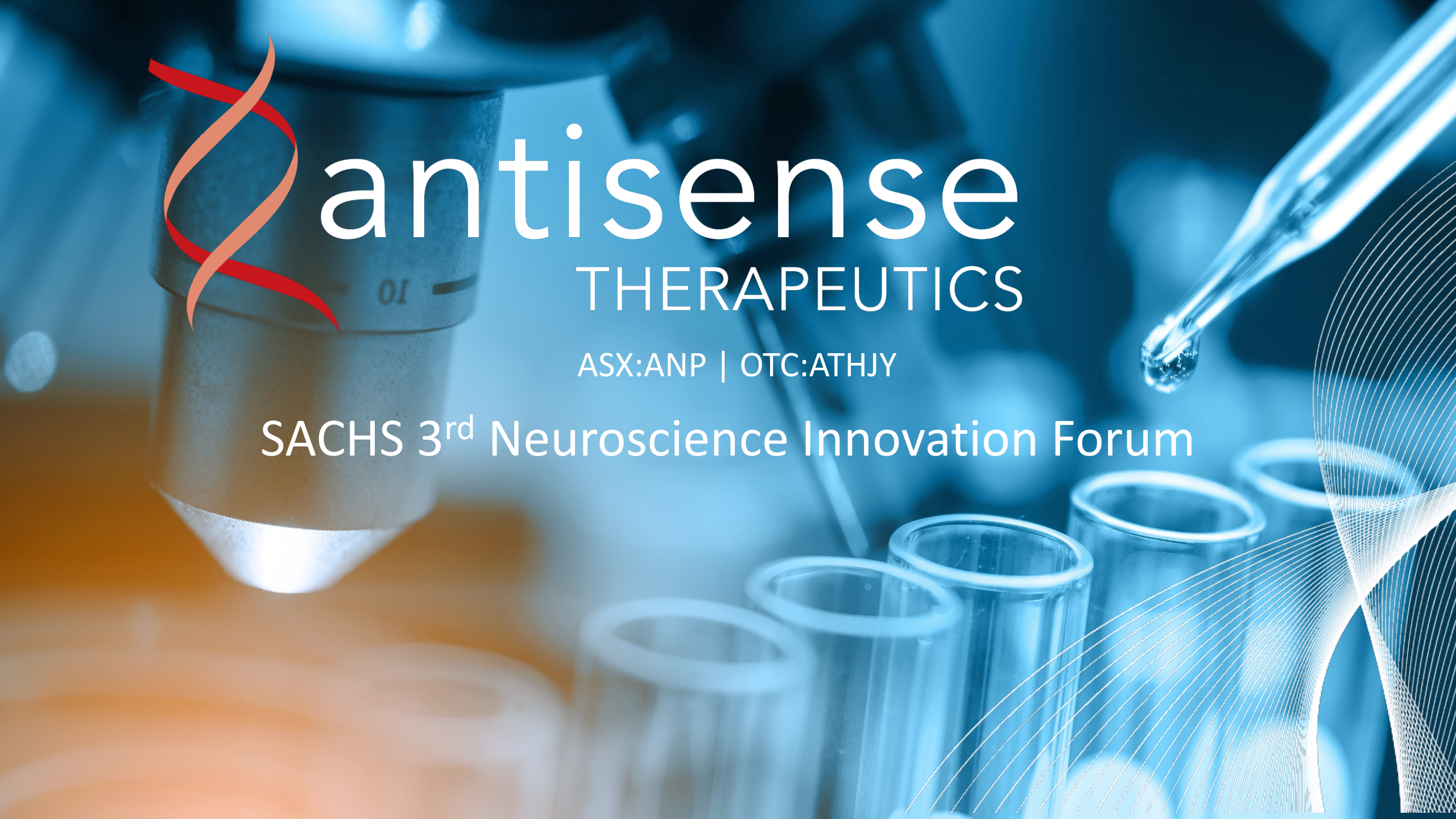

antisense
THERAPEUTICS
ASX:ANP | OTC:ATHJY
SACHS 3rd Neuroscience Innovation Forum

FORWARD LOOKING STATEMENTS

This presentation contains forward-looking statements regarding the Company's business & the therapeutic & commercial potential of its technologies & products in development. Any statement describing the Company's goals, expectations, intentions or beliefs is a forward-looking statement & should be considered an at-risk statement. Such statements are subject to certain risks & uncertainties, particularly those risks or uncertainties inherent in the process of developing technology & in the process of discovering, developing & commercializing drugs that can be proven to be safe & effective for use as human therapeutics, & in the endeavor of building a business around such products & services. Actual results could differ materially from those discussed in this presentation. Factors that could cause or contribute to such differences include, but are not limited to, those discussed in the Antisense Therapeutics Limited Annual Report for the year ended 30 June 2019, which is available from the Company or at www.antisense.com.au.

ANTISENSE THERAPEUTICS OVERVIEW

Australian, Melbourne-based biopharmaceutical company **developing & commercialising antisense pharmaceuticals** for large unmet markets

Advanced stage product pipeline with positive Phase II clinical results delivered from two compounds (ATL1102 & ATL1103)

Substantial shareholders include renowned Australian institutions in life sciences: Australian Ethical Investment & Platinum Asset Management

ATL1102 Phase II clinical trial in Duchenne Muscular Dystrophy (DMD)*

Positive results reported

Potential for out-licensing of ATL1103 for acromegaly

Preliminary interest from pharmaceutical companies

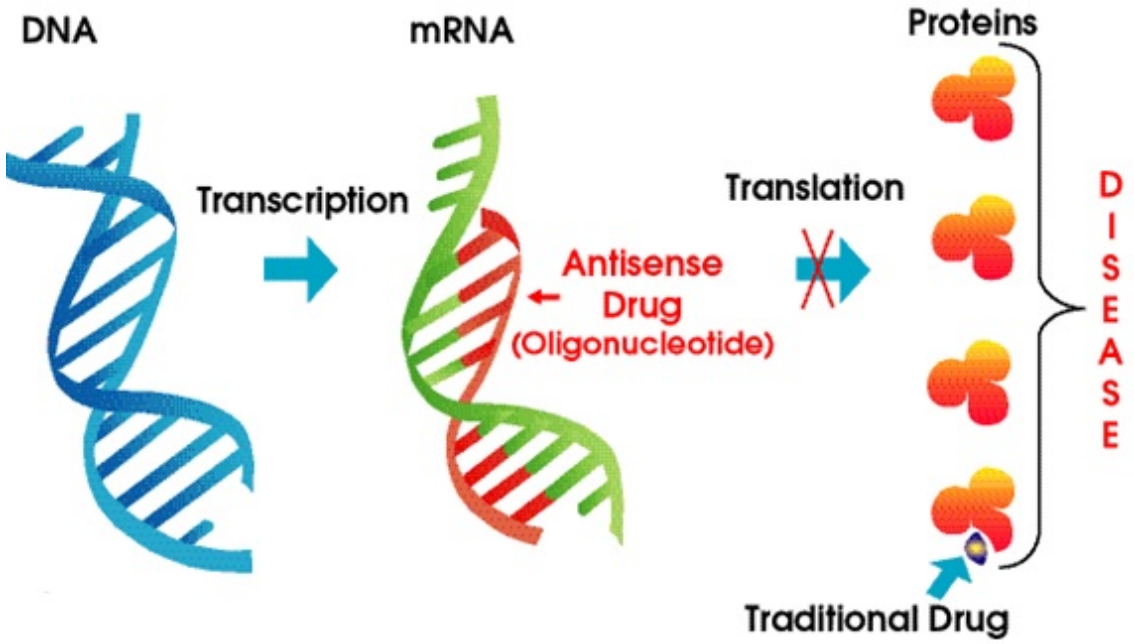
**DMD is one of the most common fatal genetic disorders caused by a mutation in the muscle dystrophin gene leading to severe progressive muscle loss & premature death in boys – high unmet medical need*

ANTISENSE – WHAT IS IT & HOW DOES IT WORK?

Antisense oligonucleotide drugs are small (12-25 nucleotides) DNA or RNA-like compounds that are chemically modified to create medicines

Antisense drugs prevent the production of proteins involved in disease processes by interrupting the translation phase of the protein production which results in a therapeutic benefit to patients

ANP is partnered with Ionis Pharmaceuticals (IONS: market capitalisation:US$9 Billion), world leaders in antisense drug development & commercialisation



ANTISENSE THERAPEUTICS ADVANCED STAGE CLINICAL PIPELINE

Targeting diseases where there is a need for improved therapies

1

ATL1102 IN DMD

- *Conducting Phase II clinical trial at Royal Children's Hospital in Melbourne, Australia*
- *Dosing completed in all 9 patients*
- *Positive efficacy and safety results reported*

2

ATL1103 IN ACROMEGALY

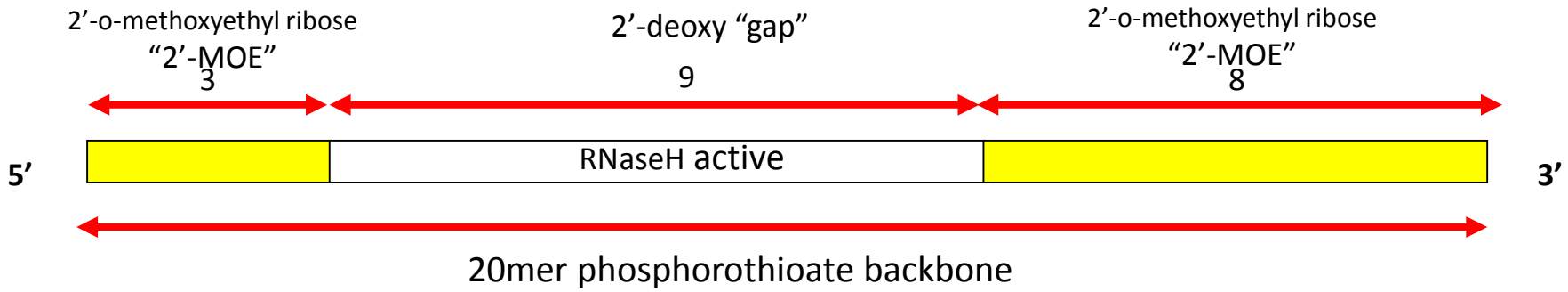
- *Phase II clinical trial completed*
- *Potential for out-licensing for further clinical development*

3

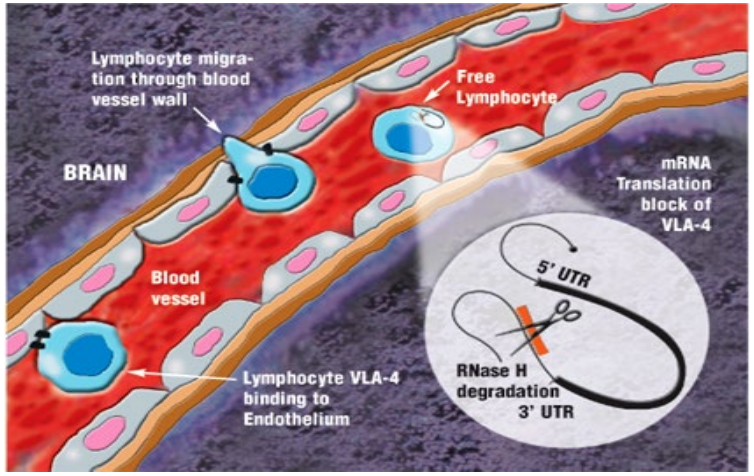
ATL1102 IN MS

- *Phase II clinical trial completed*
- *Data from DMD trial to inform on future clinical development in MS*

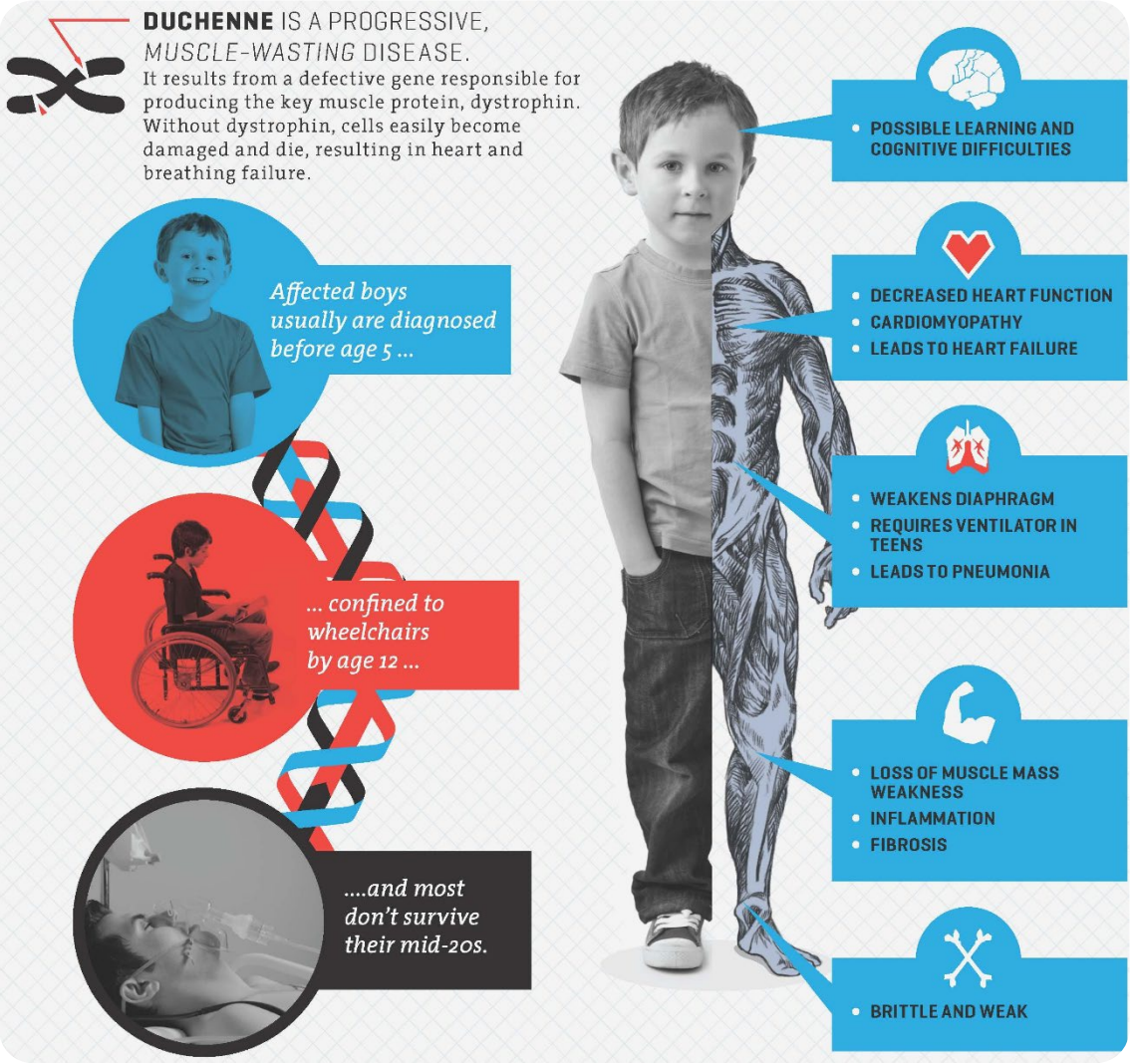
ATL1102: DRUG, TARGET & ACTIVITY OVERVIEW



- ATL1102 is designed to inhibit CD49d expression on lymphocytes and thereby reduce their survival, activation and migration from the blood into sites of inflammation
- ATL1102 is an designed to inhibit CD49d expression on lymphocytes and thereby stop/restrict there migration from the blood into sites of inflammation (e.g. the CNS in Multiple Sclerosis patients as pictured below) to reduce or modulate the adverse inflammatory effects



WHAT IS DMD?

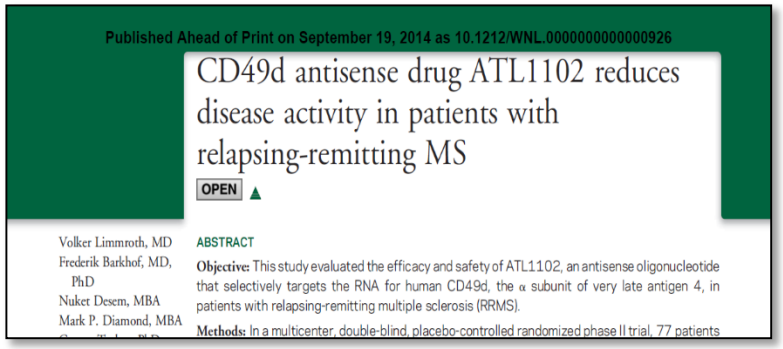


Source: CureDuchenne

- Duchenne Muscular Dystrophy (DMD) is a devastating genetic muscular disease caused by loss of dystrophin with progressive muscle wasting & associated muscle injury leading to inflammation &fibrosis (100% mortality)
- Affects boys with an incidence of ~1 in 3,500 & prevalence of ~44,000 in US & EU
- Dystrophin restoration treatments recently approved – eteplirsen (Exondys 51:Sarepta Therapeutics) for the 13% of patients amenable to Exon 51 skipping
- Key challenge in management of DMD patients is to reduce the inflammation that exacerbates muscle fibre damage
- Corticosteroids (CS) are the only therapy used to treat the inflammation in DMD but have insufficient efficacy& significant side effects including weight gain, reduced bone density & growth retardation. CS not as effective in patients with a greater number of CD49d receptors on T cells.

WHY ATL1102 for DMD?

- *Improved therapies are needed to ameliorate DMD severity & delay disease progression*
- *DMD is an orphan indication so can benefit from IP & development incentives*
- *Key publication confirms CD49d as potential target for DMD*



Published Ahead of Print on September 19, 2014 as 10.1212/WNL.0000000000000926

CD49d antisense drug ATL1102 reduces disease activity in patients with relapsing-remitting MS

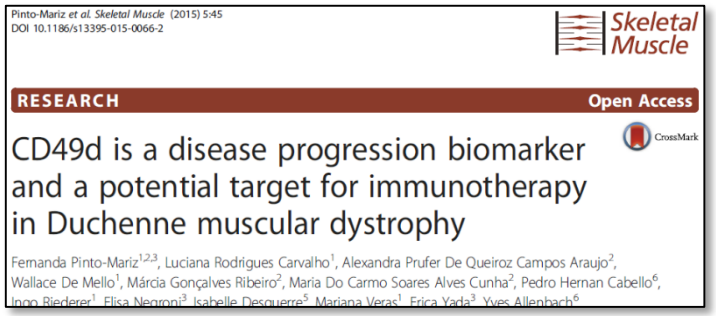
OPEN

Volker Limmroth, MD
Frederik Barkhof, MD, PhD
Nuket Desem, MBA
Mark P. Diamond, MBA

ABSTRACT

Objective: This study evaluated the efficacy and safety of ATL1102, an antisense oligonucleotide that selectively targets the RNA for human CD49d, the α subunit of very late antigen 4, in patients with relapsing-remitting multiple sclerosis (RRMS).

Methods: In a multicenter, double-blind, placebo-controlled randomized phase II trial, 77 patients



Pinto-Mariz *et al. Skeletal Muscle* (2015) 5:45
DOI 10.1186/s13395-015-0066-2

Skeletal Muscle

RESEARCH Open Access

CrossMark

CD49d is a disease progression biomarker and a potential target for immunotherapy in Duchenne muscular dystrophy

Fernanda Pinto-Mariz[1,2,3], Luciana Rodrigues Carvalho[1], Alexandra Prufer De Queiroz Campos Araujo[2], Wallace De Mello[1], Márcia Gonçalves Ribeiro[2], Maria Do Carmo Soares Alves Cunha[2], Pedro Hernan Cabello[6], Ingo Riederer[1], Elisa Negroni[3], Isabelle Desguerre[5], Mariana Veras[1], Erica Yada[3], Yves Allenbach[6]

ATL1102, an antisense drug to CD49d, shown to be a highly active immunomodulatory drug with potent effects on inflammatory processes in MS patients

- *90% reduction in inflammatory brain lesions vs placebo [Limmroth V et al Neurology 2014]*
- *Reduced CD49d on T & B cells, and T & B cell numbers by ~25 & 50% respectively*
- *Pre-clinical & clinical data in MS has supported move directly into the six-month DMD patient trial (effective leveraging of substantial investment & progress made to date in MS)*

Pivotal scientific publication confirming CD49d as a potential target for DMD therapy

- *DMD patients with greater number of circulating T cells with high levels of CD49d (alpha chain of VLA-4) expression have both more severe & rapid progression of disease [Pinto-Mariz et al Skeletal Muscle 2015]*
- *Ambulant patients on CS suggesting CS do not reduce CD49dhi expression on T cells*
- *CS treatment does not modulate CD49d expression on T cells in MS*
- *Non-ambulant DMD patients have greatest number of CD49d high expressing T cells*

Antisense Therapeutics is the only Company with a CD49d targeting drug in development for DMD

ATL1102 PHASE II STUDY

- *Trial led by RCH Head of Neuromuscular Clinic Prof Monique Ryan & RCH Neuromuscular Fellow Dr Ian Woodcock*
- *Neuromuscular clinic at RCH the largest in the Southern Hemisphere for treating boys with DMD*

Primary objective: To assess the safety and tolerability of 25 mg of ATL1102 administered once weekly (s.c. injection) for 24 weeks in non-ambulatory DMD participants

Secondary objectives: To evaluate the

- lymphocyte-modulatory potential of ATL1102 in participants with DMD
- PK profile of ATL1102 in participants with DMD
- effects of ATL1102 on functional capacity in participants with DMD (PUL2.0 and MyoGrip and MyoPinch)
- effects of ATL1102 on respiratory function in participants with DMD
- effects of ATL1102 on quality-of-life in participants with DMD

Design: Single-centre, open-labelled study conducted at the Royal Children's Hospital (RCH), Melbourne, Australia

Sample size: 9 participants

Target population:

- participants diagnosed with DMD and have been non-ambulatory for at least 3 months
- 10 to 18 years of age
- body weight of more than 25 kg and less than or equal to 65 kg

ATL1102 PHASE II STUDY – DATA OVERVIEW

Data from all 9 participants having completed 24 weeks of dosing has affirmed ATL1102's excellent safety profile and positive drug effects on disease progression endpoints at the low dose tested

Safety

- ATL1102 appeared to be generally safe and well tolerated with no Serious Adverse Events reported
- No participants withdrew from the study
- Independent Data and Safety Monitoring Board - no safety concerns
- The most commonly reported TEAEs were injection site erythema and skin discolouration

Efficacy

- Consistency in the mean reductions from baseline in the number and type of lymphocytes with a return to around starting levels post dosing supportive of the drugs positive effects on modulating T cells in the blood
- PUL2.0 data showed 7 of 9 participants demonstrated either increases or no change in their scores from baseline suggestive of an overall improvement with a positive mean change in this parameter
- MyoGrip and MyoPinch assessments showed a distinct improvement in muscle strength based on mean changes from baseline compared to the losses reported in a previous study in a similar non ambulant population on corticosteroids (Ricotti et al 2016)
- Final study report to be prepared following database lock expected in 1Q'20

ATL1102 PHASE II STUDY

Immune Cell Data

White blood cell type (X10^9 cells per litre)	Mean # and Change from baseline			Median % change from baseline	
	Baseline	24 weeks (end of dosing)	28 weeks	24 weeks (end of dosing)	28 weeks
Lymphocytes (mostly CD3+ T cells)	3.68	-0.28	+0.19	-4.22%	+11.81%
CD3+ T cells (mostly CD3+ CD4+ and CD3+ CD8+ T cells)	2.93	-0.18	+0.25	0.86%	+17.11%
CD3+ CD49d+ T cells (CD4+CD49d+ and CD8+CD49d+ cells)	2.44	-0.28	+0.11*	-9.78%	+9.93%
CD4+ T cells	1.57	-0.15	+0.11	-1.12%	+16.50
CD4+ CD49d+ T cells	1.20	-0.19	+0.01	-16.7%	+1.73
CD4+ CD49d++ T cells (are the high CD49d expressing CD4+ T cells)	0.24	-0.01	+0.01	-11.1%	+7.58
CD8+ T cells	1.22	-0.02	+0.14	-2.62%	+17.99
CD8+ CD49d+T cells	1.17	-0.05	+0.11	-5.79%	+13.37
CD8+ CD49d++ T cells (5 of 9 patients had these cells at baseline) (are the high CD49d expressing CD8+T cells)	-	-	-	-6.17%	+14.12

The Lymphocyte mean # of cells at week 24 (at the end of dosing) is trending significantly lower vs week 28 (p= 0.051 paired T test)
The CD3, CD4, CD8, CD4+CD49d+ and CD8+CD49d+ mean # of cells at week 24 are similarly trending lower vs week 28 (p= from 0.056 to 0.073)
*The mean # of CD3+CD49d+ T cells (=CD4+CD49d+ and CD8+CD49d+cells) at week 24 is statistically significantly lower vs week 28 (p= 0.030 paired T test)

ATL1102 PHASE II STUDY

Overview of Disease Progression Efficacy Parameters

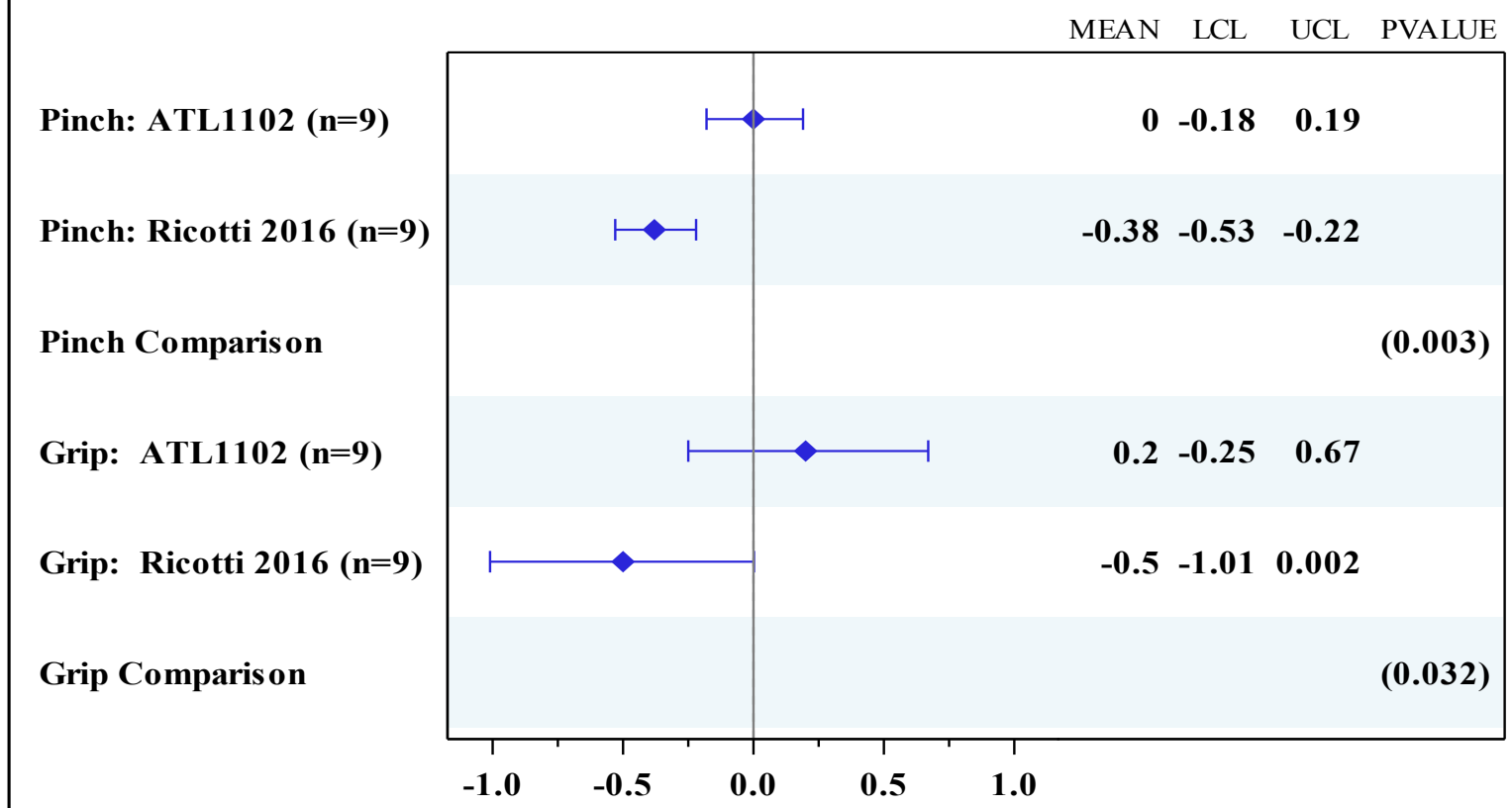
	Change from Baseline to Week 24						
Patient No.	PUL 2.0	MyoGrip (dom) (Kg)	MyoGrip (dom) (% Pred)	MyoPinch (dom) (Kg)	MyoPinch (dom) (% Pred)	% Predicted FVC	% Predicted PEF
01-001	+2	-0.63	-4.49	0.03	-0.62	-3.20	6.30
01-002	+2	0.22	0.49	-0.02	-0.29	-14.8	-17.3
01-003	0	0.68	1.02	-0.40	-6.59	-9.10	8.70
01-004	+2	1.09	1.01	0.37	2.99	0.80	7.20
01-006	-3	-0.27	-0.60	0.07	0.94	-6.50	6.90
01-008	+7	1.00	1.11	0.30	2.77	-7.70	-18.2
10-009	0	-0.33	-3.75	-0.22	-4.97	-9.10	-4.30
01-010	0	0.05	0.11	0.06	0.72	-0.40	9.20
01-011	-2	0.11	-1.31	-0.18	-3.63	-1.10	2.00
Mean Change (95% CI):	0.9 (-1.33, 3.11)	0.2 (-0.25, 0.67)	-0.7 (-2.33, 0.90)	0.0 (-0.18, 0.19)	-1.0 (-3.56, 1.63)	-5.68 (-9.60,-1.76)	0.06 (-8.33, 8.44)

ATL1102 PHASE II STUDY

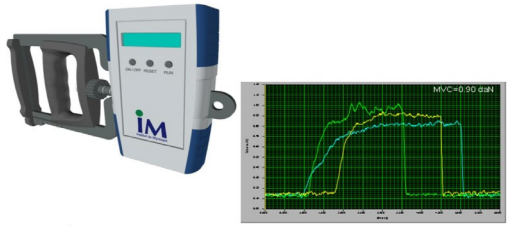
Efficacy Parameters – Comparison to Published Data for Myoset Assessments

Improvements in Grip and Pinch strength statistically significant vs Ricotti et al 2016

Forest Plot for the Mean Change (95% CIs) in Pinch and Grip from Baseline to Month 6



ATL1102 Results based on the DOMINANT Side Interim Analysis from the 1102-DMD-CT02 study
Ricotti results based on results from the published paper
PValue: Two-sided p-value from T-Test comparing change between ATL and Ricotti results



* Ricotti et. al 2016 . PLoS One, 11(9) e0162542 historical results from 8 Non – Ambulant patients on CS for 6month

ATL1102 PHASE II STUDY

Expert summation of results

- In consultation with internationally recognised DMD experts in regard to evaluating response to therapy within the trial and with reference to the loss of function in non-ambulant boys reported in the literature (Pane et al 2018 and Ricotti et al 2019), 3 patients appear to have improved by a clinically meaningful amount on both PUL2.0 and grip strength with another patient showing a similar improvement on PUL2.0 alone and a further 3 stabilized on this parameter
- Professor Thomas Voit MD, Director, NIHR GOSH Biomedical research and author on the Ricotti et al 2019 publication said of the results:
    - *"Disease stabilisation or indeed improvement in functional scores in non-ambulant DMD boys is almost unheard of and a very encouraging result. This is even more meaningful as these results have been obtained using different independent measures and over a relatively short trial time of 24 weeks. These results also advise on endpoint choice for a fully powered placebo controlled registration-enabling study"*

PHASE IIB CLINICAL TRIAL

- Scientific Advice (SA) meetings have been held with three European regulatory authorities
- SA meetings focussed on the Phase IIb trial design, dose escalation plans, applicability of the study end-points and the study duration
- General acceptance by the agencies on the trial efficacy endpoints (PUL2.0 Myoset), safety monitoring plan, dosing duration (1 year) and the use of higher doses
- Clarification provided by the agencies that the above could be a path forward to an approval on positive Phase IIb results
- Next step is to follow up development plan with the European Medicines Agency (EMA) and subsequent to the finalisation of the results from the current Phase II trial, engage with the Food and Drug Administration (FDA) on development plans for the US

EUROPEAN MEDICINES AGENCY
SCIENCE MEDICINES HEALTH

PROPOSED ATL1102 PHASE 2B/3 DMD STUDY: OVERVIEW

Design:

- Multicentre, randomised, double-blind, placebo-controlled study to be conducted in Europe
- Participants will be randomised in a 1:1:1 ratio to 2 dose levels of ATL1102 or matching placebo
- ATL1102 or placebo will be administered as a subcutaneous injection, once weekly for 52 weeks

Target population:

- Non-ambulant participants with DMD, 10 to 18 years of age

Sample size:

- Approx. 25 participants per arm

Primary objective:

To evaluate the effect of ATL1102 on upper limb muscle function in non-ambulant participants with DMD, as assessed by change in the Performance of Upper Limb Module for DMD 2.0 (PUL 2.0) score

Secondary objectives:

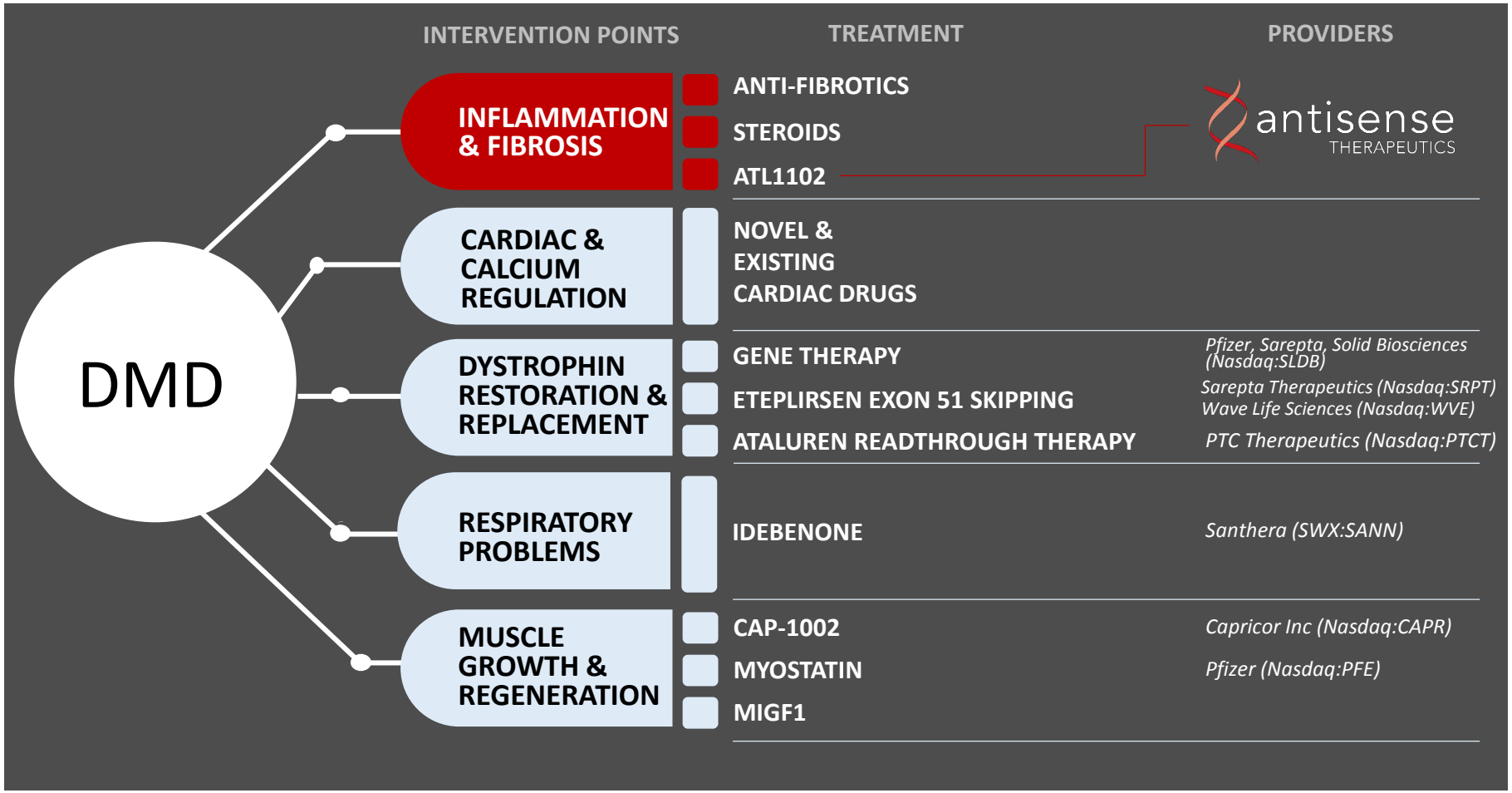
To evaluate the

- effects of ATL1102 on muscle strength
- effects of ATL1102 on respiratory function
- effects of ATL1102 on quality-of-life
- safety and tolerability of ATL1102
- PK profile of ATL1102 in participants with DMD

All participants completing the Treatment Period of the study will be offered to the enter an open label extension study of ATL1102 conducted under a separate protocol and will be given the highest dose tested in the study or the highest dose considered generally safe and well tolerated in the study. This will serve to further increase the safety and efficacy database.

TREATMENT DEVELOPMENT FOCUSING ACROSS ALL INTERVENTION POINTS

- *Prospect for these therapies to be complementary rather than competitive*
- *Other anti-inflammatory therapies are being tested in ambulant children*



frontiers in Genetics

MINI REVIEW
published: 10 April 2018
doi: 10.3389/fgene.2018.00114

Combined Therapies for Duchenne Muscular Dystrophy to Optimize Treatment Efficacy

*Gonzalo Cordova[1], Elisa Negroni[1], Claudio Cabello-Verrugio[2,3], Vincent Mouly[1] and Capucine Trollet[1]**

[1] Sorbonne Université, Institut National de la Santé et de la Recherche Médicale, Association Institut de Myologie, Centre de Recherche en Myologie, UMRS974, Paris, France, [2] Laboratorio de Patologías Musculares, Fragilidad y Envejecimiento, Departamento de Ciencias Biológicas, Facultad de Ciencias Biológicas, Universidad Andres Bello, Santiago, Chile, [3] Millennium Institute on Immunology and Immunotherapy, Santiago, Chile

Duchene Muscular Dystrophy (DMD) is the most frequent muscular dystrophy and one of the most severe due to the absence of the dystrophin protein. Typical pathological features include muscle weakness, muscle wasting, degeneration, and inflammation. At advanced stages DMD muscles present exacerbated extracellular matrix and fat accumulation. Recent progress in therapeutic approaches has allowed new strategies to be investigated, including pharmacological, gene-based and cell-based therapies. Gene and cell-based therapies are still limited by poor targeting and low efficiency in fibrotic dystrophic muscle, therefore it is increasingly evident that future treatments will have to include "combined therapies" to reach maximal efficiency. The scope of this mini-review is to provide an overview of the current literature on such combined therapies for DMD. By "combined therapies" we mean those that include both a therapy to correct the genetic defect and an additional one to address one of the secondary pathological features of the disease. In this mini-review, we will not provide a comprehensive view of the literature on therapies for DMD, since many such reviews already exist, but we will focus on the characteristics, efficiency, and potential of such combined therapeutic strategies that have been described so far for DMD.

ATL1102's novel mechanism in targeting CD49d suggests potential for drug to be used in combination with other treatments including anti-inflammatory agents

MARKET CONSIDERATIONS FOR ATL1102

- *ATL1102 - anti-inflammatory and immune modulating agent with potential for multiple clinical applications*

ANTI-INFLAMATORY

*Anti-Inflammatory Therapeutics Market^ is expected to garner **US$106.1 billion** by 2020*

(Allied Market Research)

^MS, Arthritis, Psoriasis, Respiratory, IBD

CORTICOSTEROIDS

*The global steroid market is forecast to attain the value of **US$17 Billion** by the end of 2025*

(QV Research)

DMD THERAPIES

*The global DMD drug market is expected to reach over **US$4 Billion** by 2023*

(Grand View Research)

- Corticosteroids are the only marketed therapy to treat the inflammatory damage associated with dystrophin loss in DMD
- Prevalence of DMD in EU and US est. 44,000 with most ambulant and ~2/3 of non-ambulant patients on corticosteroids*
- DMD cost of therapy considerations

  Deflazacort (Emflaza) is a CS approved in US only - average annual cost estimated > US$80K per patient per annum

  Exondys 51 (dystrophin restoration agent) cost in the US is > US$400K per patient per annum

* Cowan L et al BMC Neurology (2019), 1-10

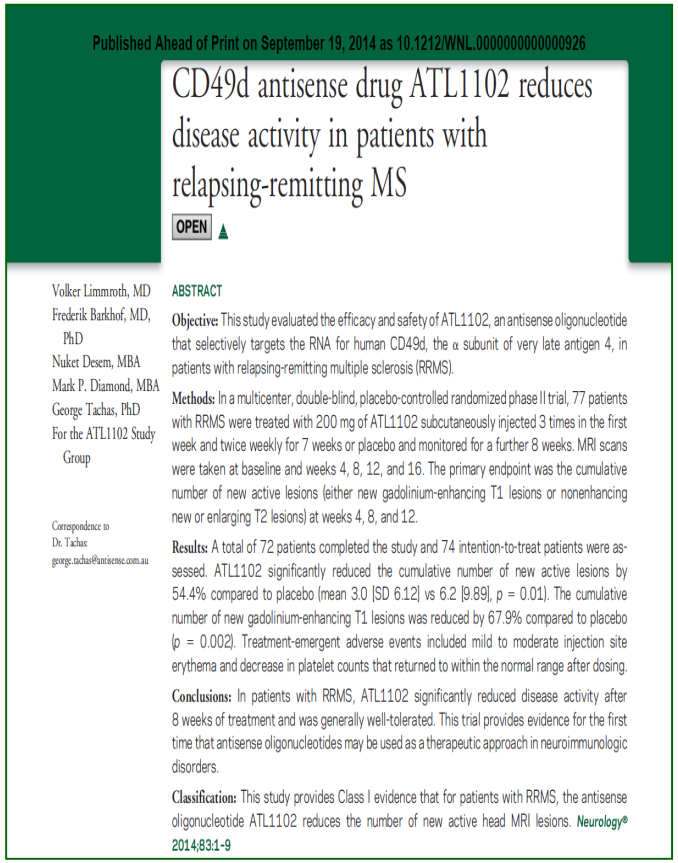
ATL1102 for MULTIPLE SCLEROSIS

Multiple Sclerosis (MS)

- MS is a chronic, progressive, and debilitating autoimmune disease that affects central nervous system, brain and spinal cord
- Approx. 400,000 people in North America and more than 2.5 million worldwide with MS
- Drug sales in 2018 were US$23 Billion forecast to grow to US$39 Billion by 2026

ATL1102

- Successful Phase II trial in patients with Relapsing Remitting-MS with trial results published in Journal of Neurology*
- Efficacy outcomes compared favorably to Tysabri and other marketed MS therapeutics with no suggestion of JCV/PML risk
- Evidence of selective activity on the adaptive immune system (in particular T and B cells)
- ANP submitted a US IND application for a 6 month, Phase 2b human trial in relapsing MS (relapsing remitting and relapsing secondary progressive MS). FDA approved the study to move forward at a lower dose of 25mg/week for 6 months (partial-hold). Same dose as used in Phase II DMD trial
- ANP are exploring the conditions that would allow MS patients to receive higher doses while monitoring the progress of the DMD trial which could provide support for undertaking studies in MS patients at the FDA approved dose



Published Ahead of Print on September 19, 2014 as 10.1212/WNL.0000000000000926

CD49d antisense drug ATL1102 reduces disease activity in patients with relapsing-remitting MS

OPEN

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For the ATL1102 Study Group

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ABSTRACT

Objective: This study evaluated the efficacy and safety of ATL1102, an antisense oligonucleotide that selectively targets the RNA for human CD49d, the α subunit of very late antigen 4, in patients with relapsing-remitting multiple sclerosis (RRMS).

Methods: In a multicenter, double-blind, placebo-controlled randomized phase II trial, 77 patients with RRMS were treated with 200 mg of ATL1102 subcutaneously injected 3 times in the first week and twice weekly for 7 weeks or placebo and monitored for a further 8 weeks. MRI scans were taken at baseline and weeks 4, 8, 12, and 16. The primary endpoint was the cumulative number of new active lesions (either new gadolinium-enhancing T1 lesions or nonenhancing new or enlarging T2 lesions) at weeks 4, 8, and 12.

Results: A total of 72 patients completed the study and 74 intention-to-treat patients were assessed. ATL1102 significantly reduced the cumulative number of new active lesions by 54.4% compared to placebo (mean 3.0 [SD 6.12] vs 6.2 [9.89], $p = 0.01$). The cumulative number of new gadolinium-enhancing T1 lesions was reduced by 67.9% compared to placebo ($p = 0.002$). Treatment-emergent adverse events included mild to moderate injection site erythema and decrease in platelet counts that returned to within the normal range after dosing.

Conclusions: In patients with RRMS, ATL1102 significantly reduced disease activity after 8 weeks of treatment and was generally well-tolerated. This trial provides evidence for the first time that antisense oligonucleotides may be used as a therapeutic approach in neuroimmunologic disorders.

Classification: This study provides Class I evidence that for patients with RRMS, the antisense oligonucleotide ATL1102 reduces the number of new active head MRI lesions. *Neurology®* 2014;83:1-9

**Limmroth, V. et al Neurology, 2014; 83(20): 1780-1788*

ATL1102 for OTHER AUTOIMMUNE – INFLAMMATORY DISEASES

- *ATL1102 targets CD49d+ immune cells involved in disease processes*
- *CD49d is a clinically validated target in multiple disease indications and a potential superior target in other autoimmune inflammatory disease*
- *Pipeline development focus*

- CD49d is clinically validated in Multiple Sclerosis and Crohns disease where antibody drugs to CD49d are used
- There are several orphan indications (like DMD) where CD49d expression is important in disease processes, and where CD49d appears to be a superior target
- ANP could move directly into clinical studies based on existing preclinical and clinical data
- Grant funding opportunities exist for such projects
- ATL1102 drug product is available for studies
- ANP to progress in indications where there are ATL1102 platform (antisense) and target (CD49d) based advantages
- Further details to be advised once appropriate Intellectual Property protection is in place

BOARD OF DIRECTORS

Mr Robert W Moses

Independent Non-Executive Chairman

Formerly Corporate Vice President of CSL Limited. Mr. Moses draws on more than 40 years' experience in the pharmaceutical/ biotechnology industry.

Mr Mark Diamond

Managing Director & Chief Executive Officer

Over 30 years' experience in the pharmaceutical & biotechnology industry. Formerly Director, Project Planning/Business Development at Faulding Pharmaceuticals in the USA, Senior Bus Dev Manager within Faulding's European operation & International Business Development Manager with Faulding in Australia.

Dr Graham Mitchell

Independent Non-Executive Chairman

Joint Chief Scientist for the Victorian Government Department of Environment & Primary Industries. Formerly Director of Research in the R&D Division of CSL Limited.

Dr Gary Pace

Independent Non-Executive Director

Dr Pace has more than 40 years' international experience in the development & commercialisation in biotechnology/ pharmaceuticals industries. Long-term board level experience with both multi-billion & small cap companies.

Mr William Goolsbee

Independent Non-Executive Director

Founder, Chairman & CEO of Horizon Medical Inc. 1987 – 2002 until acquisition by UBS Private Equity. Founding Director then Chairman of ImmunoTherapy Corporation until acquisition by AVI Biopharma, Inc. (now Sarepta Therapeutics). Former Chairman of privately held BMG Pharma LLC & Metrodora Therapeutics.

CORPORATE OVERVIEW

KEY FINANCIALS	
Market Capitalisation (at $0.085)	A$41.5M
Shares on issue	488.8M
52-week high/low	$0.145 - $0.026
Cash as at 30 September 2019*	$2M

*In December 2019 received **$5.5 million** via conversion and underwriting of listed options and **$559K** R&D tax incentive

OWNERSHIP STRUCTURE	
Top 40 holders	53.87%
Substantial Shareholders	
• *Australian Ethical Investment*	*14.57%*
• *Platinum Asset Management*	*5.15%*
• *Leon Serry*	*6.15%*

ANTISENSE THERAPEUTICS SUMMARY & VALUE DRIVERS

Advanced stage product pipeline – **two compounds with positive Phase II clinical results published in high quality peer reviewed scientific journals with multiple clinical applications**

Highly regarded Australian institutional shareholders - Australian Ethical Investment & Platinum Asset Management

Phase II clinical trial in Duchenne Muscular Dystrophy (DMD) – ATL1102

- Positive results reported from all 9 patients having completed dosing
- Phase IIb trial design and approval process running in parallel with completion of Phase II trial
- Drug potentially complementary to other DMD programs e.g. Sarepta Therapeutics
- Significantly 'underserved market' with comparable company benchmarks demonstrating substantial value creation potential

ATL1103 (atesidorsen) for acromegaly

- Potential for partnering to further develop the compound

For more information:
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