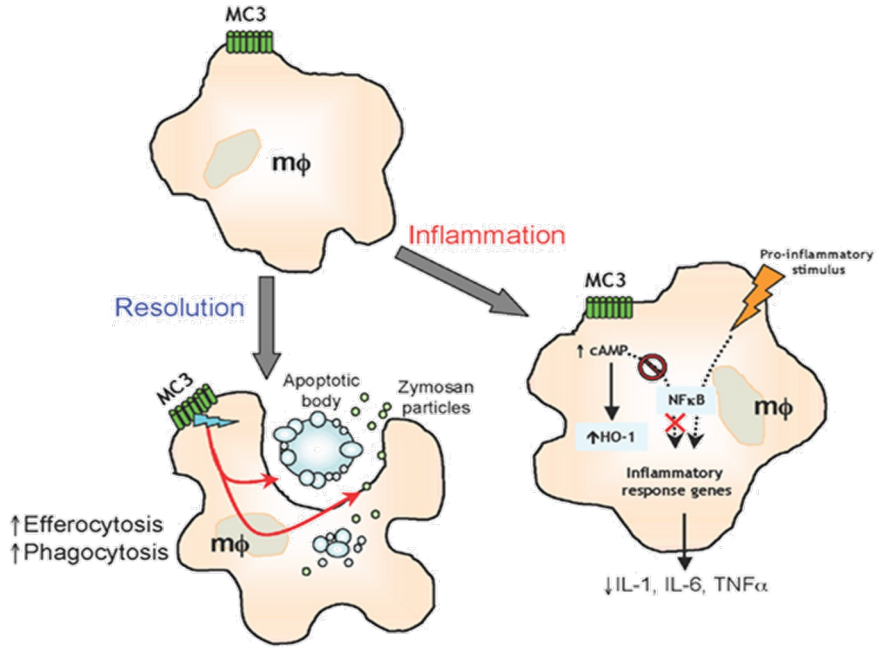
The inflammatory response

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 inflammation
5. Activation of endogenous resolution pathways has the potential to restore tissues and function

Cartoon adapted from Perretti *et al. Trends Pharmacol Sci* 2015;36:737–55

AP1189: A First-in-Class Selective Melanocortin Receptor 1 and 3 Agonist





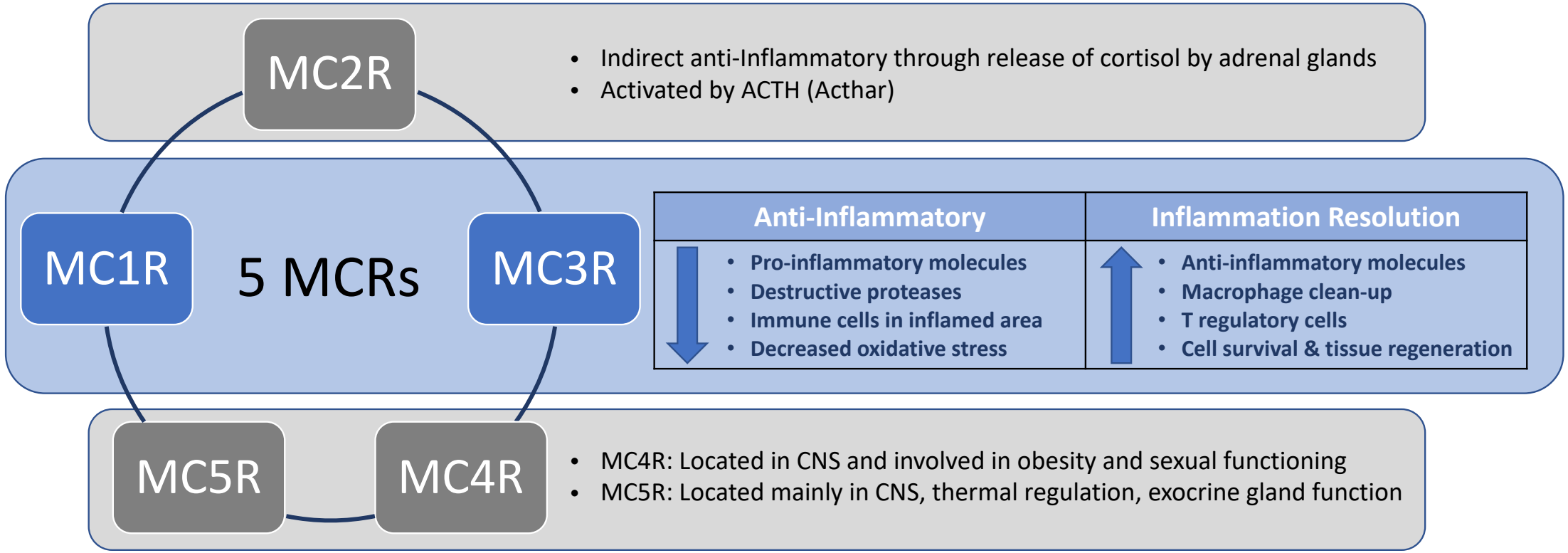
- AP1189 was designed to activate melanocortin receptors 1 and 3 (MC1R and MC3R), which are believed to be responsible for direct immunomodulatory effects
- Importantly, AP1189 does not activate MC2R, which is found on the adrenal glands and is responsible for the release of cortisol and the subsequent steroid side effects and tolerability issues that are associated with ACTH therapies

- AP1189 exhibits anti-inflammatory activity through stimulation of MC1R and MC3R on targets cells – lowering pro-inflammatory cytokines
- AP1189 promotes pro-resolution pathways following stimulation of MC1R and MC3R on targets cells – such as increasing efferocytosis in macrophages

ACTH: adrenocorticotropic hormone; MCR: melanocortin receptor

AP1189 is an selective melanocortin agonist having both anti-inflammatory and inflammation resolution activity without immunosuppression

- **The melanocortin system is an ancient modulatory system comprising a family of 5 widely dispersed melanocortin receptors and a set of naturally occurring melanocortin peptides that bind to and activate these receptors**
- **AP1189 selectively activates melanocortin 1 and 3 receptors which are responsible for direct immunomodulatory activity and importantly does not stimulate MC2R**



AP1189 – First-in-class biased MCR agonist – novel mode of action

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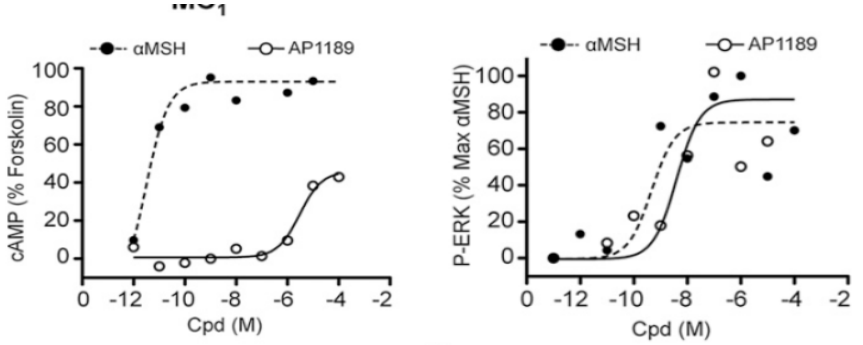
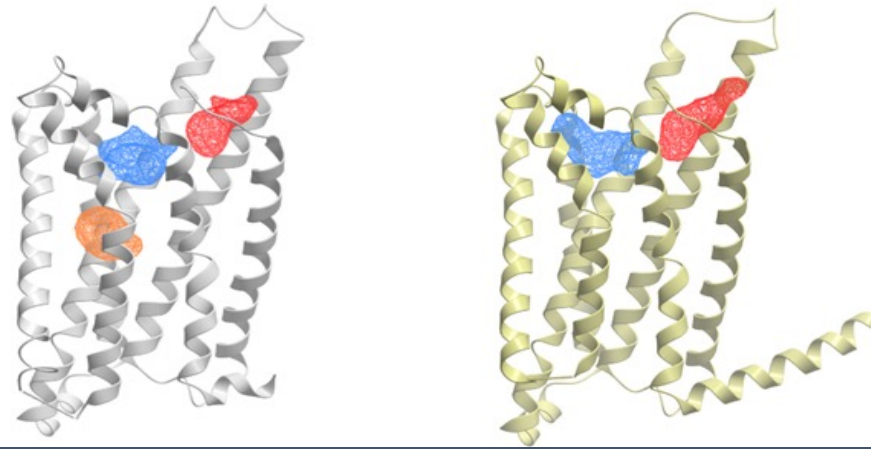
AP1189

- First-in-class biased melanocortin receptor type 1 and 3 agonist- potent inducer of ERG phosphorylation- weak inducer of cAMP stimulation[1]
- Homology modelling from recently published MCR crystal structure[2] identified allosteric binding pocket (red) in addition to othosteric binding pocket (blue)
- AP1189 fits very well into allosteric binding pocket

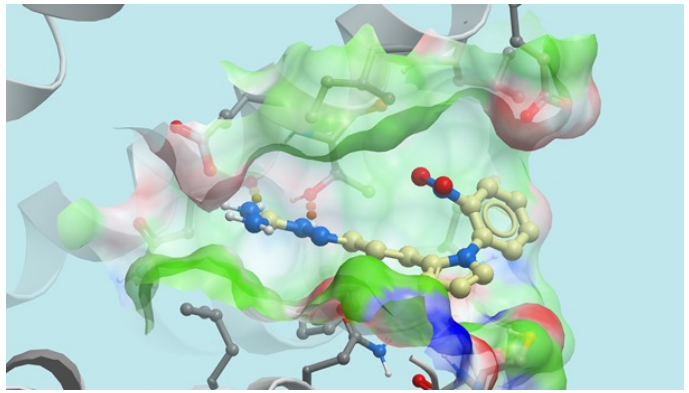
MC1 receptor activation in transfected cells[1]



AP1189 selectively engages MC1R



MC1r Model MC3r Model



Proposed binding mode of AP1189

1. Montero-Melendez et al: J Immunol. 194:3381-8, 2015; 2. Data from Completed Phase 1 study in healthy volunteers; 2. Yu, J. *et al.* Determination of the melanocortin-4 receptor structure identifies Ca as a cofactor for ligand binding. *Science* **368** , 428–433 (2020).

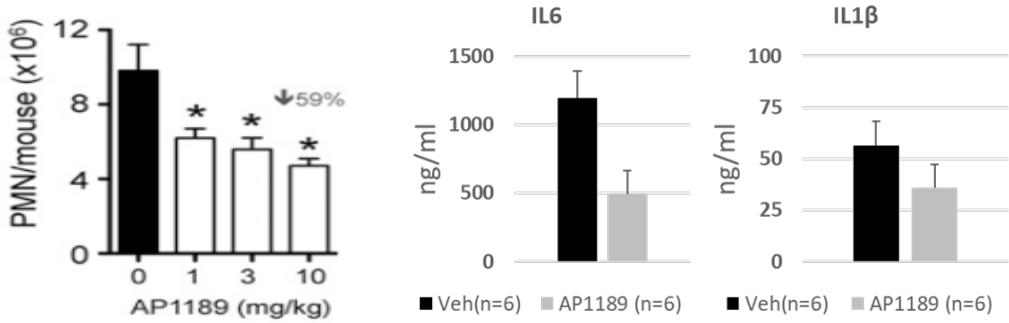
AP1189 – Combined anti-inflammatory and pro-resolving effects in vivo

?

AP1189

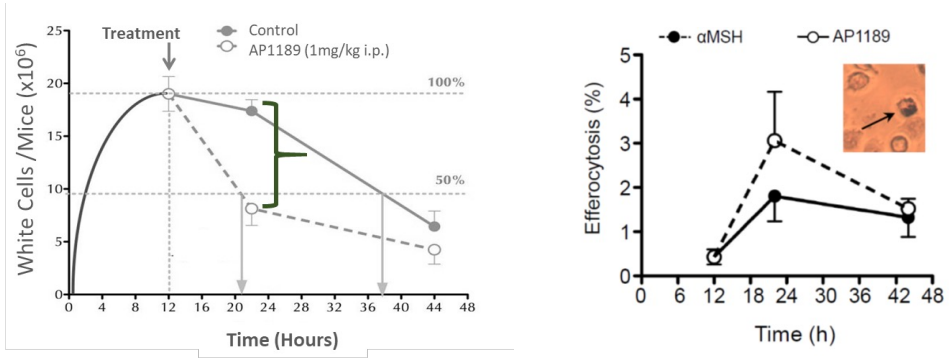
- AP1189 induces anti-inflammation as shown by reduced accumulation of neutrophils and reduced concentration of pro-inflammatory cytokines following systemic administration in murine peritonitis model –
- Therapeutic administration of AP1189 in murine peritonitis- ie treatment during ongoing inflammatory- induces fast clearance of Neutrophils
- AP1189 given therapeutic induces efferocytosis by macrophages

Administration at disease induction in peritonitis[1]



AP1189 – inhibition of inflammatory responses

Therapeutic treatment in peritonitis [1]



AP1189 – Promote inflammatory resolution

1. Montero-Melendez et al: J Immunol. 194:3381-8, 2015

AP1189 – Biased agonism is not associated with melanogenesis

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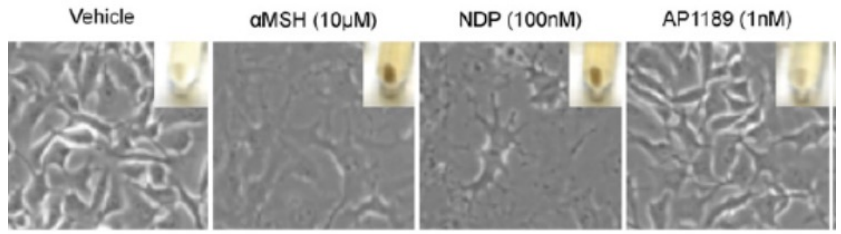
AP1189

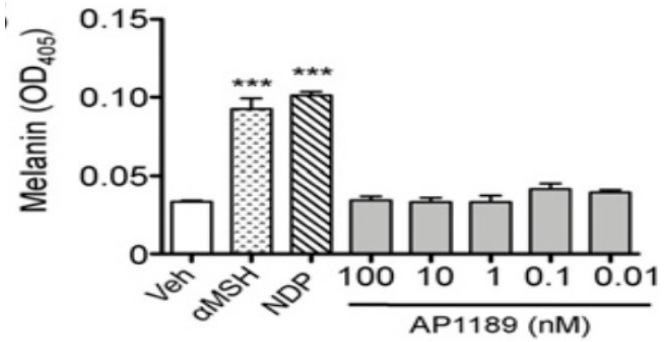
- Melanogenesis and thereby skin pigmentation is an unwanted side effect of melanocortin receptor stimulation
- Melanogenesis in melanocytes is mediated by cAMP accumulation following stimulation of MC1r by classical agonists
- Compared to classical agonists as αMSH and NDP-MSH, incubation with AP1189 in the full concentration range from 0.01-100 nM did not induce any melanogenesis
- No indication of skin pigmentation has been reported in the clinical ongoing Phase 2 clinical development program with AP1189

B16-F10 melanoma cells following 24 hours incubation



AP1189 – promote inflammatory resolution

Effects of MC compounds on melanogenesis[1]



AP1189 does not induce melanogenesis

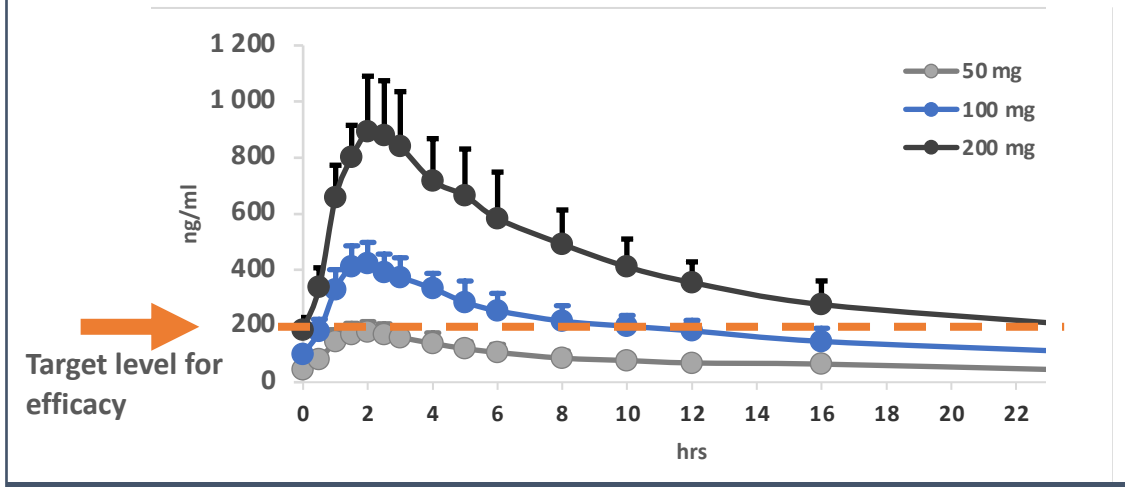
1. Montero-Melendez et al: J Immunol. 194:3381-8, 2015

AP1189 addresses key limitations faced in the development of melanocortin agonists

AP1189 – the first oral selective MC agonist

- The naturally occurring melanocortin agonists including ACTH (Acthar) and the majority of designed analogs are peptides with short half-lives creating delivery and development challenges
  - AP1189 is a small molecule with good oral bioavailability and a half-life that enables once-daily dosing
- By selectively stimulating the MCRs responsible for direct immunomodulatory effects and not stimulating cortisol release, AP1189 does not cause the immunosuppression seen with most approved RA therapies
- AP1189 is also a biased agonist that preferentially stimulates the ERK phosphorylation pathway which may result in fewer off-target effects like hyper skin pigmentation

PK profile following repeat dosing in man[1]



PK profile demonstrates once daily potential

1. AP1189-CS001 study MAD study

Ready for 12 weeks development with once daily dosing with tablets

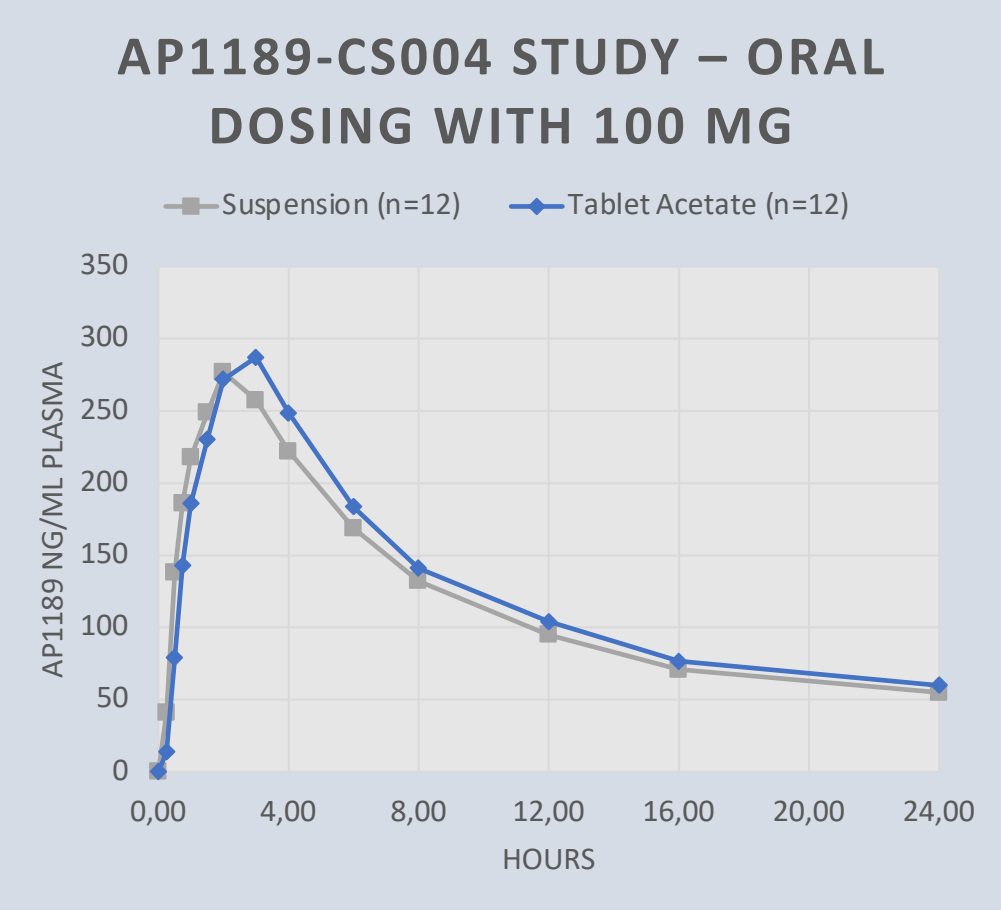
Toxicology:

- Three Months Tox studies with recovery groups successfully been completed
- NOAEL rat: 100 mg/kg
- NOAEL pig: 50 mg/kg
- No new findings compared to the 4 weeks studies completed before entering FIM

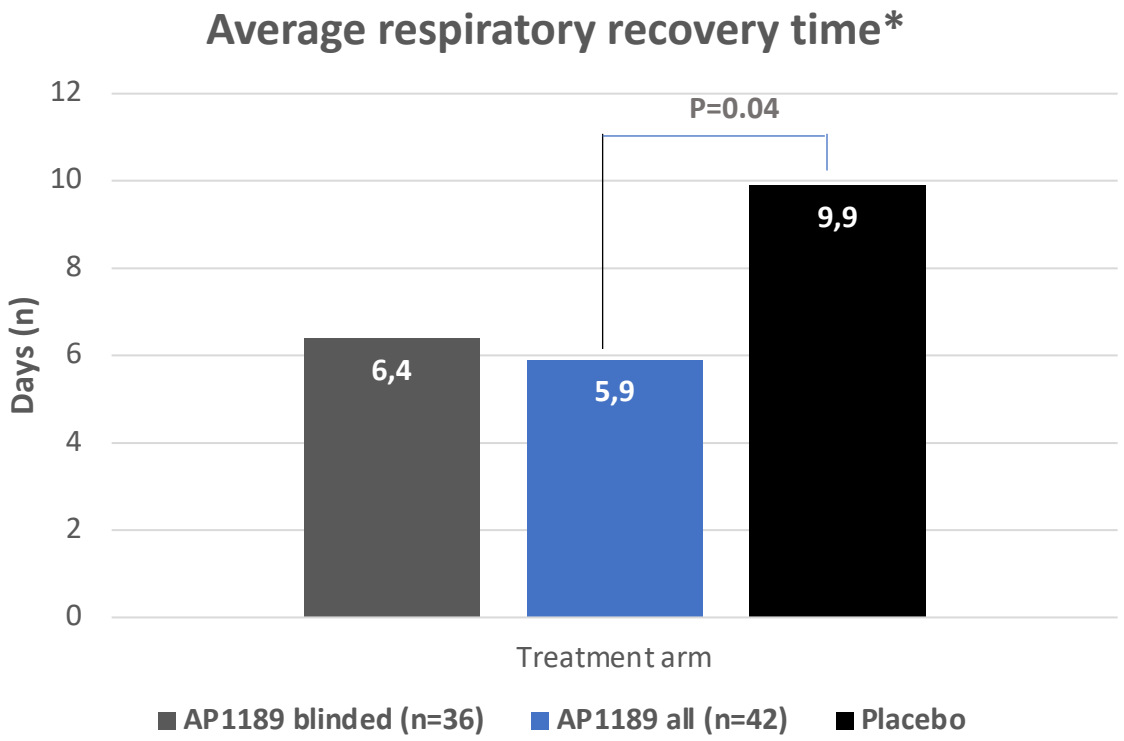
Tablets humans:

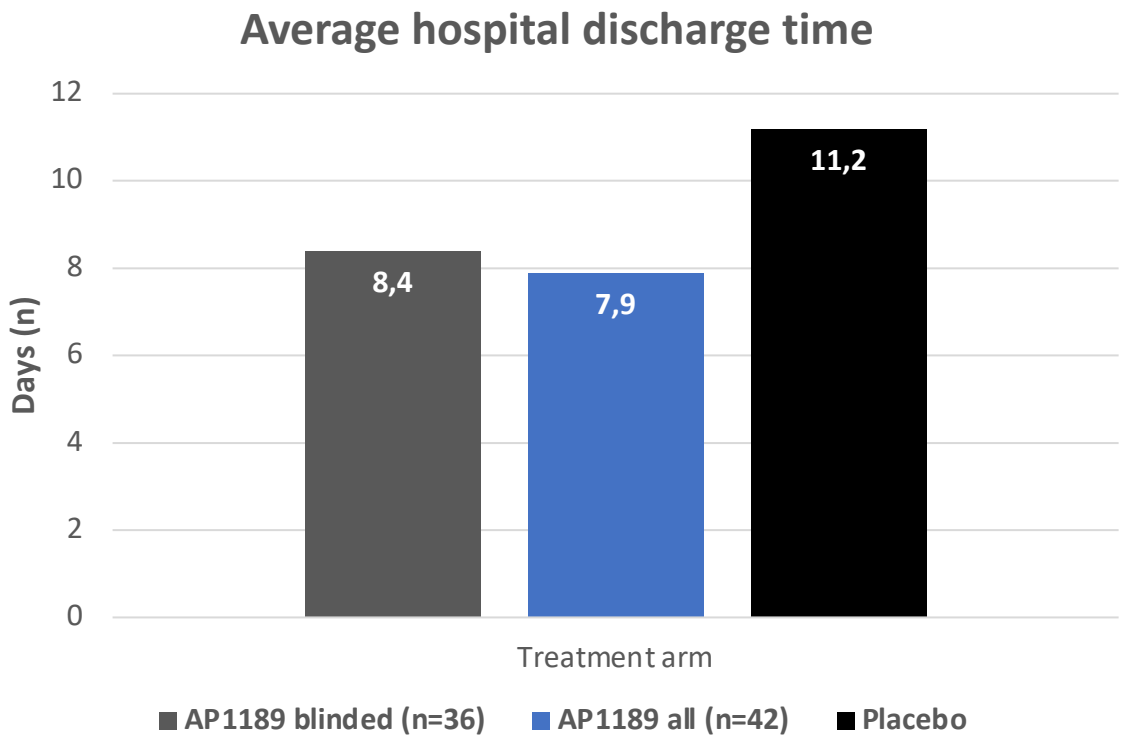
Crossover study in 12 healthy male volunteers completed

Tablet well tolerated – reports of nausea following dosing with suspension, up to now no reports following dosing with tablets



AP1189: Once daily dosing to COVID-19 pts with respiratory insufficiency: patients achieved recovery (no need for supplementary oxygen) 4 days and hospital discharge 3.5 days earlier than placebo-treated patients



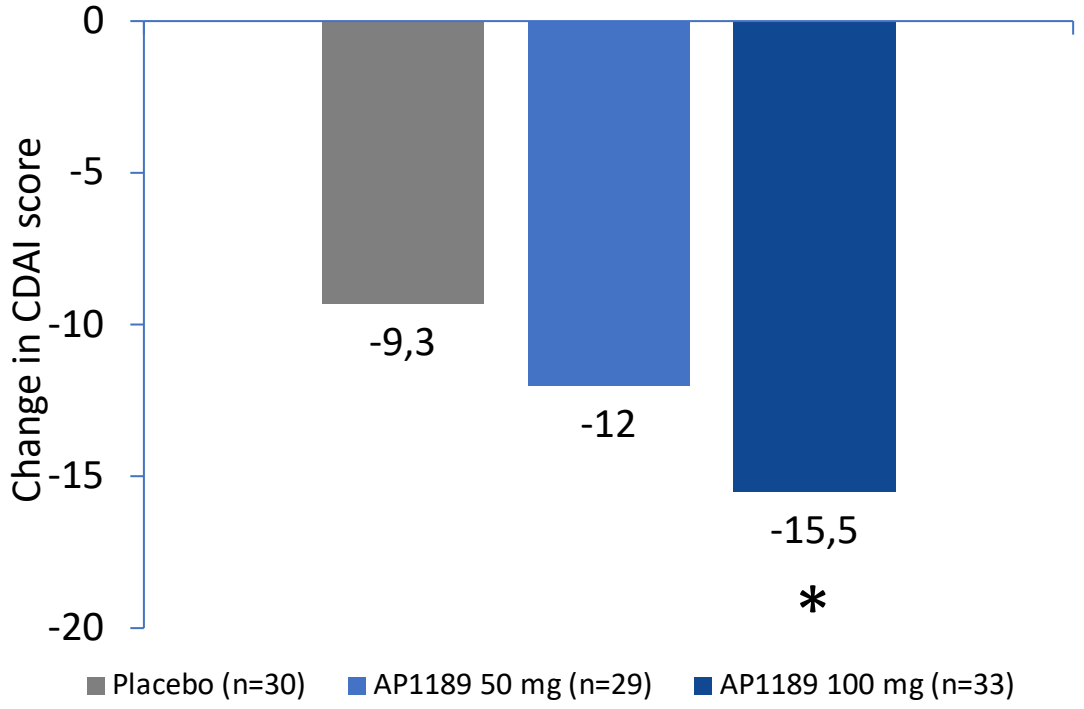


*Normal saturation without oxygen supplementation. The "AP1189 all" includes 6 open-label safety run-in patients

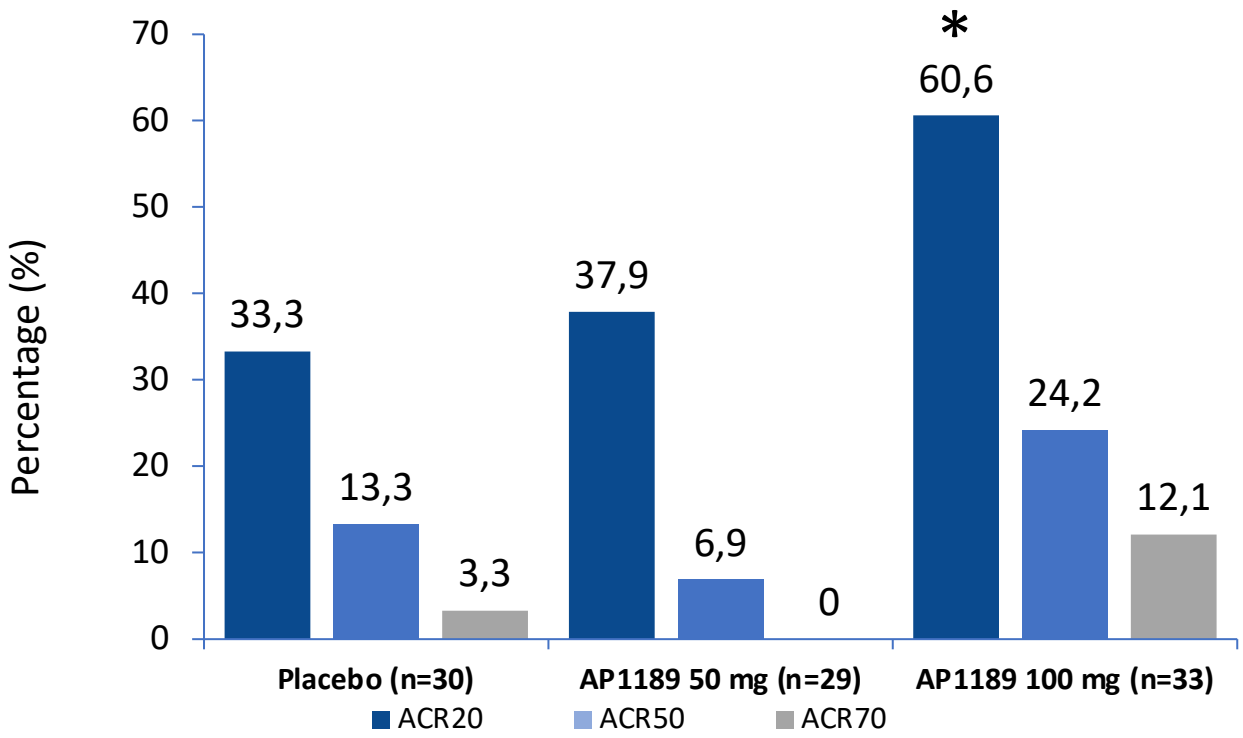
AP1189- PoC as first line treatment in combination with MTX-

-4 wks once daily oral dosing

Statistically significant reduction in disease activity (primary study readout)



Statistically significant higher ACR20 response rate (secondary study readout)



*:p<0.05 vs placebo

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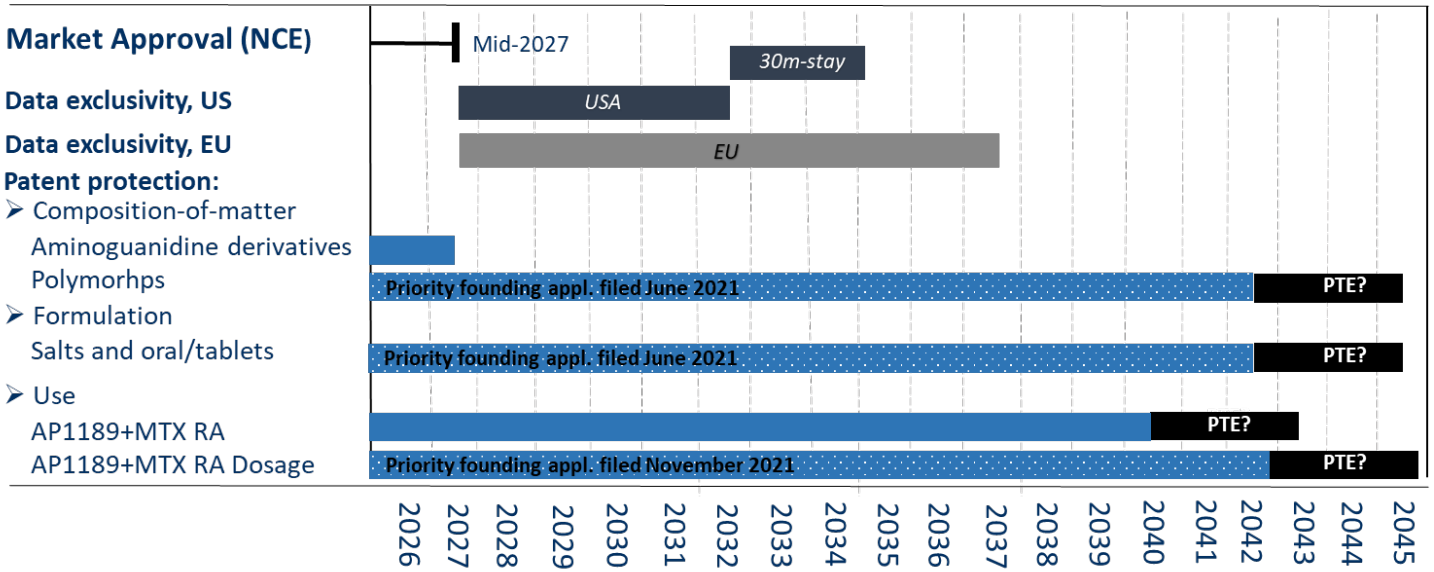
AP1189 is well protected with composition running through 2042 with additional layers of protection

Portfolio Coverage

Composition of Matter	Granted: Genus of AP1189
	Pending: Polymorphs; Salts*
Formulation	Pending: Oral administration/tablets*
Method of Use	Granted (EP): Arthritis/AP1189+MTX in RA; Kidney disease
	Pending: Viral diseases (incl COVID-19 symptoms); Cardiovascular diseases; Dose effects AP1189+MTX in RA

Example of Exclusivity if AP1189 is Approved in RA as First Indication



- *Data exclusivity: US 5 years for NCE, EU 8+2 years (8 year data protection, 10-year market protection)*
- *PTE = Patent term extension (=SPC in EU, 'Supplementary Protection Certificate'), applicable for one patent only, and duration uncertain*

IP Strategy

- Critical composition of matter coverage directed toward the genus of AP1189
- Filings directed toward the AP1189 salt forms provide extended coverage of AP1189 proposed marketed product
- Filings directed toward method of use for AP1189 provide further lifecycle management
- IP strategy in collaboration with Høiberg, DK and Baker & Hostetler, Philadelphia
- 2nd Opinion and Portfolio Review, Oct 2021: Cooley, New York, Boston

*Salts and oral formulations are included in the same application

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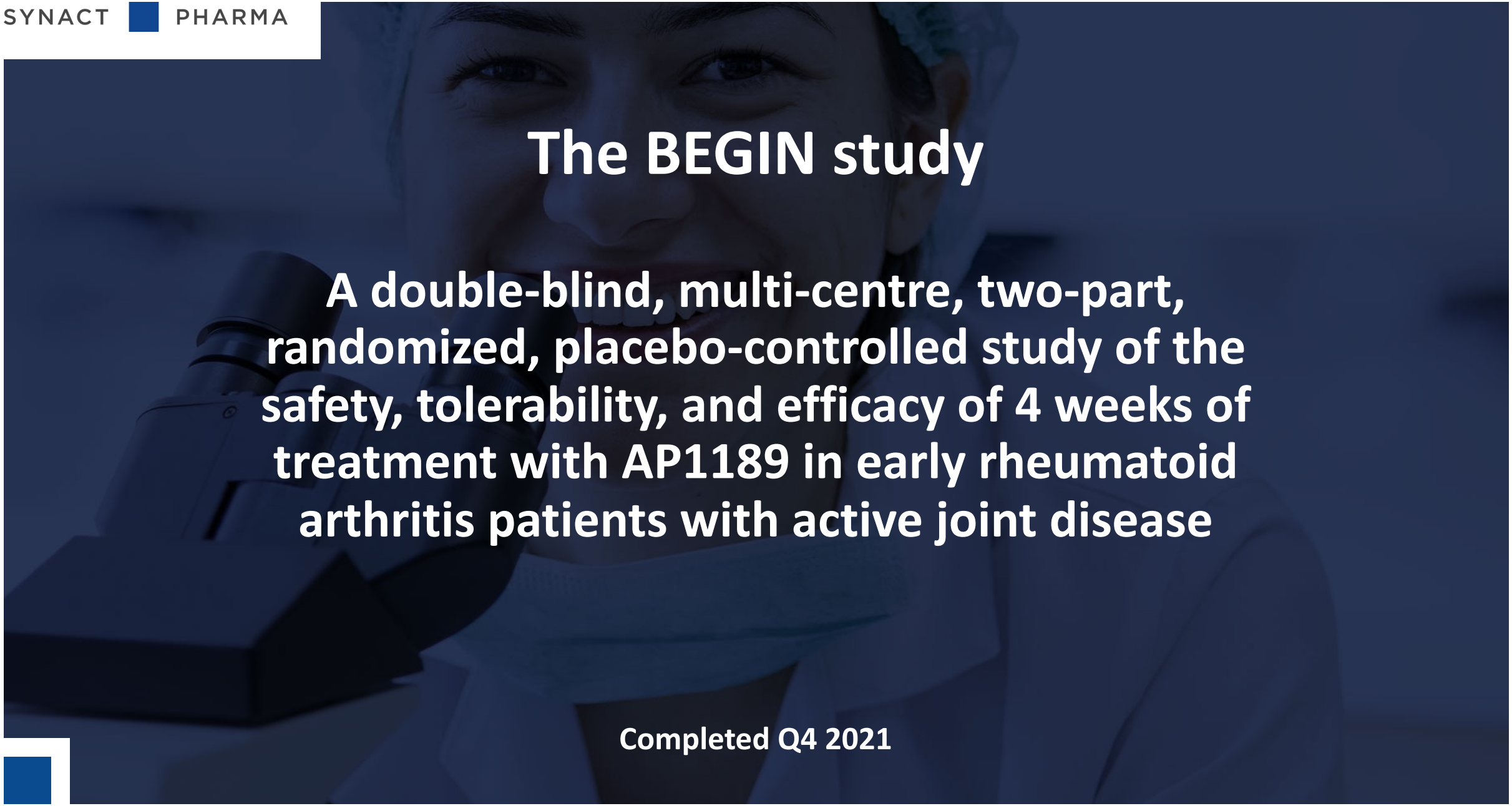


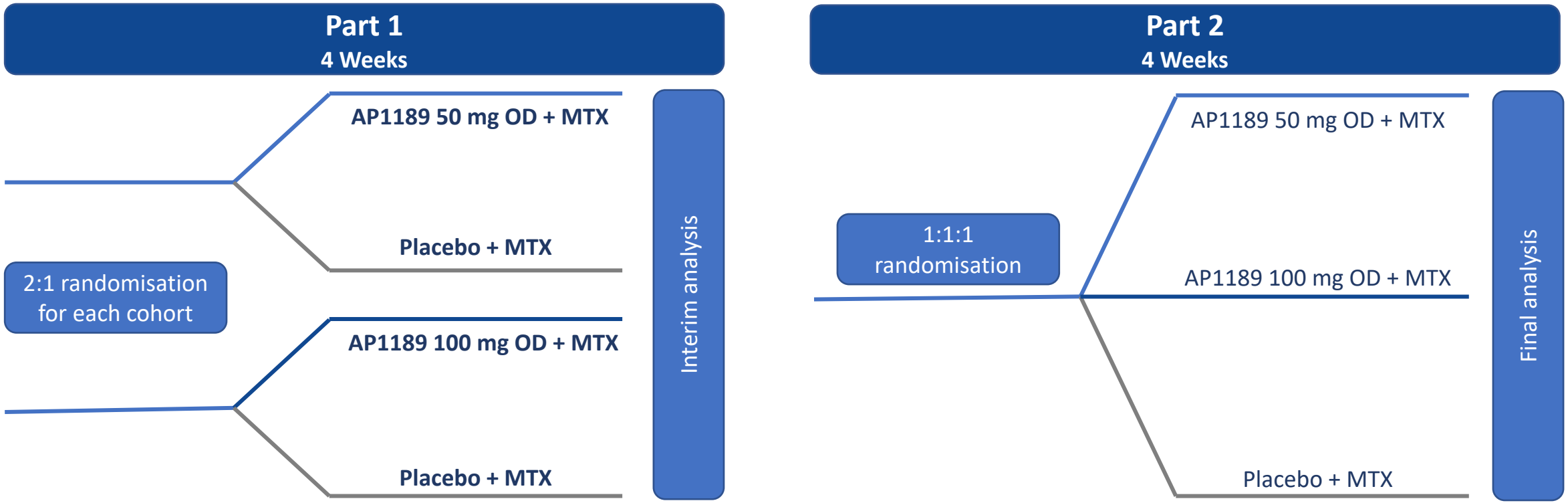
The BEGIN study

A double-blind, multi-centre, two-part, randomized, placebo-controlled study of the safety, tolerability, and efficacy of 4 weeks of treatment with AP1189 in early rheumatoid arthritis patients with active joint disease

Completed Q4 2021

BEGIN: P2a POC 4-week study of AP1189 + MTX in severely active early RA

Study Population	Primary safety endpoint	Primary efficacy endpoint
Adult patients (aged 18 to 85 years) with severe RA defined as CDAI >22, who are about to begin up-titration with MTX	Safety of AP1189 vs placebo (AEs and SAEs)	• Mean change in CDAI or • % of patients improving from severe (CDAI >22) to at least moderate (CDAI ≤22)



Baseline characteristics of randomized population

	Placebo (n=34)	AP1189 50 mg (n=35)	AP1189 100 mg (n=36)
Female, %	79.4	77.1	77.8
Age (years)			
Mean±SD	56.4±13.1	56.4±13.1	55.3±13.9
Median (range)	61 (26–78)	57 (28–79)	56.5 (27–77)
18–64 years/65–84 years (%)	73.5/26.5	71.4/28.5	72.2/27.8
Race, White/Asian/Other (n)	34/0/0	34/1/0	35/0/1
Weight (kg)			
Mean±SD	75.2±19.7	75.9±17.7	79.3±16.7
Median (range)	71.6 (48–145)	75.3 (42–111)	76.6 (48–118)
Height (cm)			
Mean±SD	167.5±7.8	167.5±6.6	166.3±8.4
Median (range)	167 (155–185)	167 (157–183)	165.5 (151–183)

cm, centimetre; kg, kilogram; SD, standard deviation

Baseline disease activity in efficacy assessment population

	Placebo (n=30)*	AP1189 50 mg (n=29)*	AP1189 100 mg (n=33)*
CDAI	36.7±8.7	36.1±9.7	39.5±9.1
SJC	10.4±3.7	10.2±3.9	12.1±4.9
TJC	13.7±6.1	13.6±6.0	15.2±6.0
DAS-28	5.5±1.0	5.4±0.8	5.7±0.9
Patient Global Assessment (VAS)	6.5±1.8	6.1±2.1	5.8±2.2
Physician Global Assessment (VAS)	6.2±1.1	6.3±1.3	6.4±1.3
CRP (mg/L)	18.7±26.6	14.9±18.9	26.3±41.9

All values are mean±SD. * mean baseline levels per group in the efficacy analyse set:. 2 randomised pts in each group did not complete dosing. 7 patients were taken out of the efficacy evaluation due to protocol violation. Placebo: n=2; AP1189 50 mg: n=4; AP1189 100 mg: n=1-

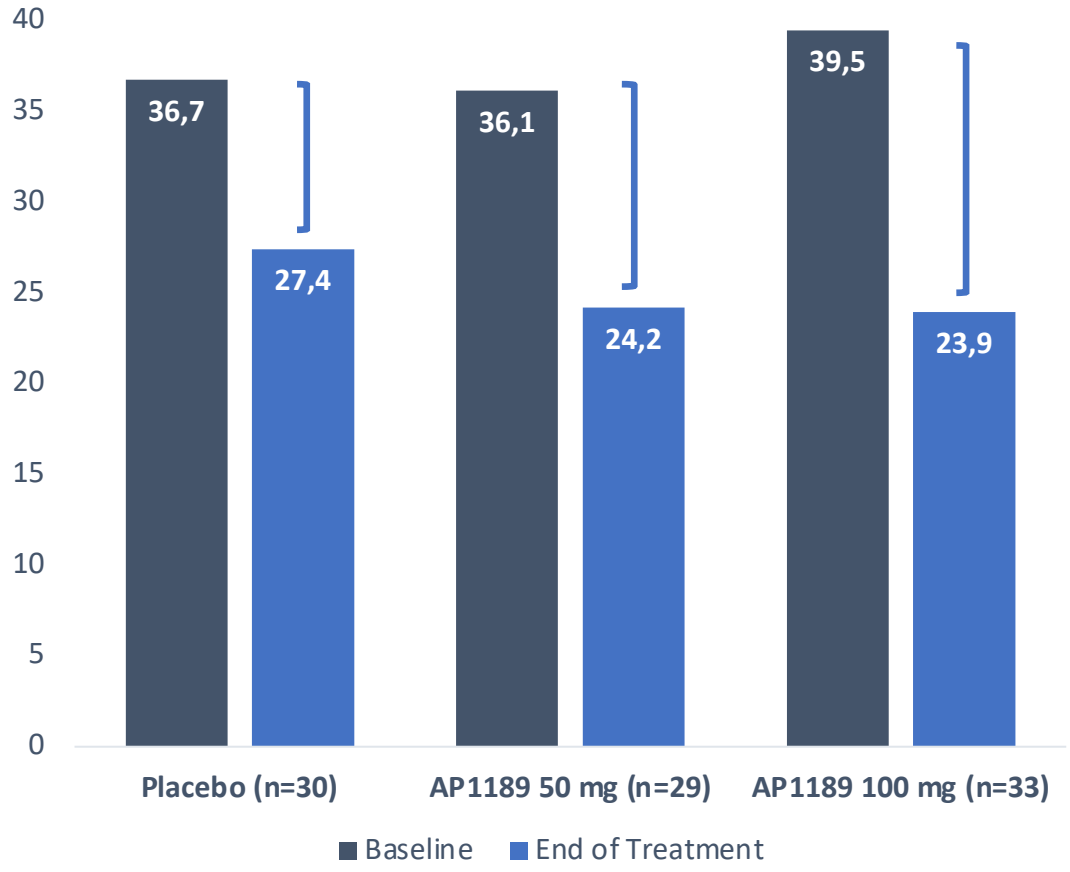
CDAI, Clinical Disease Activity Index; CRP, C-reactive protein; DAS-28, Disease Activity Score in 28 joints; SJC, Swollen Joint Count; TJC, Tender Joint Count; VAS, visual analogue scale

BEGIN P2a POC RA study topline results: AP1189 100mg once-daily oral dose group was efficacious and safe

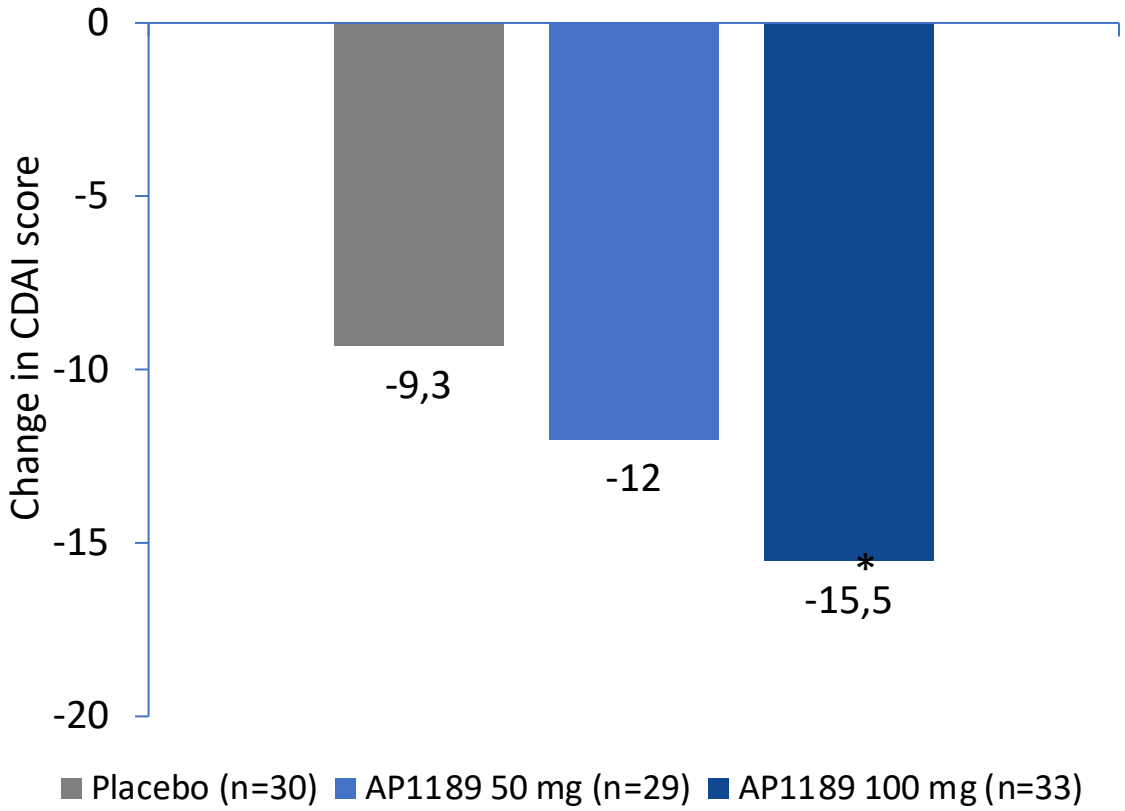
- **After 1-mo of treatment, the 100mg once-daily oral AP1189 group had:**
  - **Significantly better CDAI improvement Vs placebo and that was above the established minimally important clinical difference (MCID)** (15.5-point improvement for 100mg and 9-point for placebo)
  - **Significantly higher ACR20 response than placebo** (61% for 100 mg and 33% for placebo)
  - **Good DAS28(CRP) response in severely active patients** (68% for 100 mg and 37% for placebo)
  - **Mean improvement in FACIT-Fatigue score that was 2x the established minimally important clinical difference (MCID)** (8 for 100 mg and 4.8 for placebo)
- **The 50mg AP1189 dose was found to be partially effective**
- **AP1189 was safe and well tolerated with no SAEs, no discontinuations deemed due to study drug, no infections and no discernible impact on wbc count or vital signs**

AP1189 100mg group had a mean improvement in CDAI above the established minimally important clinical difference (MCID)[+] score and was significant Vs placebo

CDAI at Baseline and End of Treatment



Mean change in CDAI from baseline to Week 4



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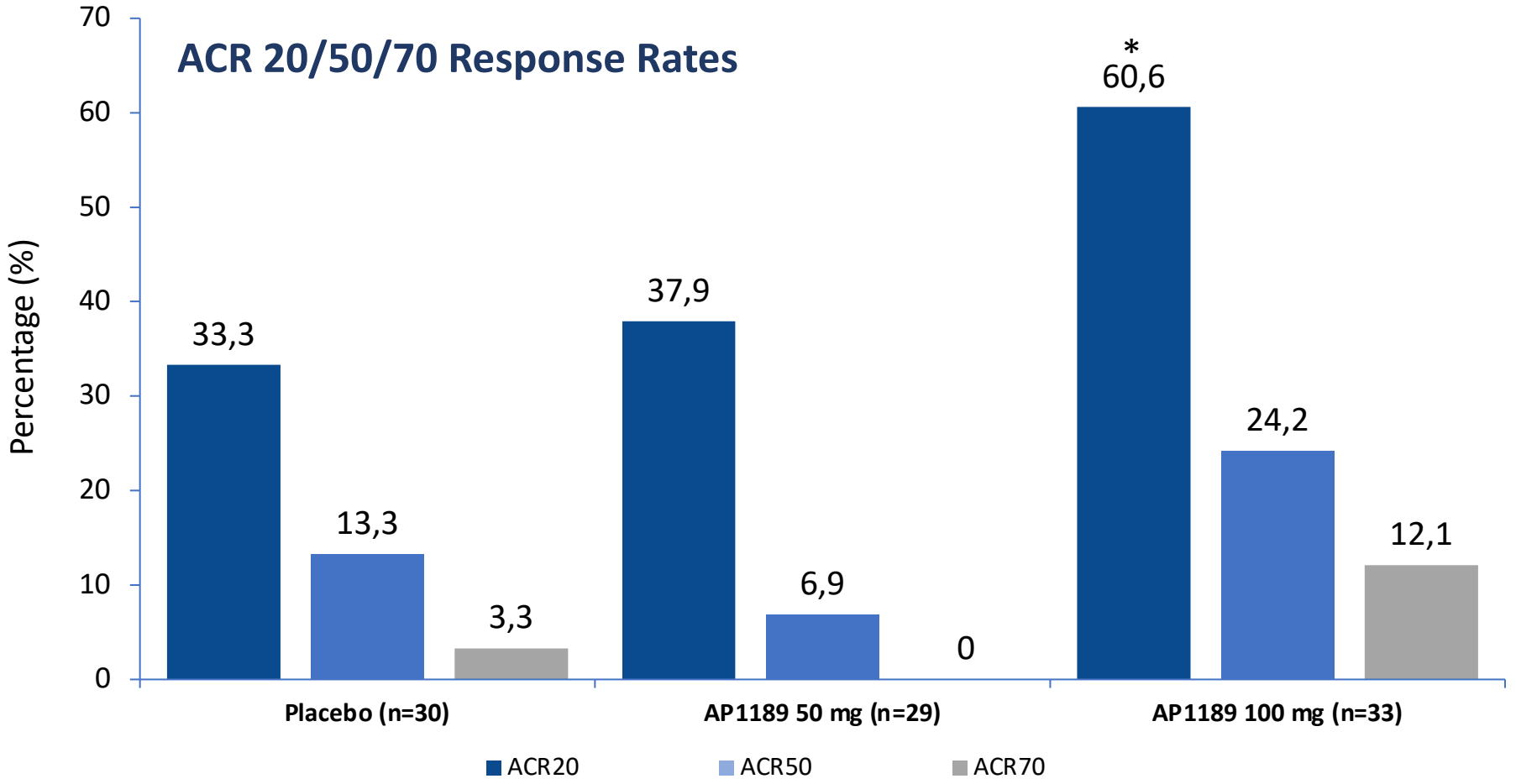
* = p<0.05 vs placebo, study primary endpoint

(+) Curtis et. Al *Arthritis Care Res (Hoboken)*. 2015 October ; 67(10)

Proportion of patients achieving ACR20/50/70 response at 1-mo

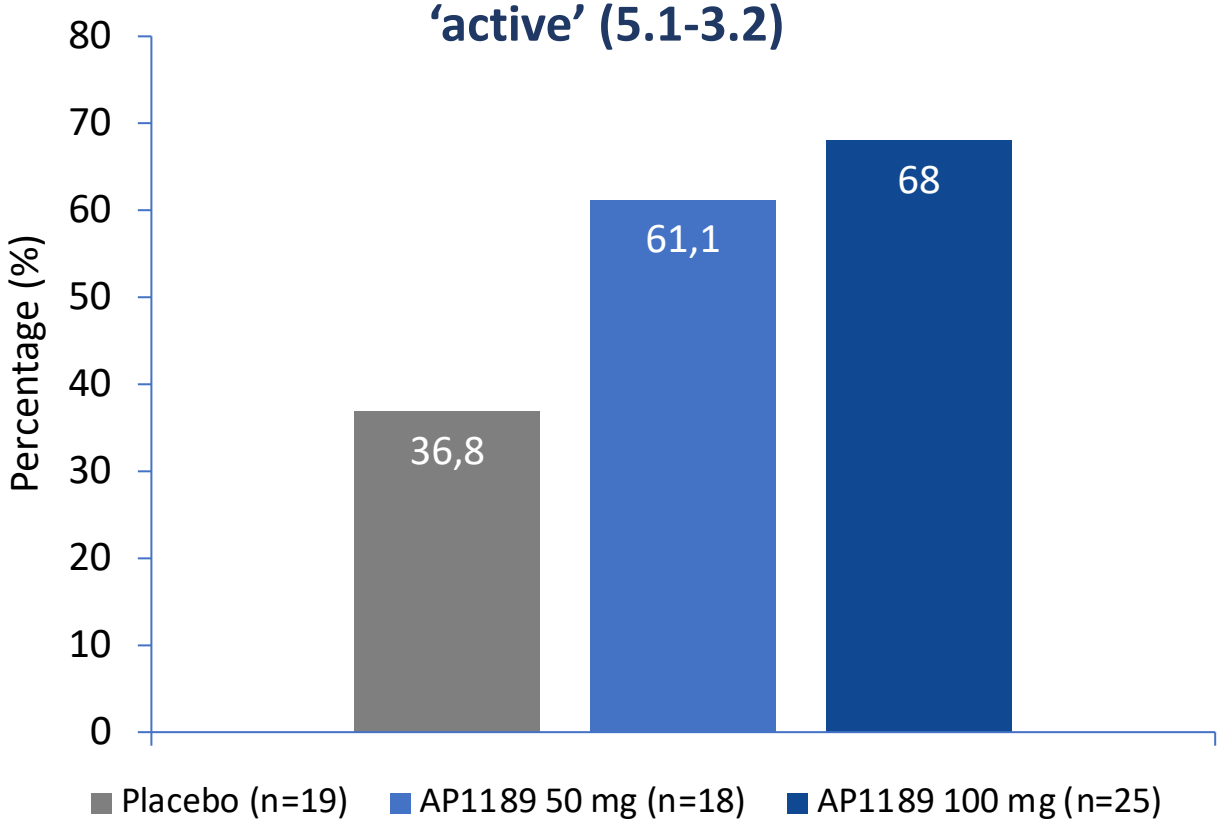


Robust 1-Mo ACR20/50/70 response rates for 100mg AP1189

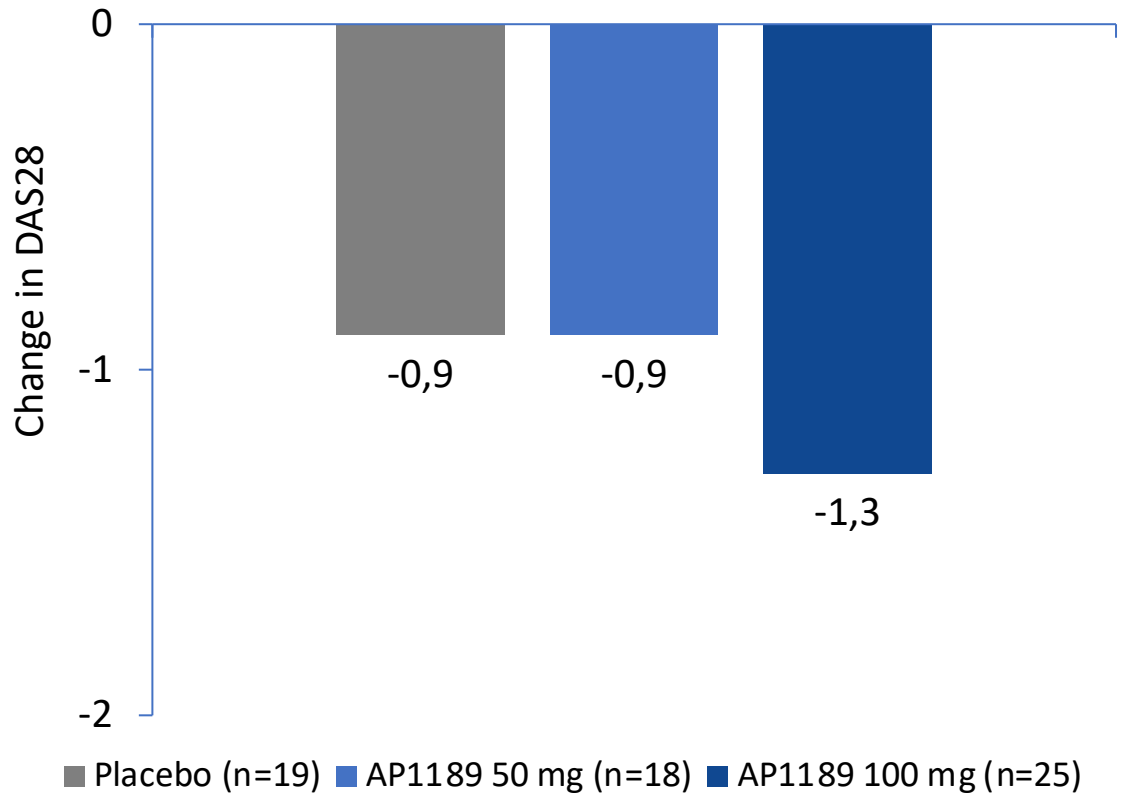
* = p<0.05 vs placebo

Change in DAS28 (CRP) - subset of ‘very active’ with >5.1 score at baseline

Patients going from ‘very active’ (>5.1) to ‘active’ (5.1-3.2)

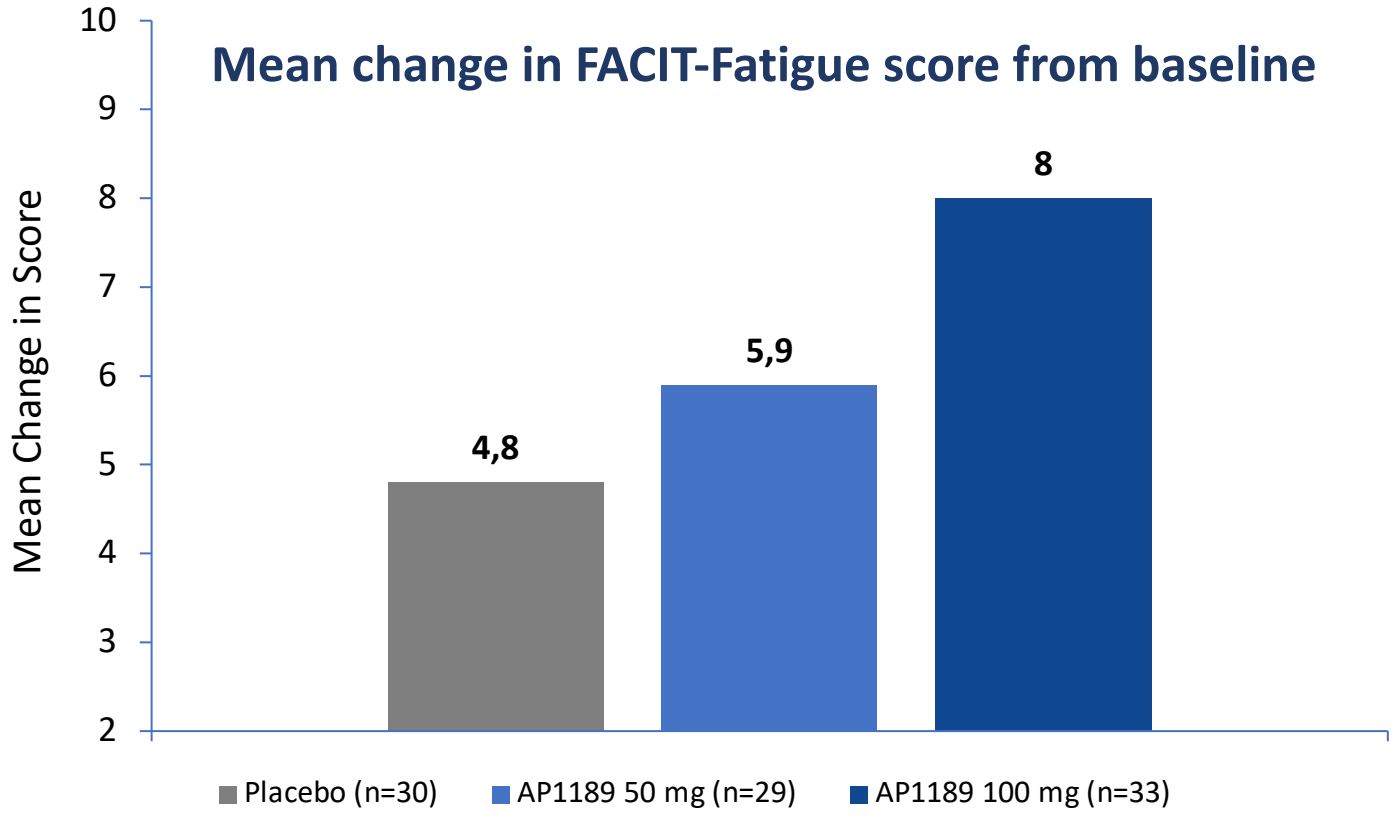


Mean Change in DAS28 from Baseline to Week 4



Good effects seen with 100mg in patients with very active DAS28 (>5.1) at baseline

Mean change in FACIT-Fatigue score at 1-mo



Mean change in 100mg group is 2x the established minimally important clinical difference (MCID)⁺

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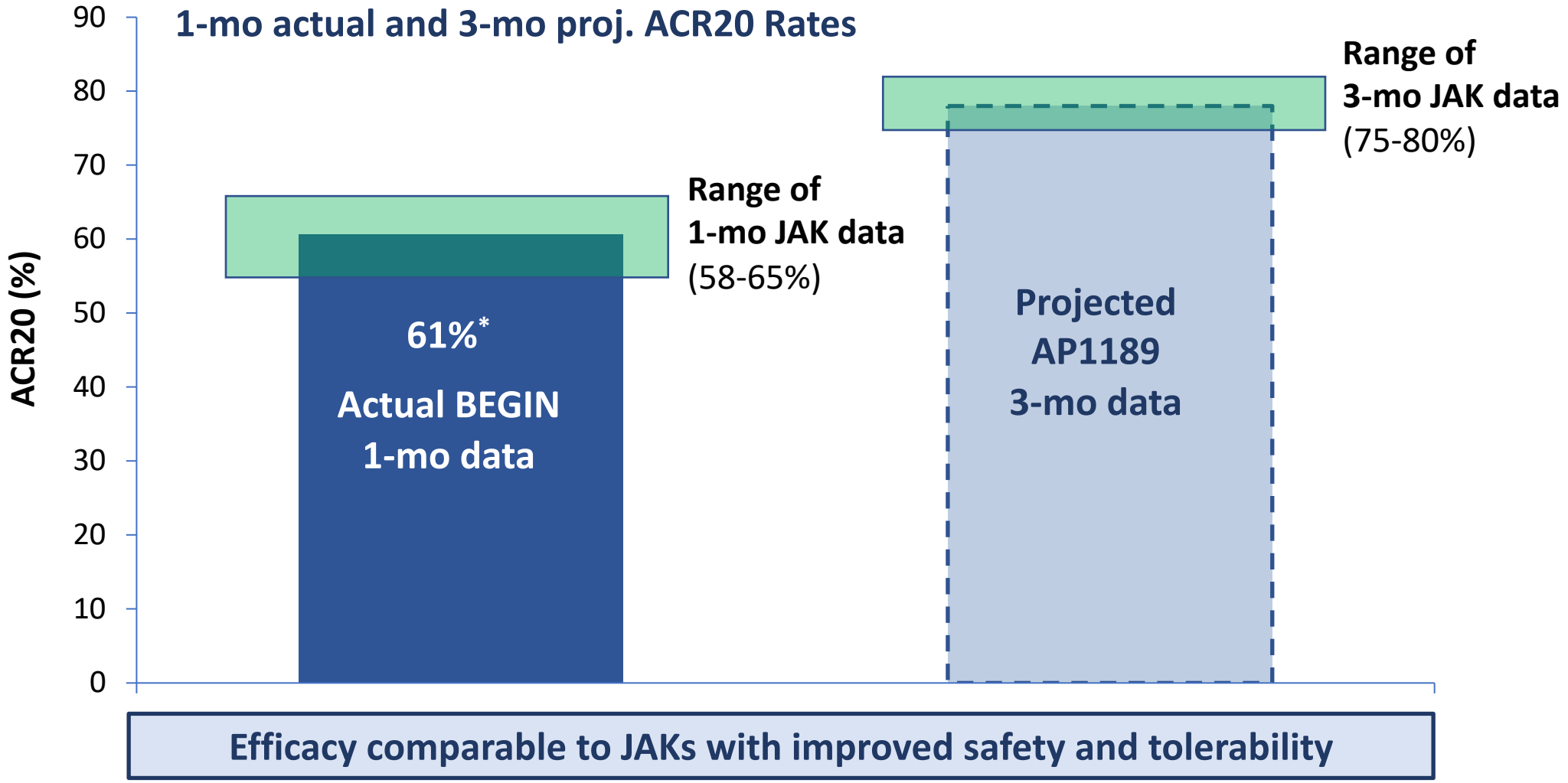
(+) Cella et. Al., J Rheumatol 2005;32:811–9

Primary safety endpoint: AEs

	Placebo (n=32)	AP1189 50 mg (n=31)	AP1189 100 mg (n=35)	Total (n=98)
SAEs, n (%)	0	0	0	0
AEs, n reported from baseline and on	21	38	27	86
AE severity (mild/moderate/severe)	18/2/1	26/12/0	22/5/0	66/19/1
Discontinuation due to IMP related AEs, n (%)	0	0	0	0
Discontinuation due to MTX related AEs, n (%)	1	0	0	1
AEs occurring in >5% patients:				
Nausea , numbers	4	3	4	11
Increase in amino transferases, numbers/clinical significant	3/2	6/2	0/0	9/4
Gastrointestinal AE´s other than nausea	2	3	7	12

- Nausea and other transient GI side effects are believed to be attributable to the suspension formulation - the new oral tablet under development has not elicited these effects to date
- AP1189 was safe and well tolerated with no SAEs, no discontinuations deemed due to study drug, no infections and no discernible impact on wbc count or vital signs

AP1189 100mg 1-mo ACR20 scores are comparable to JAKs ACR20 in MTX-Naïve P3 trials - Response rates should increase over time with 3-mo dosing as with JAKs



* = p<0.05 vs placebo

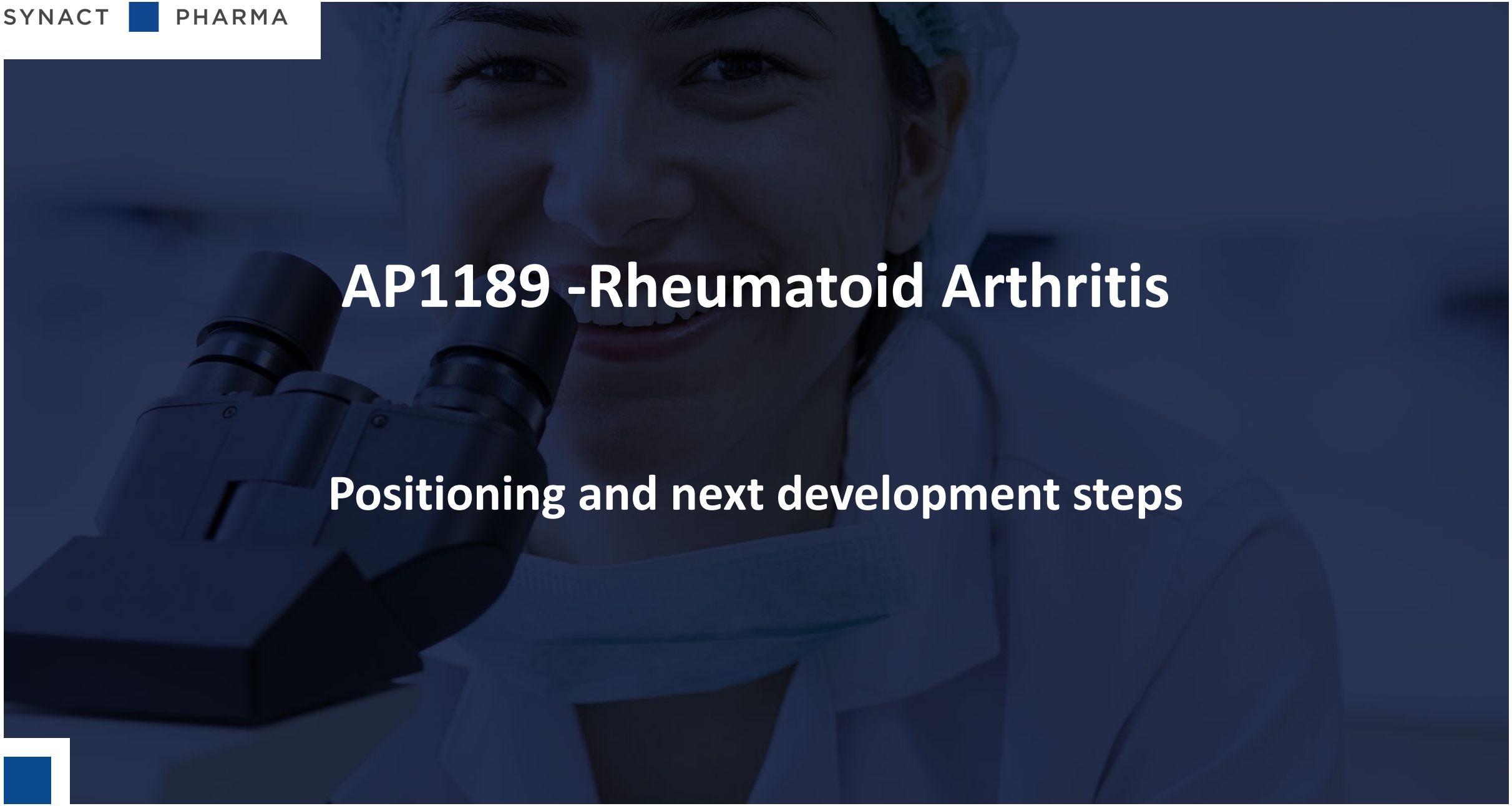
1) Lee et. Al., N Engl J Med 2014;370:2377-86.
2) Fleishman et. Al., Arthritis & Rheumatology, Vol. 69, No. 3, March 2017, pp 506–517
3) van Vollenhoven et. Al., Arthritis & Rheumatology, Vol. 72, No. 10, October 2020, pp 1607–1620
4) Westhovens et al. Ann Rheum Dis 2021;80:727–738.

BEGIN study summary

- In this 4-week study, patients treated once-daily with 100 mg AP1189 achieved a significantly greater reduction in mean Clinical Disease Activity Score (CDAI) as compared to placebo
- Change in disease activity from severe to moderate was numerically higher in groups treated with AP1189 as compared to placebo, with the lack of statistical difference likely explained by higher baseline CDAI and inflammation (C-reactive protein, CRP) in the 100 mg group
- While this was a relatively small trial, consistent dose-dependent effects were seen across mean CDAI change and all secondary read-outs including DAS-28, ACR score, investigators global disease assessment (VAS), FACIT-fatigue score and pain (VAS)
- AP1189 was well-tolerated and presented with a favorable safety profile with no serious adverse events were reported in the study
- We believe that the level of efficacy will increase with longer dosing duration and we will be assessing adding a higher dose group(s) in the ongoing P2 program

Based on the positive results, SynAct will seek scientific advice and open an IND with the FDA to prepare for Phase 2 trial in DMARD-IR patients to be initiated in 2022

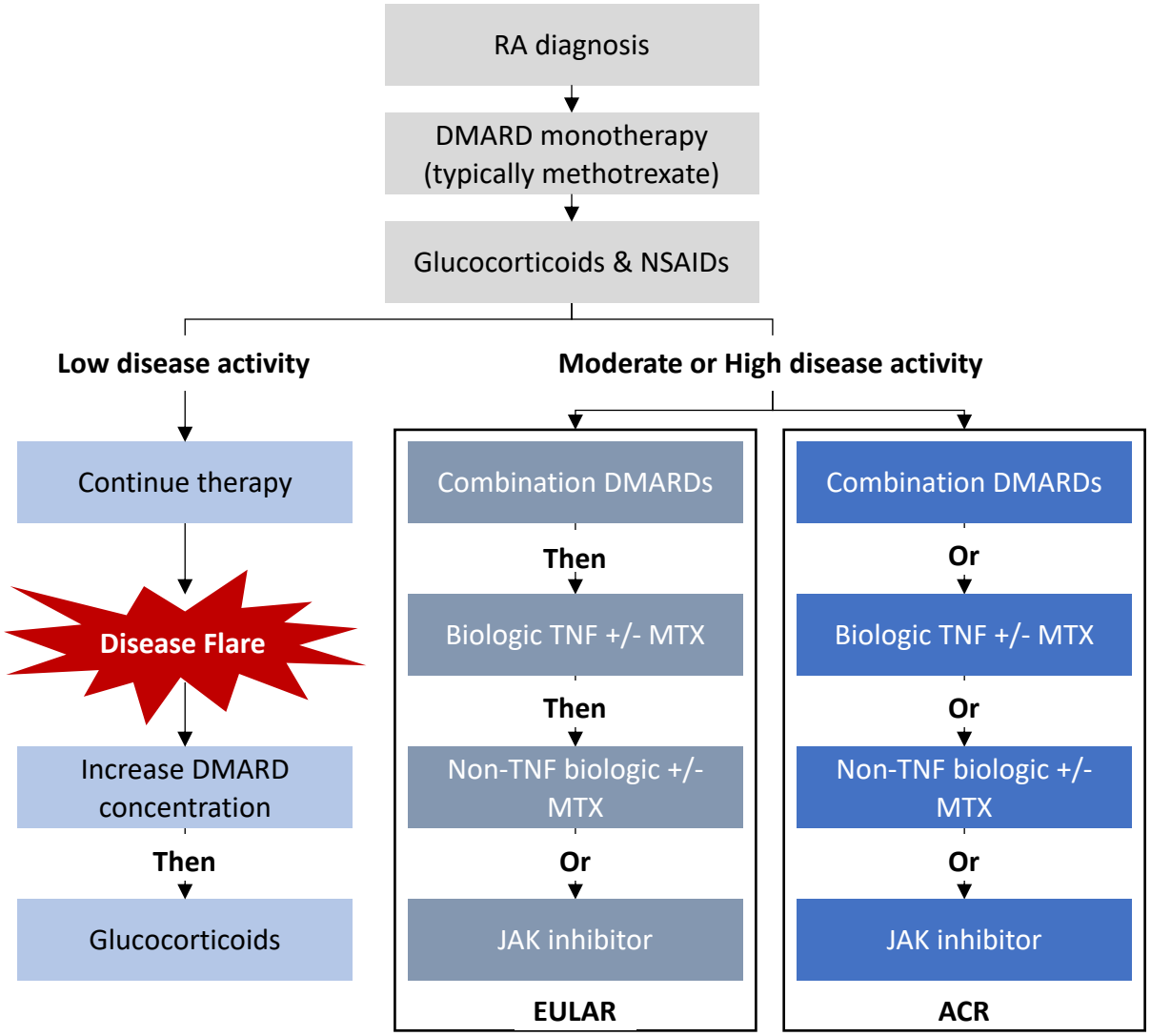
SYNACT PHARMA



AP1189 -Rheumatoid Arthritis

Positioning and next development steps

The current treatment paradigm for RA leaves significant potential for new meaningful therapies



Unmet need in the RA treatment paradigm

- Rheumatologists tend to treat aggressively with the variety of treatment options available, but RA treatment is still largely an art form
- DMARDs and biologics are well established in the treatment paradigm but don't work for all patients
- Despite a large variety of treatment options, remission remains elusive. In a study of 700+ RA patients followed for 3 years, 47% never achieved remission[1]
- The FDA has placed boxed warnings on the package inserts of JAK inhibitors over increased risks for serious opportunistic infections, cancer, and serious cardiovascular events
- Shortcomings of currently available treatment options create significant opportunity for new modalities that can safely treat RA

Treatment paradigm adapted from Global Data *RA: Global Drug Forecast and Market Analysis to 2027, 2019; (1) Byerk et al., J Rheumatol 2014;41:227-34*

AP1189 has the potential to be positioned for several potential uses in RA

Emerging AP1189 Clinical profile

- **Once-Daily Oral Dosing** – new oral solid formulation to be used in next clinical trial
- **Quick Onset of Action** – as early as days
- **Efficacy approaching JAKs** - within 4 weeks
- **Safe and Well Tolerated** - no emerging AEs and **No Immunosuppression**
- **Steroid-Free MoA** – potential to be steroid sparing
- **Compatible with MTX** – no known theoretical DMARD drug interactions

Multiple RA Positioning Opportunities

- **The emerging AP1189 clinical profile supports RA development at several treatment inflection points:**
  - **DMARD-IR -** patients who have had an incomplete response, lost response or are intolerant of DMARDs
  - **First line treatment in previous treatment naïve patients** – Newly diagnosed patients where the compound can be given in combination with DMARDs
  - **Flares** - Short-term use to treat for moderate or severe flares in patients who experience multiple flares per year

Rheumatologists expressed high degree of interest in AP1189 in DMARD-IR

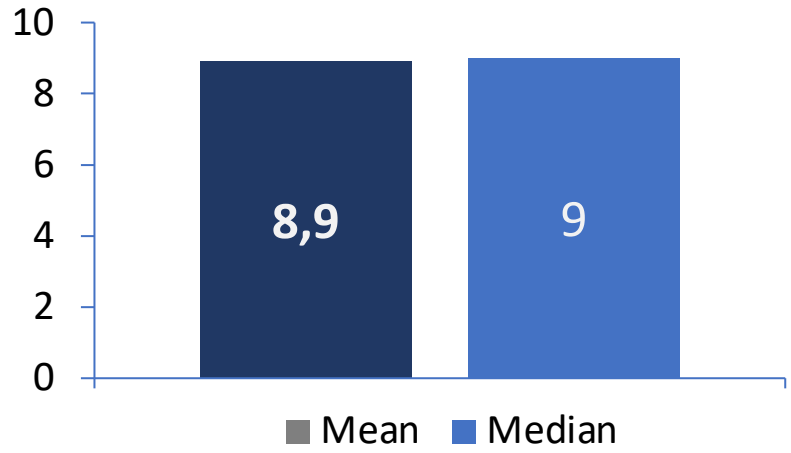
The combination of efficacy, safety, tolerability and oral convenience positions AP1189 well for

DMARD-IR Recent FDA labelling actions with JAKs in RA reduces direct competition in the space

SynAct Pharma has conducted a market research in high subscribing Rheumatologists to evaluate the potential of the drug in DMARD-IR

Rheumatologists support use in DMARD-IR and expressed a willingness to both use it earlier in select patients

Rheum Interest in AP1189 in DMARD-IR



- **Stated intent to use in 45% of DMARD-IR patients**
- ***"Oh man. I'd love to use this up front. I'd love to use it right after methotrexate. I'd love to use it before. I'd love to see this upfront. I mean the non immunosuppressive working kind of endogenously and not doing all the steroid evils, but almost kind of kicking butt like a steroid, uh, yeah, count me in for that one. . ."***

GlobalData RA Primary Research Project December 2021; 10 1:1 interviews with US high volume rheumatologists in December 2021
Y-axis is Intent to use of a 1-10 scale

High Volume US Rheumatologists Reacted Very Favorably to the AP1189 Profile

Respondents were all pleased that so many of their unmet needs were addressed with the AP1189 Profile:

Novel MOA ✓

Oral ROA ✓

Clean Safety Profile ✓

Potential to be combined with other advanced therapies ✓

We know from oncology…there's definitely benefit in combining things. And sometimes I get away with triple therapy myself and Plaquenil, MTX, and a TNF inhibitor, you know, there's some cocktail they're on that seems to provide a lot of relief with some patients. This seems very safe so, it'd be nice to swap out one of the oldies and put that in there - US08

Initial impression is very good...they've really narrowed down these specific melanocortin receptors so that's actually very, very exciting. -US02

I'd say rock on. I mean, this is pretty awesome. I like the fact that it's not immunosuppressive, it's working with the immune system. It's really producing some powerful ACR responses…So, I mean, it's very exciting. -US07

Wow. Okay. That's good. Okay. That sounds extremely interesting, very promising. The safety profile looks like a huge plus, that they're not seeing significant or severe adverse events. No opportunistic infection, no cancer, no cardiovascular. That would be a huge plus. Because I think the Jack inhibitors now, are being marketed now as patients need to fail a TNF inhibitor first. Yeah. So, if this drug is an oral drug with comparable efficacy, but better safety, then they should theoretically have a huge market. - US10

GlobalData RA Primary Research Project December 2021; 10 1:1 interviews with US high volume rheumatologists in December 2021

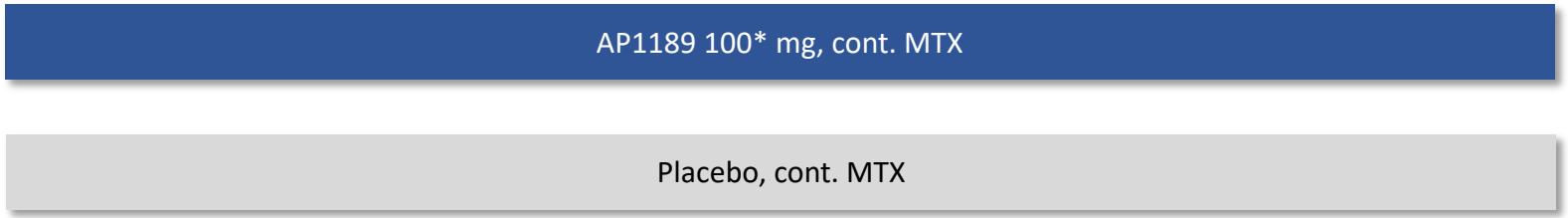
SYNACT PHARMA

Further Clinical Development in RA

EXPAND STUDY P2 study in previous treatment naive RA patients

Patient Population:

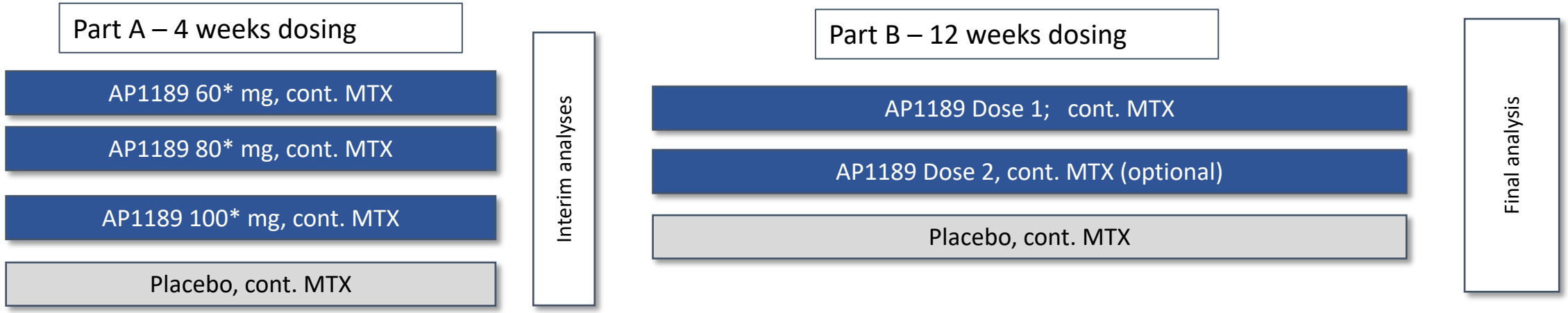
- Previous treatment naïve, eligible for initiation of DMARD treatment (MTX)
- CDAI >22 at baseline – min of 6 swollen and tender joints
- Rheuma factor positive



Final analysis

Key Proposed Study Parameters	
Dosing and Duration	▪ 12 weeks of once-daily dosing of solid tablet AP1189 or placebo
Study Size and Sites	▪ 60 patients per group for a total of 120 subjects
Primary Endpoints	▪ ACR20 response rate at 12 weeks as compared to placebo
Secondary Endpoints	▪ CDAI score ▪ DAS28 score ▪ FACIT-Fatigue ▪ HAQ/RAQol

AP1189- Proposed adaptive P2 trial design in DMARD-IR patients



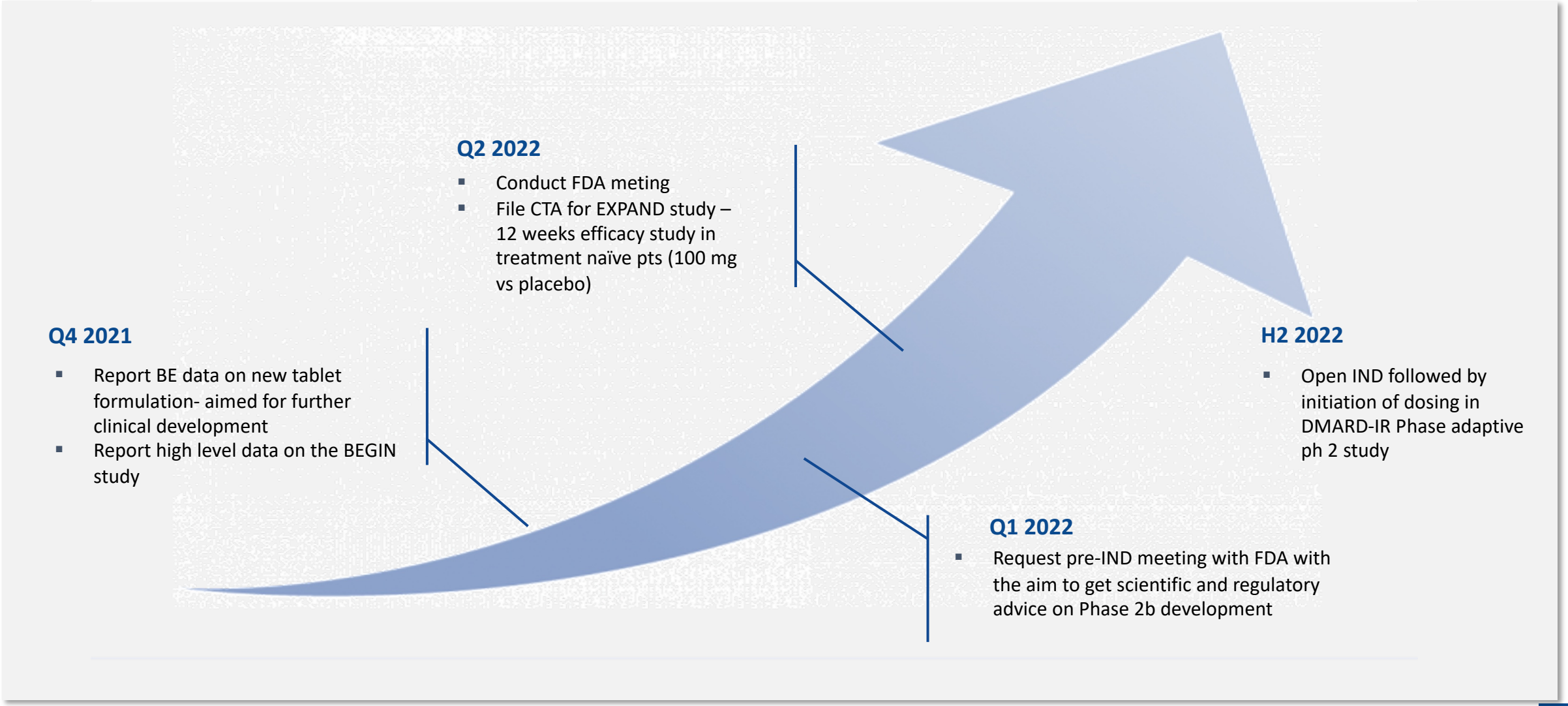
Patient Population:

- >3 mo MTX treatment with moderate to severe disease activity
- Documented incomplete response , loss of response or intolerance to MTX-
- Min of 6 swollen and 6 tender joints , increased CRP

Key Proposed Study Parameters	
Dosing	▪ Once-daily dosing of solid tablet AP1189 or placebo
Study Size and Sites	▪ Part A: 30 pts per group ▪ Part B: 75 patients per group
Primary Endpoints	▪ ACR20 response rate at 4 Weeks (part A) and 12 weeks as compared to placebo
Secondary Endpoints	▪ CDAI score; DAS28 score; FACIT-Fatigue; HAQ/RAQol

*:Free base – correspond to 75, 100 and 125 mg acetate salt used in the BEGIN study

AP1189 as novel compound treatment of Rheumatoid Arthritis- High level milestones for 2022



Important milestones 2022 and 2023

Kvartal 2 2022
- ❑ Rights issue
- ❑ Pre-IND
- ❑ Re-design NS
- ❑ Nasdaq uplift

Kvartal 3 2022
- ❑ IND
- ❑ Initiate EXPAND study

Kvartal 4 2022
- ❑ Initiate DMARD-IR

Kvartal 3 2023
- ❑ Topline results EXPAND
- ❑ Interimdata DMARD-IR part I

Kvartal 4 2023
- ❑ Data on NS

SYNACT PHARMA

AP1189 – Phase II Development in Nephrotic Syndrome

Positioning as alternative to immuno-suppressive Therapy

Nephrotic Syndrome

SYNACT PHARMA

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

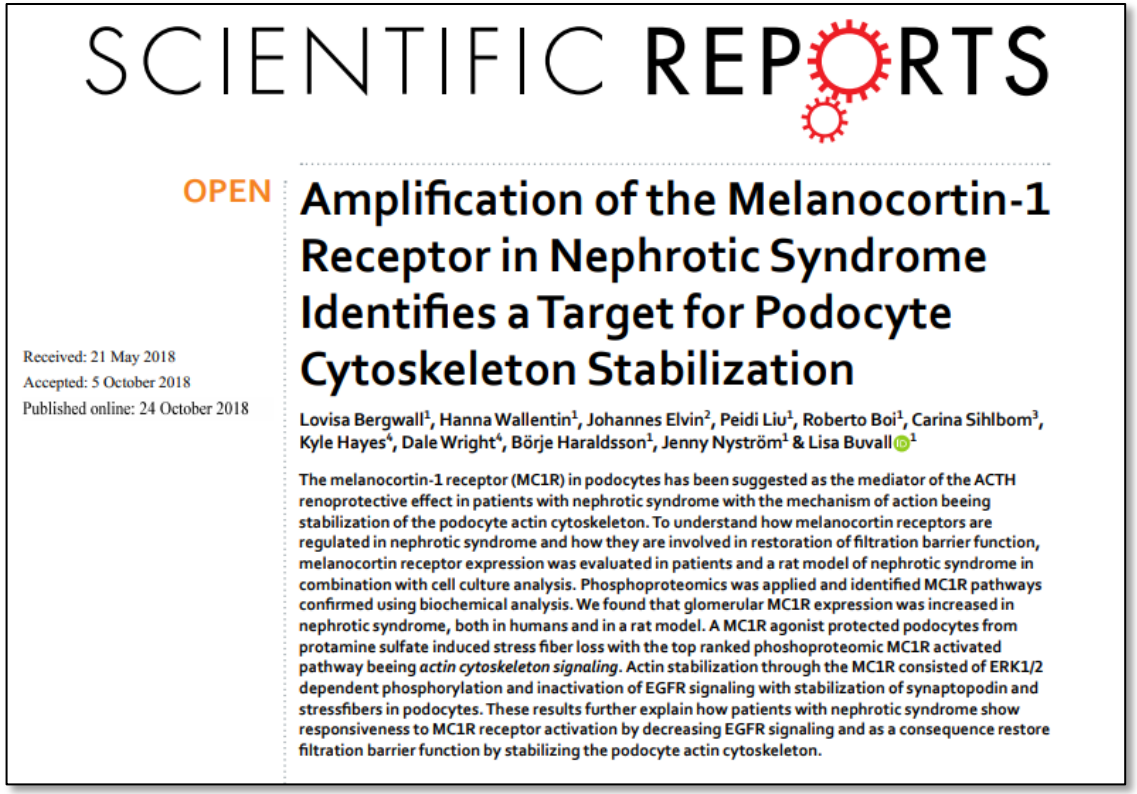
Unmet Need

- 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

SYNACT PHARMA



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

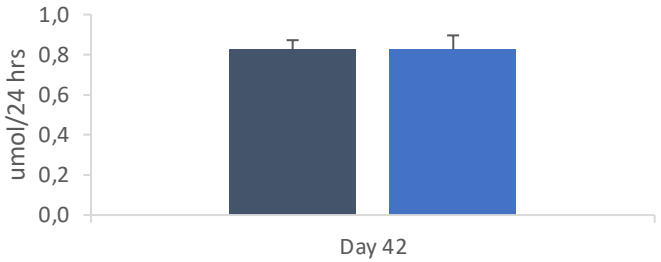
AP1189 Shows Benefits in Models of Nephritis

SYNACT PHARMA

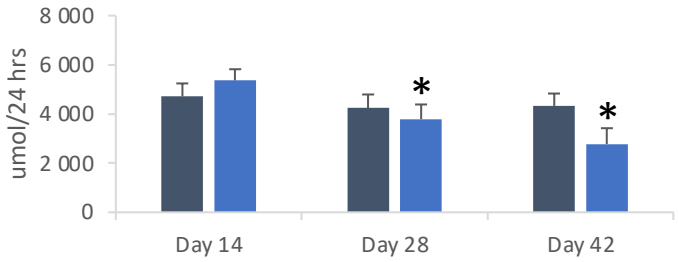
Membranous nephropathy rat model (passive Heymann Nephritis)

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

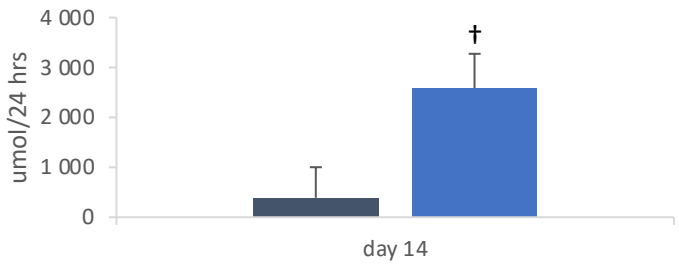
Creatinine Clearance (ml/min/g KW)



Albumin excretion rate



Reduction in AER (day 14-42)

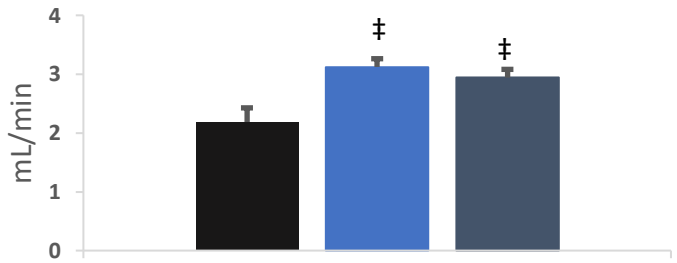


AP1189 showed no effect on creatinine clearance where albumin excretion decreased significantly

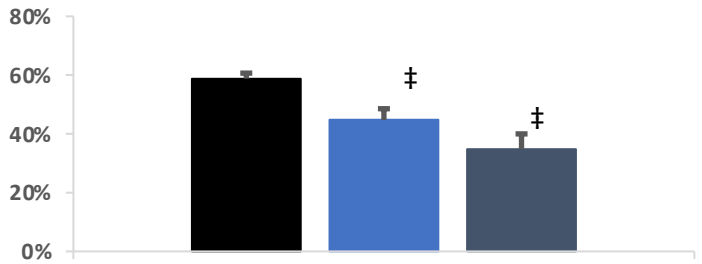
AP1189 vs ACTH in a model of Nephrotic Syndrome

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

Creatinine Clearance



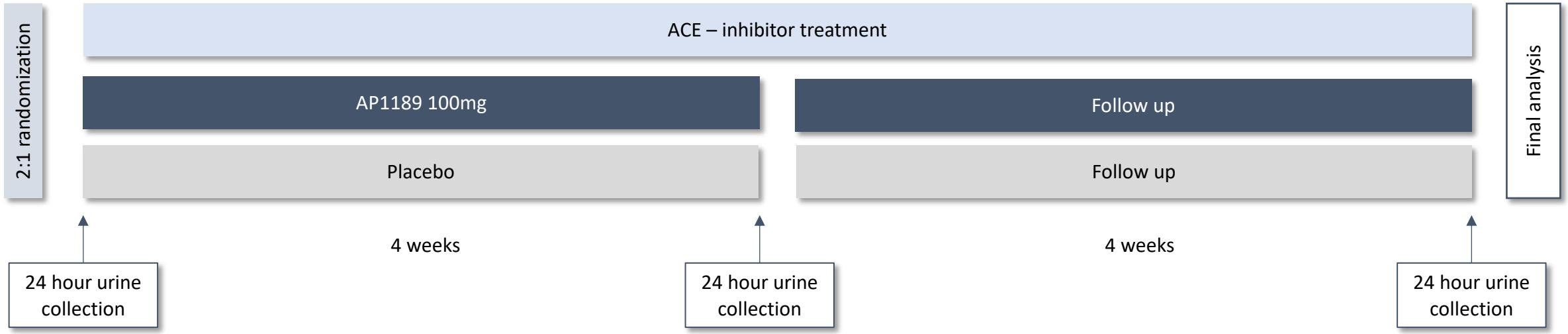
Fractional Albumin Excretion



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- - Revision aim to apply 12 weeks dosing with tablets is pending



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

SYNACT PHARMA

Leadership

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 and VP BD of Action Pharma

TXP pharma
action pharma
CLC bio
chemometec

Patrik Renblad, MSc – CFO

- >20 years' experience from finance roles in pharma
- Former head of R&D Finance at LEO Pharma
- Experience from divestments, acquisitions and licensing deals

LEO
AstraZeneca

Thomas Jonassen, MD – Co-founder and CSO

- Associate Professor, KU in Denmark
- Visiting Professor, WHRI, UK
- Co-founder of TXP Pharma and Resother
- Co-founder and former CSO of Action Pharma

TXP pharma
action pharma

Thomas Boesen, PhD – COO

- 20 years' experience in biotech and pharma industry
- Inventor of several new chemical entities
- Co-founder of TXP Pharma
- Former VP Discovery, Action Pharma

TXP pharma
action pharma
novo nordisk
EpiTherapeutics

James Knight, MBA – CBO

- >25 years' experience in biotech ranging from R&D through commercial strategy and business development at Biogen, Dura, Elan, Questcor and BioTime
- Formerly VP of Portfolio Strategy at Questcor overseeing expansion of Acthar®-promoted indications, growing sales from $110m to $1b

Biogen
QUESTCOR PHARMACEUTICALS, INC.

SynAct Pharma – Board of Directors

Torbjørn Bjerke, MD – Chairman

- >25 years track record from pharma industry as Head, R&D and CEO (private and public), BD
- Co-founder of Action Pharma, TXP Pharma, Arctic Aurora LifeScience and Biotech Select and Carelight Ltd
- Chairman TXP Pharma, Carelight Ltd

Thomas Jonassen, MD – Board Member, Founder

- Associate Professor, KU in Denmark
- Visiting Professor, WHRI, UK
- Co-founder of TXP Pharma and Resother
- Co-founder and former CSO of Action Pharma

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
- Board member of a number of European biotech companies

Terje Kelland, MD, PhD – Board Member

- >30 years of international experience from management positions in the life science industry.
- SVP at Novo Nordisk A/S, head of research and development at Biovitrum AB (now SOBI AB), and has held various positions within Pharmacia AB.

Uli Hacksell, PhD – Board Member

- Former CEO of Medivir
- Former CEO of Acadia Pharmaceuticals, taking it from private startup to multibillion USD public company
- Board member of many other life sciences companies

Marina Bozilenko, BA, MA – Independent Board Member

- 30 years of investment banking and other healthcare industry expertise, including raising >$30b in capital and executing numerous M&A transactions
- Strategic Advisor to William Blair & Company, having joined in 2010 as Head of Biotech & Pharma and Managing Director
- BA in molecular biology and MA in economic history from the University of Chicago

SynAct Pharma – Board of directors

Kerstin Hasselgren, MSc – Board Member

- CFO Xspray Pharma, NASDAQ Stockholm
- Former VP Corporate Business Control SSAB, CFO Alstom Transport Nordic, VP Finance Global Operations AstraZeneca and VP Finance Global FoU AstraZeneca.
- Board member since 2022