Innate Immunotherapeutics

ABN 16 165 160 841

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American Academy of Neurologists Posters and Key Opinion Leader Meeting Presentation

Innate Immunotherapeutics Limited (ASX Code: IIL), together with a number of Australian, Canadian, and Danish scientific collaborators presented five posters at last week's American Academy of Neurology (AAN) Annual Meeting held in Boston. These posters can be viewed at: www.innateimmuno.com/aanposters

In parallel with the AAN meeting the Company hosted a meeting of clinical and scientific key opinion leaders in the field of progressive multiple sclerosis to provide an update on the MIS416 clinical development programme and latest insights into the drug's mechanism of action. This presentation is attached.

- End

About Innate Immunotherapeutics

Innate Immunotherapeutics Limited is an Australian biotechnology company with a novel technology that targets the human innate immune system. The innate immune system is the body's first line of defence against external disease causing pathogens such as bacteria and viruses, and internally caused diseases such as cancer. While innate immunity is responsible for mounting these important and immediate defences, it also plays a critical role in controlling the overall immune response as well as many for the body's tissue protective and reparative functions. The Company's lead drug candidate MIS416 can trigger these anti-inflammatory and reparative functions inside the central nervous system. This makes MIS416 a highly relevant drug candidate for the treatment of secondary progressive multiple sclerosis and other neurological conditions where inflammation inside the CNS contributes to disease pathology.

About the Phase 2B trial of MIS416 in patients with secondary progressive multiple sclerosis

Innate Immunotherapeutics is sponsoring a 2:1 randomised, double-blind, placebo-controlled trial of the efficacy and safety of MIS416 in the treatment of subjects with the SPMS. The study enrolled 93 patients with progressing, non-relapsing SPMS, and who had an Expanded Disability Status Scale (EDSS) score between 3.0 and 6.5 at screening. Patients are receiving MIS416 or placebo once weekly for 52 weeks. The efficacy of MIS416 is being assessed using measures of neuromuscular function as well as several patient reported outcomes related to disability and health status.

MRI is also being used to assess the effects of treatment on whole brain atrophy and magnetization transfer ratio. More details are available at clinicaltrials.gov, trial identifier NCT02228213. The study completed in the clinic in April 2017 and a clinical study report is expected in late August or September 2017.

For Further Information
Simon Wilkinson, CEO
+64 21 661 850

www.innateimmuno.com

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A Phase 2 Company Treating Secondary Progressive Multiple Sclerosis

AAN Breakfast Briefing

Innate Immunotherapeutics

Forward Looking Statements

This Presentation (and any financial information that may be provided by Innate Immunotherapeutics Limited - the "Company") may contain forward looking statements that involve risks and uncertainties. Such statements include statements regarding the Company's belief or current expectation and are necessarily based on the Company's current understanding of the markets and industries in which it operates. That understanding could change or could prove to be inconsistent with actual developments. The Company's actual results could differ materially from the results discussed in this Presentation, including those anticipated in or implied by any forward looking statements.

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AAN breakfast briefing for KOLs

7.15 Welcome and Introductions

7.20 Brief background and clinical experience to date

7.35 Patient case studies

7:50 Mechanism of action

8.10 Discussion

8.25 Close

Innate Immunotherapeutics

Welcome and introductions

- Innate Immunotherapeutics is a New Zealand based / Australian listed biotech with a drug candidate for non-relapsing secondary progressive multiple sclerosis
- A 90 patient double blinded placebo controlled Phase 2 efficacy trial of MIS416 completed in the clinic on 20 April 2017 with a study report due in September ‘17
- In anticipation of a potentially favourable outcome, Innate would like to take this opportunity to raise awareness of the programme among SPMS clinical and scientific key opinion leaders
- Representing Innate today …
  - Robert Peach, Independent Director (formerly CSO Receptos)
  - Michael Silverman, Clinical Development Consultant
  - Gill Webster, CSO
  - Janette Dixon, VP Corporate Development
  - Simon Wilkinson, Managing Director & CEO

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Brief background

2005 – 2007	Preclinical safety and efficacy evaluations of MIS416 as a standalone, systemically delivered innate immune stimulator
Oct 2008	SPMS 'Patient Zero' – 58 yrs female EDSS 8.0 with rapidly progressing disease approached Innate for compassionate access to MIS416
2009	Five further patients (SPMS=4, PPMS=1) access MIS416. Five of six report various improvements after ~6 months of once weekly MIS416
May 2010	Fast Forward (NMSS) and NZ Govt approve $1m funding for Phase 1
Oct 2010	Medsafe waives Phase 1A. Phase 1B dose escalation study commences
2012	Phase 2A dose confirmation study completed
2012 – present	Compassionate use programme expanded to include 1B/2A patients
Oct 2014	Phase 2B SPMS study commences
April 2017	Last Patient Last Visit
Ongoing	~90% of patients who completed 12 month 2B study have requested / gained access to MIS416 on a special access scheme basis

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Clinical programme - Phase 1B/2A (completed)

Study Design	
Phase	1B/2A (NCT01191996)
Design	Open-label, non-randomized, safety study conducted in two phases: - Dose escalation (DE) - Dose confirmation (DC)
NZ Sites	1 (Christchurch, NZ)
N	16 of 19 completed the 4 week Phase 1B dose escalation study 11 of 15 completed the 12 week Phase 2A dose confirmation study
Doses	DE: 4 once weekly IV doses ranging from 125 µg (cohort 1) to 600 µg (cohort 4) DC: 12 once weekly IV doses. Dose 1=125 µg, dose 2=250 µg, dose 3 onwards = 500 µg
Primary Endpoints	Safety and tolerability, dose-limiting toxicities, maximum tolerated dose, recommended Phase 2 dose, pharmacodynamics
Secondary Endpoints	MSFC, FSS, SF-36, EDSS, MRI

Key Objectives

- To determine safety and tolerability
- To determine the recommended Phase 2 dose

Outcomes

- Appears safe and well tolerated at doses of 125ug-600ug. RP2D = 500ug
- Most common AEs – pyrexia, headache, fatigue, myalgia and muscle stiffness, were expected/transient
- Interferon-inducible proteins and soluble adhesion molecules are up-regulated in the absence of a detectable systemic inflammatory response
- In patients receiving all doses 80% of subjects showed >20% improvement in at least 1 measure of clinical status
- In patients receiving all doses no patients appeared to experience progression or exacerbations

Luckey, A. M., et al. (2014). "Safety, tolerability and pharmacodynamics of a novel immunomodulator, MIS416, in patients with chronic progressive multiple sclerosis" Multiple Sclerosis Journal - Experimental, Translational and Clinical **1**: 1-12.

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Phase 2B trial – MIS416-202 (results pending)

Study Design	
Phase	2B (NCT02228213)
Design	Randomized, double-blind, placebo-controlled study of the efficacy and safety of MIS416 in the treatment of subjects with SPMS Eligibility criteria includes EDSS 3.0 to 6.5, relapse free for 2yrs, clinical evidence of progression
Sites	5 x Australia and 2 x New Zealand
N	93 subjects with lapse-free SPMS randomized 2:1 to MIS416 or saline placebo
Doses	Weekly i.v. injection of MIS416 or saline over 13 cycles of 4 doses per cycle (52 weeks), titrated at 125ug of MIS416 for the first dose, 250ug for the second dose, and 500ug for subsequent doses

Key Objectives

- To determine the efficacy of MIS416, relative to placebo, as assessed by various measures of neuromuscular function
- To explore the effect of MIS416 on disease activity & neurodegeneration by measuring a wide range of blood markers, imaging markers and patient reported outcomes
- Study is exploratory by design in preparation for phase 3
- Multiplicity of outcomes will be controlled for (linear step-up BH procedure) along with assessing the consistency of results such that decisions are not made totally on the basis of p-values

Status

- First patient in October 2014
- Last patient out 20 April 2017
- Clinical study report Q3 2017

Phase 2B trial – efficacy parameters

Neuromuscular Function: will be assessed on a three monthly basis and include;

- MS Function Composite (MSFC), comprising the; timed 25 Foot Walk, 9 Hole Peg Test, and Paced Auditory Serial Addition Test;
- Symbol digit modalities test (SDMT) [*potential replacement for PASAT in MSFC*];
- Sloan low-contrast letter visual acuity (SLCVA) [*possible addition to MSFC*];
- Jebsen Hand Function Test (JHFT) [*standardized and objective evaluation of fine and gross motor hand function using simulated activities of daily living*];
- Grip, tip and key pinch strength [*as type of pathology weakness is mainly pyramidal tract dysfunction, hand grip might be an easy to perform and very reproducible muscle strength test*]:
- 6-minute walk test (6MWT) [*potential objective measure of fatigue*];

Disability and Health Status: will be assessed on a three monthly basis and include;

- Expanded Disability Status Scale
- Patient Reported Outcomes including;
  - SF-36 and its components;
  - MS Impact Scale (MSIS-29);
  - Neurological Fatigue Index for MS (NFI-MS);
  - Brief Pain Inventory (BPI).

Cranial MRI: will be performed at Baseline, 3 months, and End of Study Visit to assess;

- Whole Brain Atrophy (WBA) and Magnetisation Transfer Ratio (MTR).

Phase 2B trial advisors

- **Clinical:** **Dr Jeffrey Cohen**, Director, Experimental Therapeutics Program, Mellen Center for MS Treatment & Research, Cleveland Clinic
- **Patient Reported Outcomes:** **Deborah Miller**, PhD, Mellen Center for Multiple Sclerosis Treatment and Research, Cleveland Clinic
- **Neuromuscular Assessments:** **Kristy Rose**, PhD, Institute for Neuroscience and Muscle Research, The Children's Hospital at Westmead (Sydney)
- **MRI:** **Dr Doug Arnold**, Neurology Professor, McGill University, Montreal Neurological Institute
- **Statistics:** **Gary Cutter**, PhD, Professor of Biostatistics and Head of the Section on Research Methods and Clinical Trials, UAB School of Public Health

Case Studies

(incorporating patient responder analysis performed by Prof. Nancy E Mayo, Department of Medicine, McGill University – See AAN poster P5.362)

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Responder ranking of Phase 2A subjects

Table 4: Number of Responses on PerfROs and PROs

	PerfRO	PRO	Total	Rank	Subsequently joined the compassionate use program
DC01	3	2	**5**	1	✓
DC05	2	2	**4**	2	✓
DC12	3	0	**3**	3	
DC10	2	1	**3**	4	✓
DC11	1	2	**3**	5	✓
DC07	2	0	**2**	6	✓
DC09	1	1	**2**	7	✓
DC06	0	2	**2**	8	✓
DC02	0	1	**1**	9.5	
DC03	0	1	**1**	9.5	✓
DC14	0	0	**0**	11	

Table 4 gives the number of outcomes on which each person was classified as a responder ranked in order by total number of responding outcomes. Seven persons responded on PerfROs and 8 on PROs; all but one person (DC14) showed a response on at least one variable. The ranking of subjects was based on total number of responses with priority given to response on perfROs.

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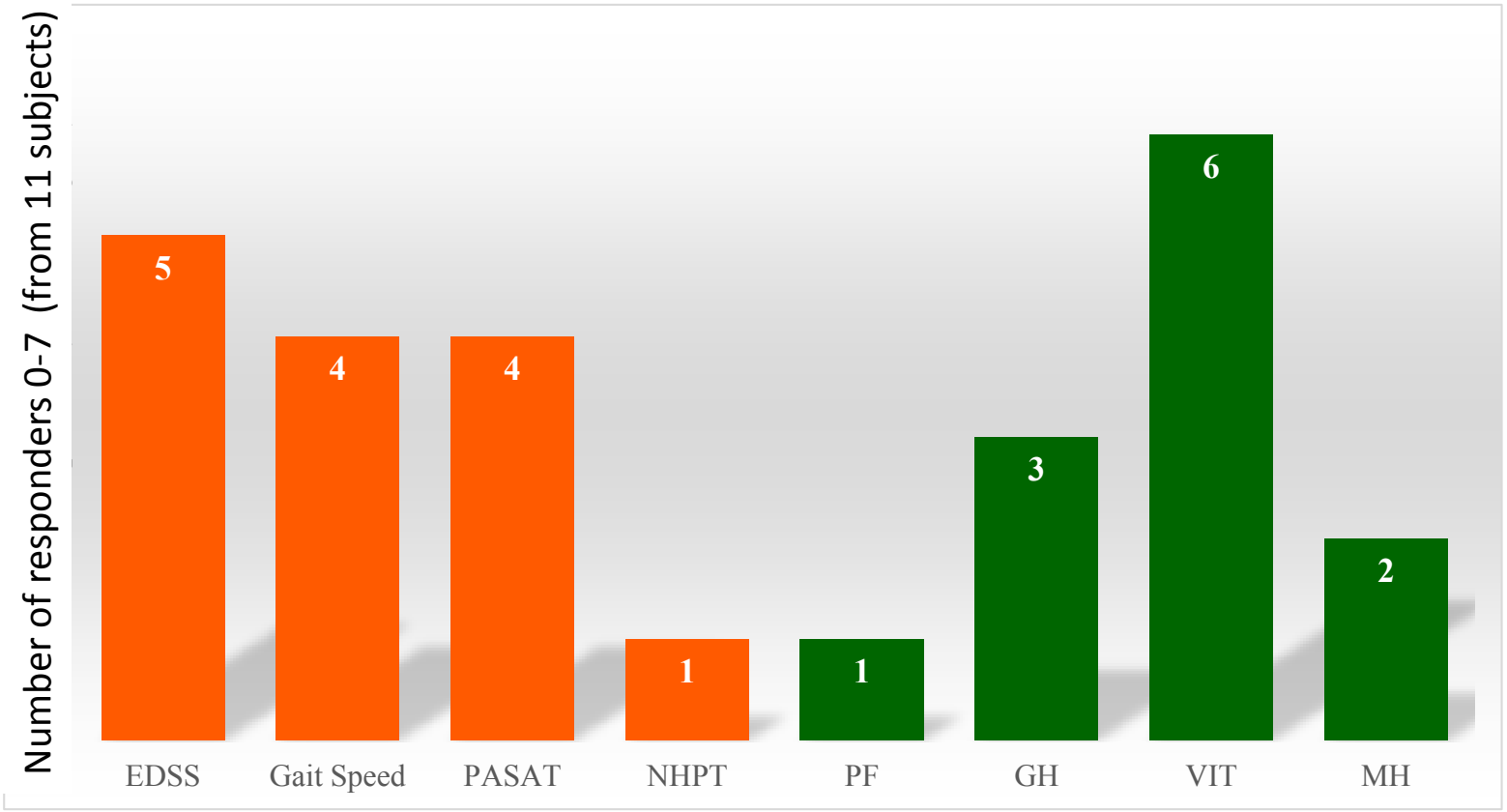
Phase 2A– 'Responders' based on PerfROs and PROs MIC



Responders were identified relative to Minimally Important Change (MIC) determined by Professor Mayo on the following basis:

Gait speed: ≥0.10 m/sec; NHPT: ≥20% in speed; PASAT: > 9 error improvement; EDSS: ≥ 0.5 reduction in score;

SF-36: 10 point change in raw score in any separate domain (not summaries PCS or MCS)

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Case study #2 – DC03

Status on enrolment: Subject suffered from extreme fatigue and spent most of the day in bed. Too tired to cook and rarely went out. She needed help with showering and a support worker would cook, do washing and assist with dressing. She had extremely sensitive skin, constant chronic headaches and body pain (taking gabapentin, codeine, Panadol). She was able to stand and take some steps, but it was painful and falls were frequent. She had constant bladder urging, about 5 UTIs per year and poor thermoregulation.

65Y

RS Rank: 9.5/11

Change and Responder Status (RS) on EDSS and Performance Rated Outcomes (PerfRO)

ID	EDSS		Gait Speed		PASAT		NHPT	
	Change	RS	Change	RS	Change	RS	% Change	RS
DC03	-0.5	0	-0.17	0	-2	0	-4.81	0

Change and Responder Status (RS) on Patient Reported Outcomes (PRO)

ID	PF		GH		VIT		MH	
	Change	RS	Change	RS	Change	RS	% Change	RS
DC03	0	0	12.9	1	-2.9	0	2.6	0

EDSS 6

No. months on drug:
40+

No. of doses to date:
approx. 130+

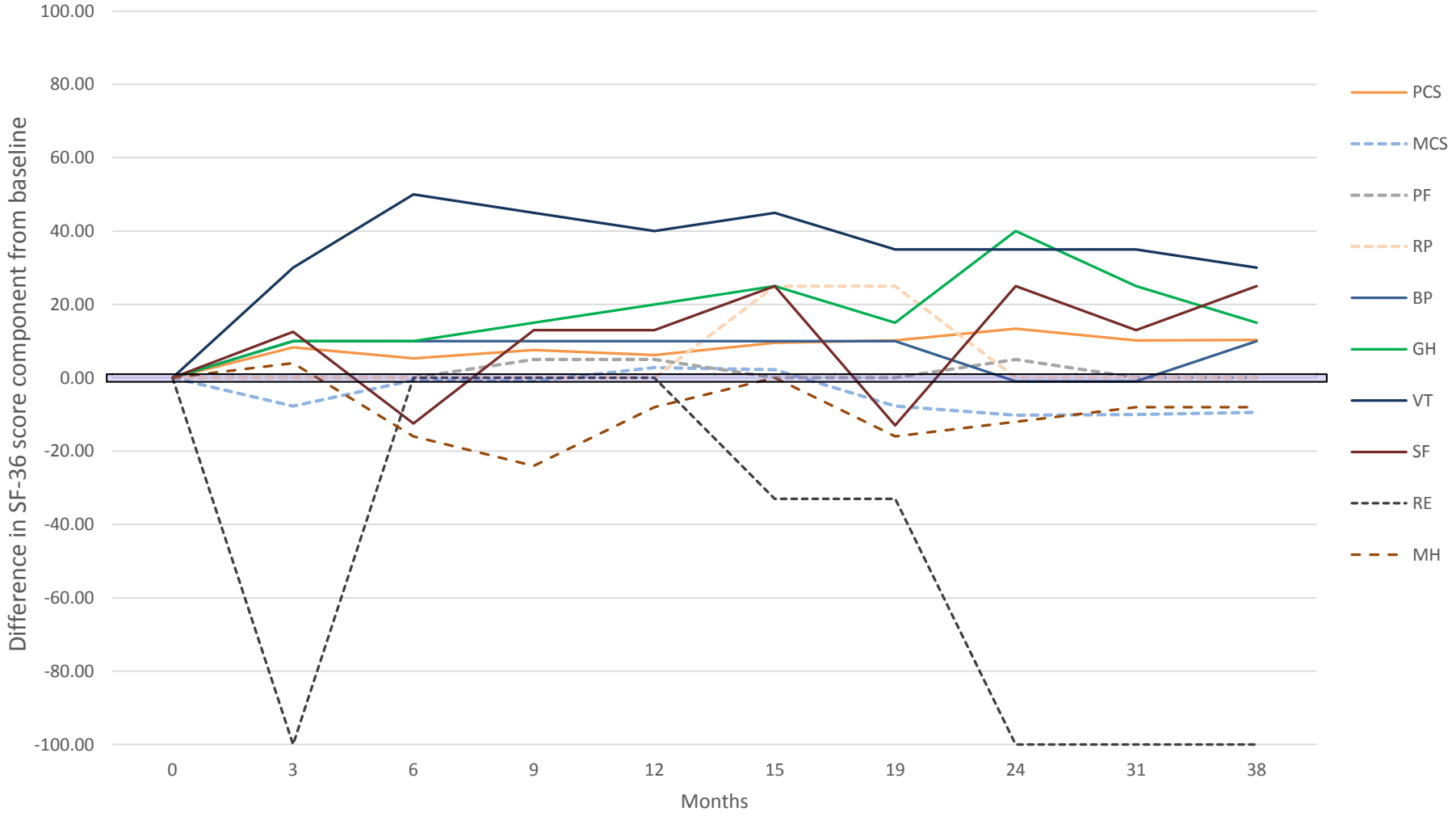
Dosing:
500ug fortnightly

Response to 2A study: No real response until week 7 when subject began to feel improvement in fatigue – more energy, less need for sleep, better ability to stand. This was followed by a reduction in bladder urging and she had no UTIs during the 12 week trial.

Time off drug: Within one month the subject had lost all improvements. She was sleeping all day, had constant headaches, bladder urging and weakness in legs. Patient reported that the extreme fatigue was the worst.

Compassionate use experience: *"Your whole life opens up, you cook better, eat better and socialize better".* No noticeable difference until dose 4 when walking improved. By dose 6 fatigue had improved greatly and subject was able to work in her garden. She now showers herself, shops and cooks, can hang out washing and dye her own hair. Naps are seldom needed, bladder urging and UTIs improved but eventually a suprapubic catheter was installed to facilitate travel, allowing the subject to travel to Brisbane to visit grandchildren. Headaches, whole body aching and oversensitive skin have all ceased and medication has reduced. Thermoregulation has greatly improved. After one year subject lost ability to walk but has maintained all other benefits. Upper body strength and dexterity are much improved, balance improved, transferring to her car is much easier.

Compassionate use program
SF-36 differences from baseline - Patient DC03



Notes:
1. Components with solid lines represent clinically significant difference from baseline for majority of time points
2. Components with dashed lines do not represent a clinically significant difference from baseline for majority of time points
3. Clinically significant difference for each domain (at the individual level to a 95% confidence level) as suggested in: Maruish, M. E. (Ed.). User's manual for the SF-36v2 Health Survey (3rd ed.). Lincoln, RI: QualityMetric Incorporated

PCS = Physical Component Summary
MCS = Mental Component Summary
PF = Physical Functioning
SF = Social Functioning
RP = Role-Physical
RE = Role-Emotional
BP = Bodily Pain
VT = Vitality
GH = General Health
MH = Mental Health

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Case study #1 – DC11

Status on enrolment: Subject suffered extreme fatigue, sleeping about 18 hours per day. Poor hand/eye coordination made driving his electric wheelchair difficult. He had substantial visual distortion and poor thermoregulation. Poor cognitive function meant he rarely socialized due to the embarrassment of forgetting the thread of a sentence or people's names. Subject also suffered spasticity in his legs, a useless clawed hand and severe pain in his tailbone requiring various medications. He could walk 2m holding onto a wheelchair.

56Y

RS Rank: 5/11

Change and Responder Status (RS) on EDSS and Performance Rated Outcomes (PerfRO)

ID	EDSS		Gait Speed		PASAT		NHPT	
	Change	RS	Change	RS	Change	RS	% Change	RS
DC11	0	0	.	.	11	1	-13.79	0

Change and Responder Status (RS) on Patient Reported Outcomes (PRO)

ID	PF		GH		VIT		MH	
	Change	RS	Change	RS	Change	RS	% Change	RS
DC11	7.6	0	14.3	1	11.9	1	5.3	0

EDSS 7

No. months on drug:
40+

No. of doses to date:
148+

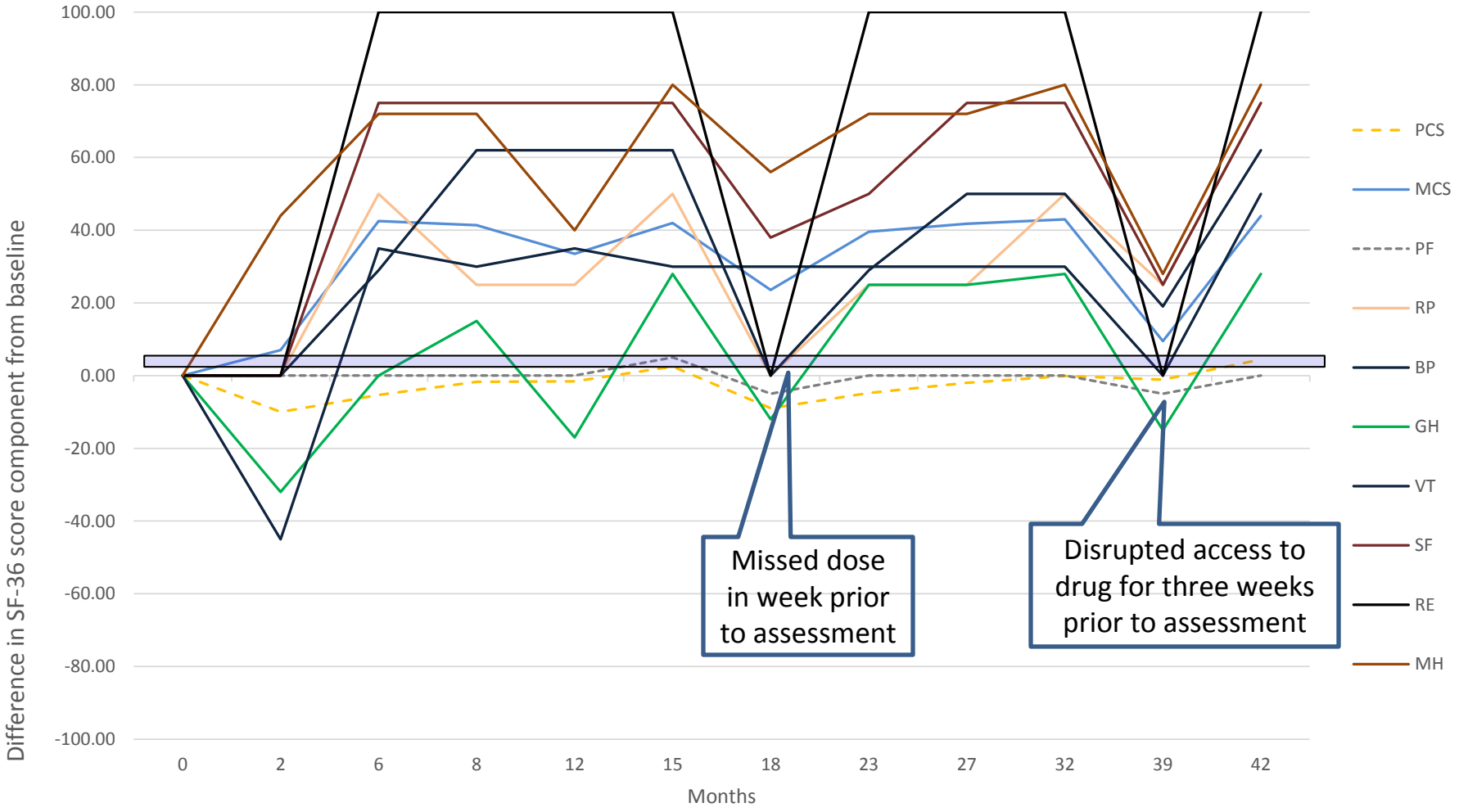
Dosing:
500ug weekly

Response to 2A study: Greatly improved vitality, significant reduction in pain, clear improvement in visual disturbances, enhancement in cognitive function, sleeping reduced to 9 hours per day, regained fine motor skills in upper body and was able to make better use of his mobility scooter. *"Felt pretty good, like there was light at the end of the tunnel, like the Primorus trial saved my life".* His clawed hand opened up and he could touch all fingers with his thumb, and his leg spasticity and jerking reduced. Could walk 12m holding onto chair.

Time off drug: Within two weeks he lost the ability to stand and walk. He also lost hand/eye coordination making it difficult to drive his wheelchair. His cognitive function deteriorated markedly and he stopped socialising. He lived in a deteriorating relationship with his wife and son, saw no one and slept all day. He washed from a basin, unable to stand to shower.

Compassionate use experience: Back on drug *"my brain lit up again".* Energy and cognitive function improved so much that the subject organized on his own to leave his wife and moved into respite care with independent living. Hand/eye coordination and thermo-regulation improved. He is out and about in his wheelchair visiting friends, has a new girlfriend, is able to shower and dress with minimal help. His hand is mostly unclawed and for some time he was able to change his wheelchair tyres himself.

Compassionate use program
SF-36 differences from baseline - Patient DC11



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MIS416 and Mechanism of Action

MIS416 targets innate sterile CNS inflammation and restores CNS homeostasis

Myeloid cells are an important target in progressive MS

- Relapsing remitting MS has both autoimmune and peripherally derived inflammatory components whereas progressive forms of MS reflect a clear shift towards innate inflammation being a significant cause of continuing neuro-degeneration
- Gandhi, Laroni, and Weiner conclude in their 2010 paper that:

  "Until now, there are no specific therapies to target innate immune cells in MS. As the role of innate immune system in MS becomes better defined, it will be possible to design therapy to transform immuno-pathogenic innate immune cells to a more immuno-regulatory innate immune cells."

NIH Public Access
Author Manuscript
J Neuroimmunol. Author manuscript; available in PMC 2011 April 15.

Published in final edited form as:
J Neuroimmunol. 2010 April 15; 221(1-2): 7–14. doi:10.1016/j.jneuroim.2009.10.015.

Role of the innate immune system in the pathogenesis of multiple sclerosis

Roopali Gandhi[*], **Alice Laroni**[*], and **Howard L. Weiner**
[1]Center for Neurologic Diseases, Brigham and Women's Hospital, 77 Avenue Louis Pasteur, NRB641, Boston, MA 02115

- Myeloid cells have been identified as a significant potential therapeutic target in SPMS as they have natural anti-inflammatory activities that are critical to innate immune homeostasis as well as stimulating wound healing/tissue repair pathways that are established following injury or infection

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Drug candidate MIS416 – a myeloid directed immune modulator

- MIS416 is a suspension of intact, heat killed and extracted Propionibacterium acnes
- Unwanted pro-inflammatory ligands that target cell surface TLRs as well as antigens are removed
- Retains naturally occurring bacterial ligands for TLR9 and NOD2 pathogen recognition receptors
- Size and shape means that only phagocytic myeloid cells are able to take up MIS416
- MIS416 is functionally inert until biodegraded intracellularly following phagosomal uptake
- MIS416 targeted myeloid cells can enter the CNS using homeostatic trafficking routes which are distinct to the blood brain barrier (e.g. choroid plexus)

Cartoon of MIS416 – retains size and shape characteristics of *p.acnes*

TEM images of MIS416

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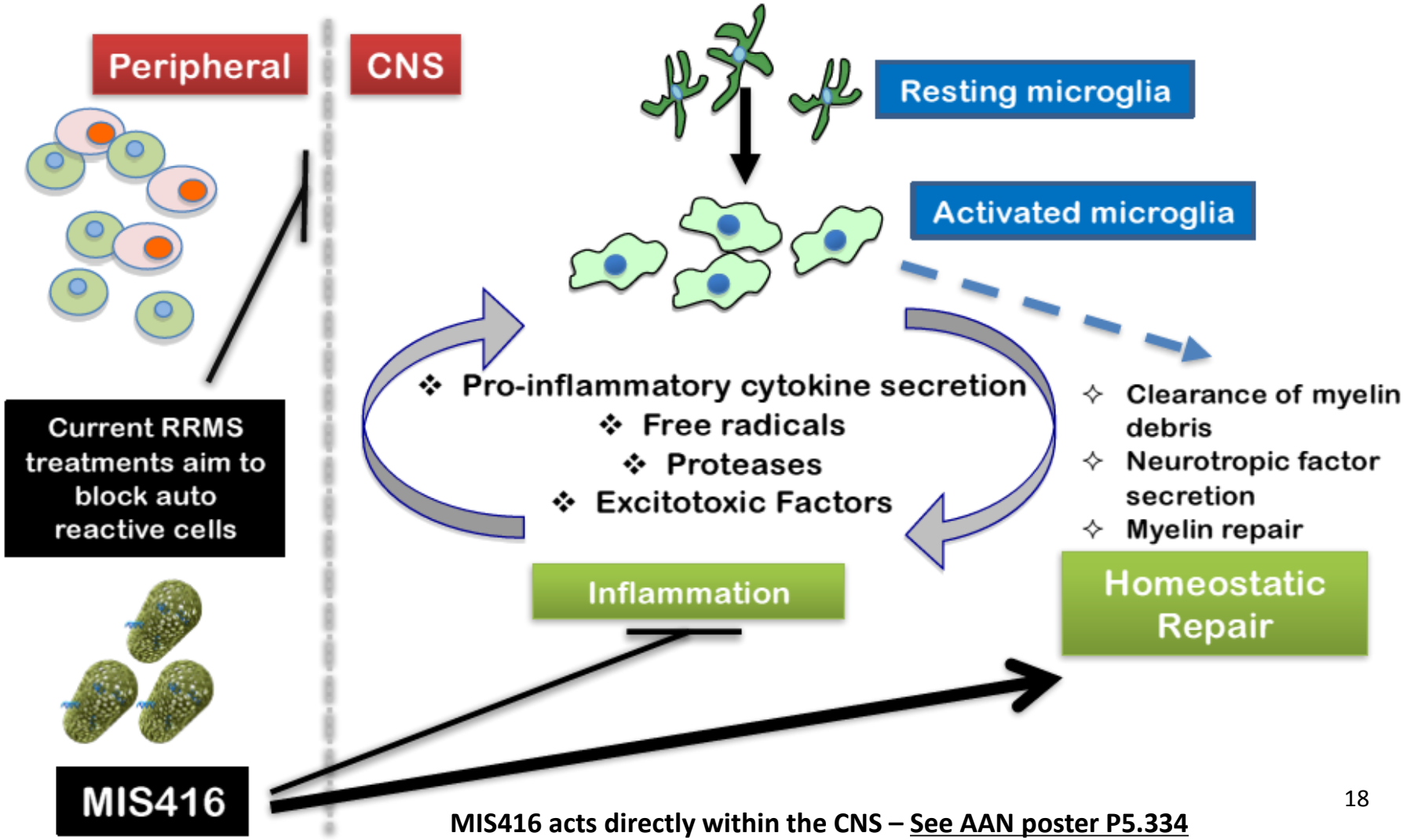
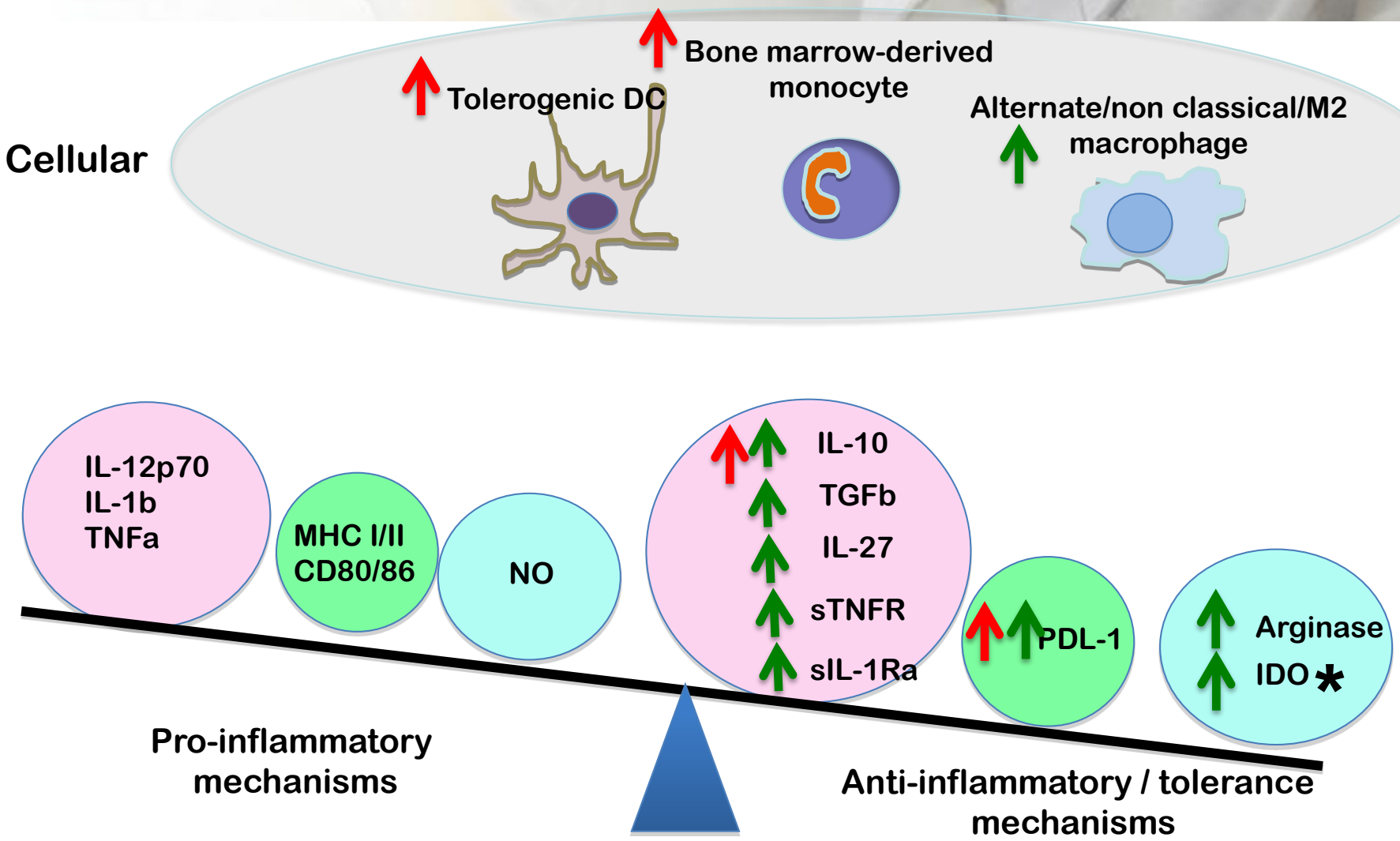
MIS416 targets sterile CNS inflammation and enhances endogenous myelin repair



MIS416 acts directly within the CNS – See AAN poster P5.334

Summary of MIS416-induced myeloid cellular and soluble pathways that can counteract inflammation



Upregulation following MIS416 treatment:
- ↑ in Mouse
- ↑ in Human

* See AAN poster P2.356

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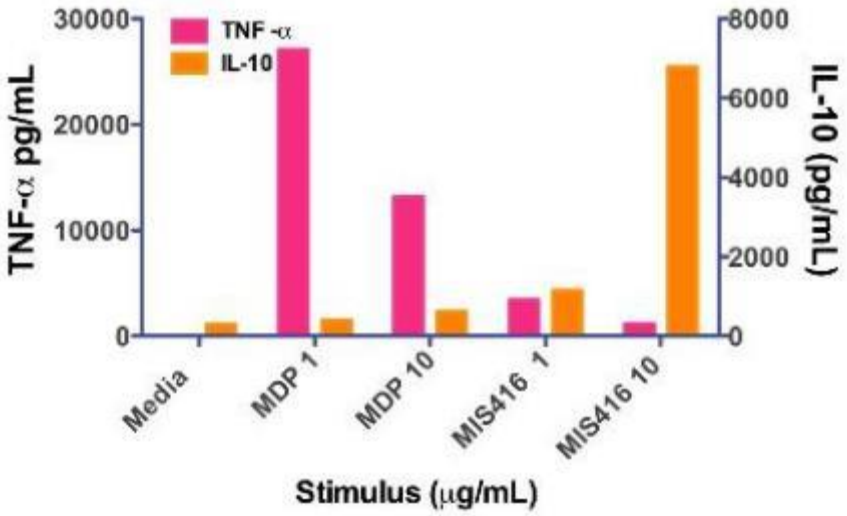
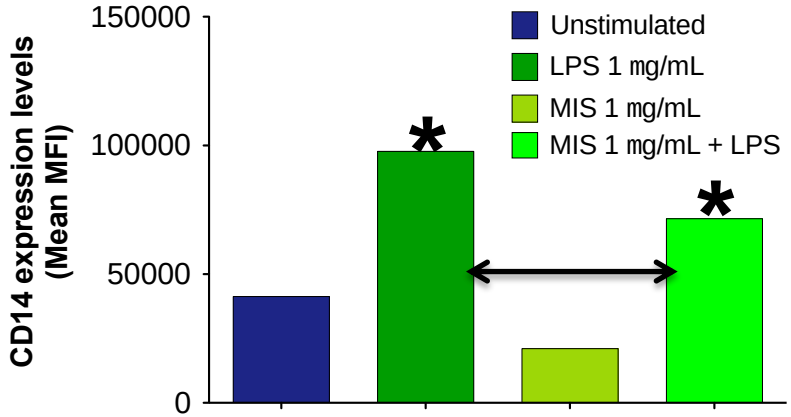
MIS416 up regulates IL-10 in human monocyte assays & patient plasma

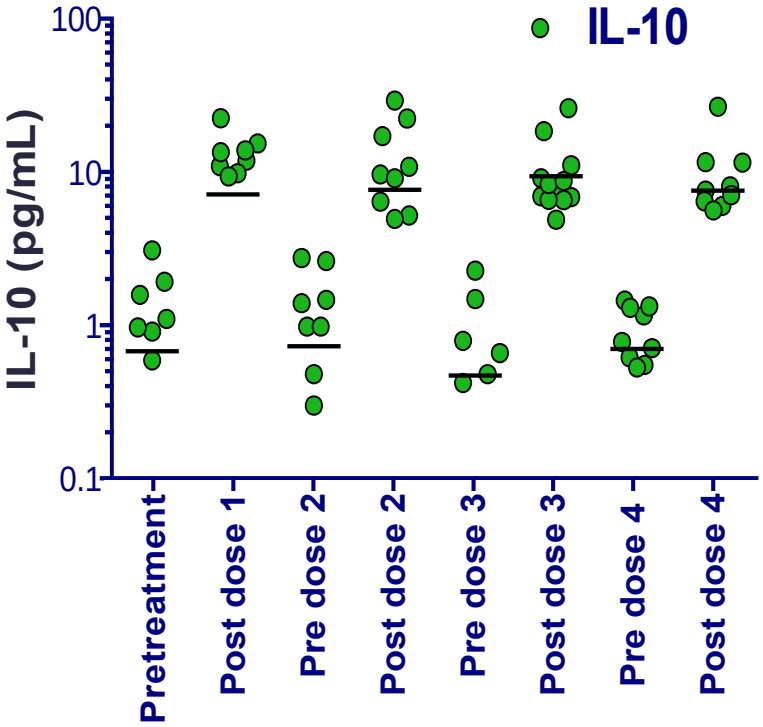
MIS416 favours anti-inflammatory IL-10 production in human monocyte assay



MIS416 dampens pro-inflammatory CD14



MIS416 enhances systemic levels of anti-inflammatory IL-10 in Phase 2A patient peripheral blood plasma samples



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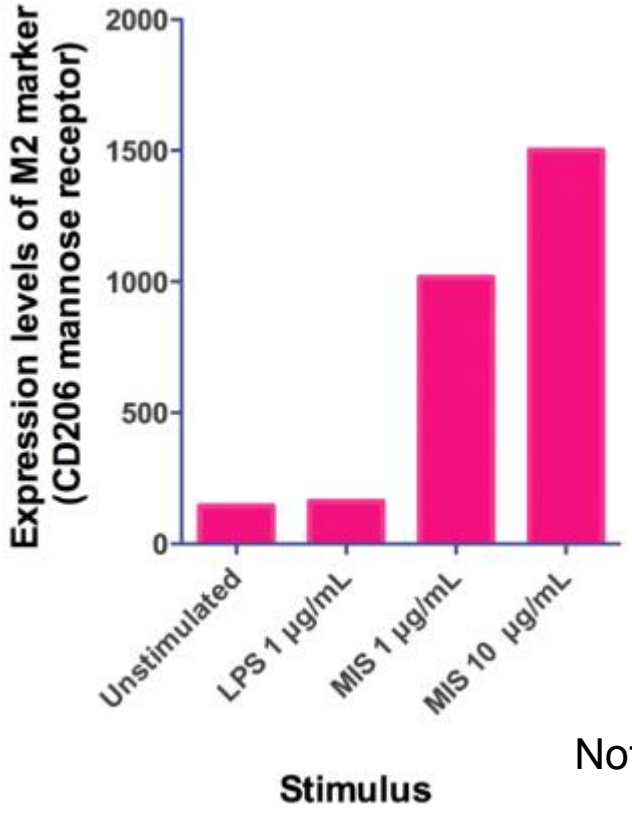
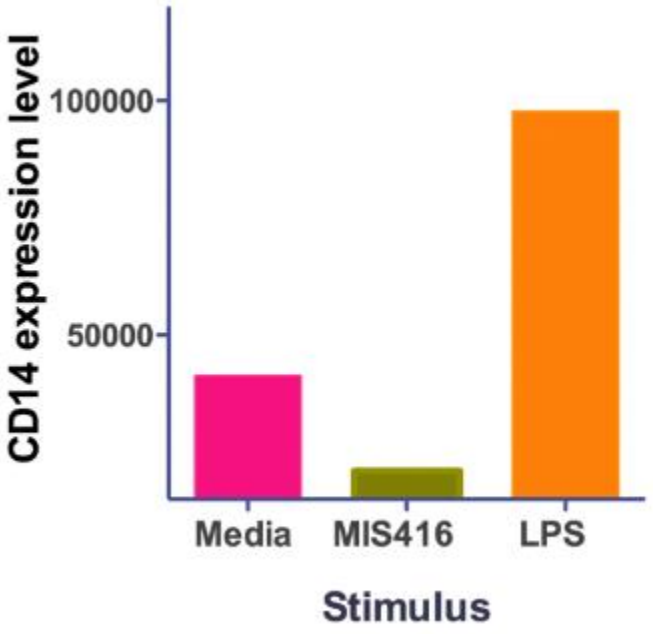
In vitro evidence that MIS416 can can generate "M2" regulatory myeloid cells in HUMAN monocyte assays

Up-regulation of CD206 is a marker for M2 macrophages



Down-regulation of CD14 which is associated with M2 differentiation



Note: LPS is an activator of "M1" macrophages

Myeloid cells from the periphery can access the CNS and promote myelin repair in CNS injury / disease models

Innate immunity: the missing link in neuroprotection and neurodegeneration?
Nguyen MD, Julien J-P, Rivest S. Nat Rev Neurosci. 2002;3(3):216-27

Trafficking CD11b-positive blood cells deliver therapeutic genes to the brain of amyloid-depositing transgenic mice. Lori Lebson, Kevin Nash, Siddharth Kamath, Donna Herber, Nikisha Carty, Daniel C. Lee, at el. J Neurosci **30**(29): 9651-9658.

Recruitment of beneficial M2 macrophages to injured spinal cord is orchestrated by remote brain choroid plexus.
Shechter R, Miller O, Yovel G, Rosenzweig N, London A, Ruckh J, et al.. Immunity. 2013;38(3):555-69.

Infiltrating blood-derived macrophages are vital cells playing an anti-inflammatory role in recovery from spinal cord injury in mice. Shechter R, London A, Varol C, Raposo C, Cusimano M, Yovel G, et al.. PLoS Med. 2009;6(7):e1000113. Clinical and Developmental Immunology. 2013

Harnessing monocyte-derived macrophages to control central nervous system pathologies: no longer 'if' but 'how'.
Shechter R, Schwartz M. The Journal of Pathology. 2013; 229(2):332-46)

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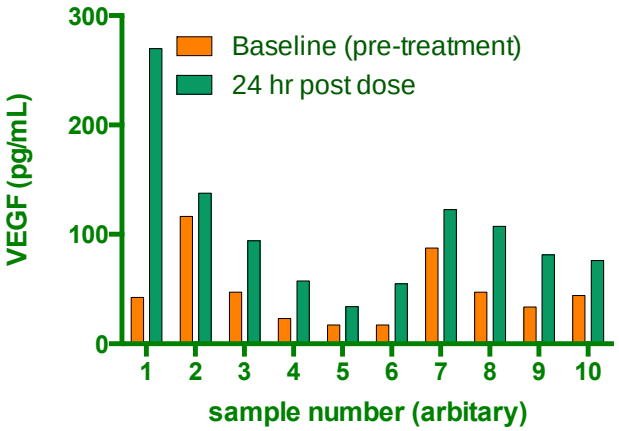
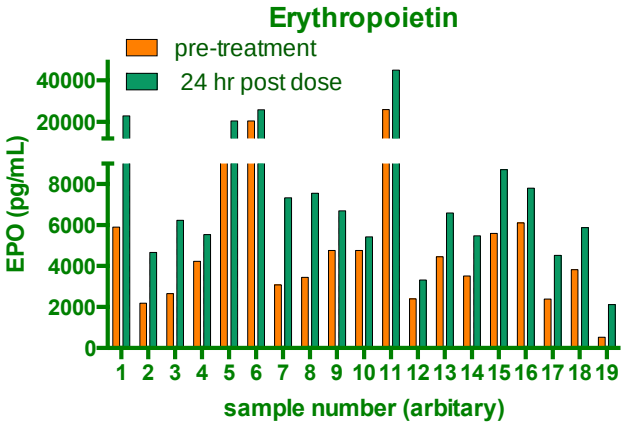
MIS416 treated patients demonstrate increased expression of tropic factors associated with CNS protection / repair (see AAN Poster P5.333)

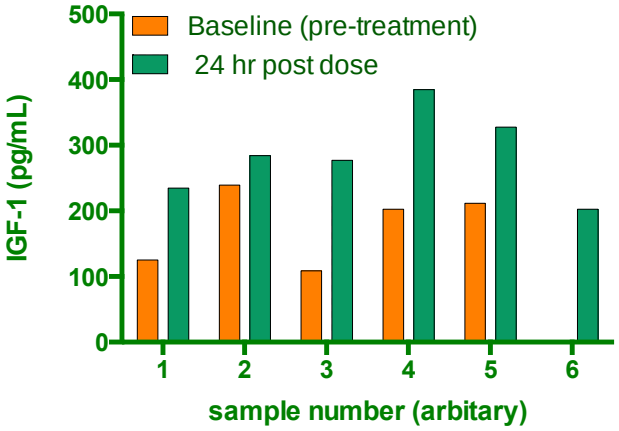
Vascular Endothelial Factor



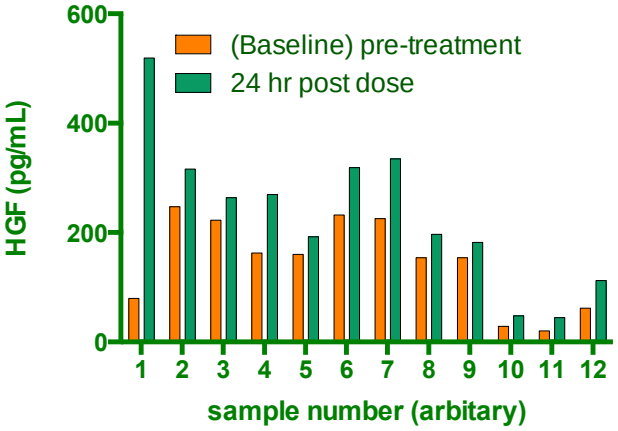
Erythropoietin



Insulin Growth Factor-1



Hepatocyte Growth Factor



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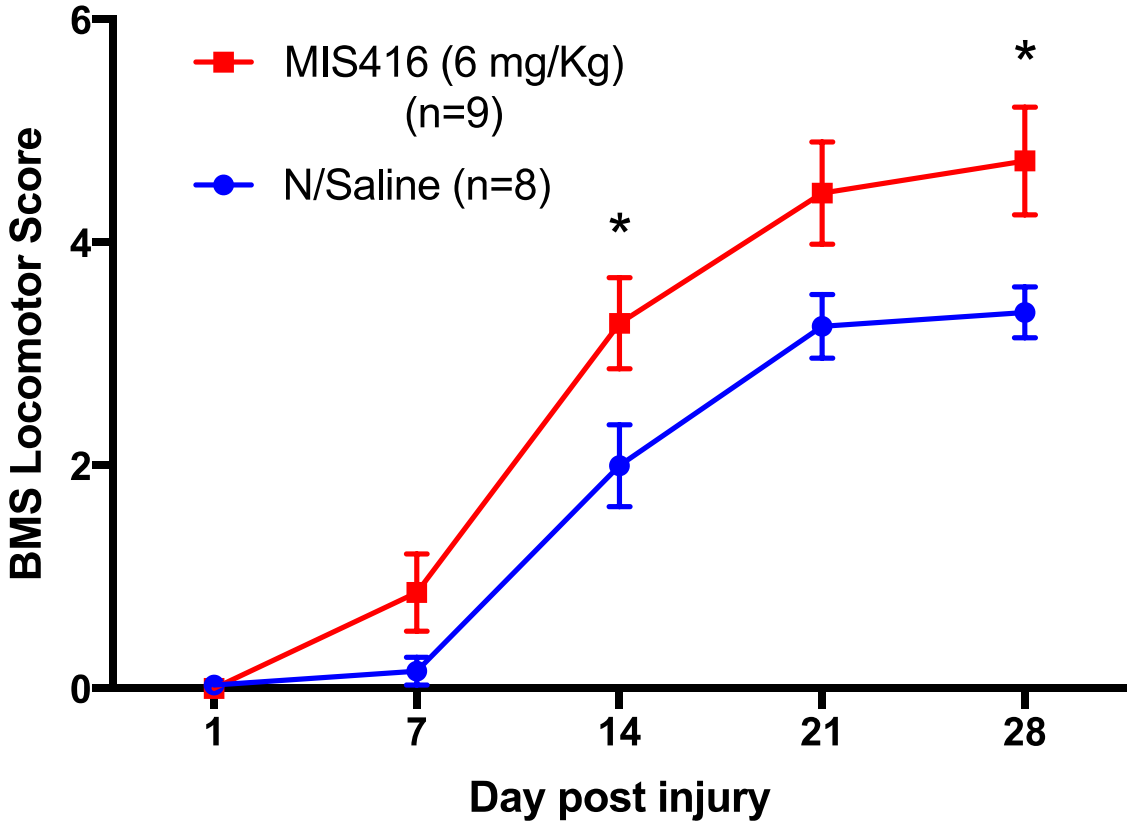
MIS416 enhances motor function recovery in Spinal Crush Injury model



* denotes significantly different (saline V MIS416)
(p <0.0001)

MIS416 activated myeloid cells are central to improvements in the SCI model. These myeloid directed anti-inflammatory/neurorepair pathways are broadly applicable to the treatment of other neuro-inflammatory disorders such as inflammatory epilepsy, traumatic brain injury and ischemic stroke.

See AAN Poster P5.333

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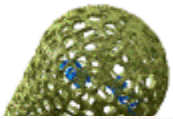
Drug candidate MIS416 – a myeloid directed immune modulator

- MIS416 is a suspension of intact, heat killed and extracted Propionibacterium acnes
- Unwanted pro-inflammatory ligands that target cell surface TLRs as well as antigens are removed



- Retains naturally occurring bacterial TLR9 and NOD2 pathogen recognitior
- Size and shape means that only phag myeloid cells take up MIS416
- MIS416 is functionally inert until biod intracellularly following phagosomal
- MIS416 targeted myeloid cells can en using homeostatic trafficking routes distinct to the blood brain barrier (e.g plexus)

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Phase 2B trial – MIS416-202 (results pending)

Study Design	
Phase	2B (NCT02228213)
Design	Randomized, double-blind, placebo-controlled study of the efficacy and safety of MIS416 in the treatment of subjects with SPMS Eligibility criteria includes EDSS 3.0 to relapse free for 2yrs, clinical ev progression
Sites	5 x Australia and Zealand
N	93 subjects with e-fre SPMS rand d 2:1 to or salin pla
Doses	Weekly i.v. usion of MIS416 or s over cycles of 4 doses per cycle weeks), titrated at 125ug of MIS41 the first dose, 250ug for the secon and 500ug for subsequent doses

Key Objectives

- To determin fficacy of 16, relative to as assed by various measures cular function
- explore the ffe of MIS416 on disease activity and neurodegeneration by ring a wide range of blood markers, ima g markers and patient reported outcomes

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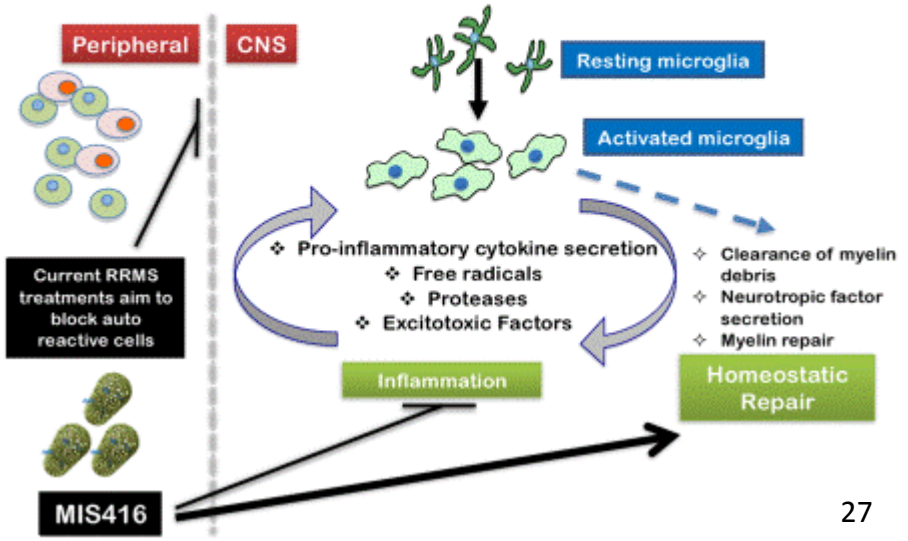
MIS416 targets sterile CNS inflammation and enhances endogenous myelin repair



Discussion

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Thank you

Janette Dixon - email: janette@innateimmuno.com phone +64 21 450 778
Gill Webster - email: gill@innateimmuno.com phone +64 21 640 885
Simon Wilkinson - email: simon@innateimmuno.com phone +64 21 661 850

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