WILSONS
Equity Research

Date
30 September 2021

Theme
Company Update

Company
Antisense Therapeutics Limited (ANP)

Mechanism strengthened; reach expanded

We maintain our OVERWEIGHT recommendation on Antisense Therapeutics (ANP) and risked PT of $0.63 per share. Recent presentations at the World Muscle Society (WMS) meeting (20-24 Sept) from those working in DMD have reminded us that ATL1102 is positioned well versus peer approaches (i.e. Capricor) and that DMD gene therapies continue to struggle with safety setbacks (e.g. recent Pfizer narrowing of Phase III trial inclusion due to serious adverse events). Importantly, we have a growing understanding and comfort in the mechanistic basis of ATL1102's effects in DMD boys from the Phase II trial data but also from the recently presented proteomics analysis that has shown potential for ATL1102 to expand into ambulant DMD cohorts in the future. In this note we summarise these developments, outline the mechanistic data supporting ATL1102 thus far and provide a head to head update on ATL1102 efficacy vs CAP-1002, a competitor in the non-ambulant space that has just released final Phase II data.

As a reminder, the key catalyst we await is feedback from the European regulator to bed down ANP's Phase IIb trial design and plans which is due at any point. This paves the way for CTA submission, approval and trial initiation, still planned for CY21.

Recommendation	**OVERWEIGHT**
12-mth target price (AUD)	**$0.63**
Share price @ 28-Sep-21 (AUD)	$0.21
Forecast 12-mth capital return	200.0%
Forecast 12-mth dividend yield	0.0%
12-mth total shareholder return	**200.0%**
Market cap	$121m
Enterprise value	$115m
Shares on issue	574m
Sold short	
ASX 300 weight	n/a
Median turnover/day	$0.2m

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Key points

Analysis of Phase II data further supports ATL1102 mechanism; expands potential TAM. Data showing that ATL1102 treatment has caused positive changes to known genetic modifiers of loss of ambulation (LTBP4, THBS1) supports its future expansion into ambulant DMD cohorts. We note ANP have already included scope for ambulant populations within their EU regulator discussions. This doubles the potential DMD TAM (in the longer term) which we model as restricted to non-ambulant DMD patients only.

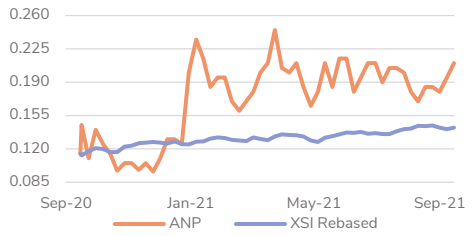
PDCO feedback due at any point. We anticipate feedback from the European regulator to confirm Phase IIb study design at any point in the next quarter. This is a key step in providing clarity on trial design but also allows for CTA submission and trial start (CY21).

Capital required to support Phase IIb study. Antisense had $6M net cash as at end FY21. We understand that the manufacture of ATL1102 for the Phase IIb study is complete and trial preparations are under way. We have previously noted we expect the EU Phase IIb study to cost ~$20-25M with potential further costs for open-label extension phases.

Risks and catalysts

Risks: a) unfavourable clinical trial results; b) lack of capital to support expenses; c) share dilution; d) competitor development of DMD therapies **Catalysts:** a) EMA trial approval; b) FDA engagement & IND approval; c) board renewal; d) partnering opportunities.

12-mth price performance ($)



	1-mth	6-mth	12-mth
Abs return (%)	13.5	5.0	82.6
Rel return (%)	14.1	-4.5	59.0

Earnings forecasts

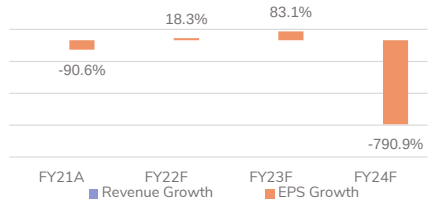
Year-end June (AUD)	FY20A	FY21A	FY22F	FY23F	FY24F
NPAT rep ($m)	-5.9	-8.1	-13.3	-2.5	-22.6
NPAT norm ($m)	-5.9	-9.7	-13.3	-2.5	-22.6
Consensus NPAT ($m)			-14.3	-5.4	-0.8
EPS norm (cps)	-1.3	-2.5	-2.0	-0.3	-3.1
EPS growth (%)	-71.1	-90.6	18.3	83.1	-790.9
P/E norm (x)	-16.2	-8.5	-10.4	-61.2	-6.9
EV/EBITDA (x)	-19.8	-14.1	-8.6	-40.2	-5.0
FCF yield (%)	-3.3	-4.8	-8.3	1.8	-17.7
DPS (cps)	0.0	0.0	0.0	0.0	0.0
Dividend yield (%)	0.0	0.0	0.0	0.0	0.0
Franking (%)	0	0	0	0	0

Source: Company data, Wilsons estimates, Refinitiv

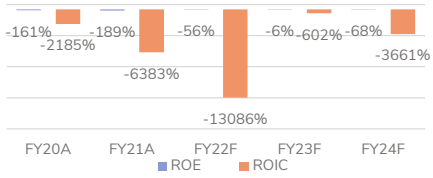
Key changes

		08-Sep	After	Var %
NPAT:	FY22F	-13.3	-13.3	0.0%
norm	FY23F	-2.5	-2.5	0.0%
($m)	FY24F	-22.6	-22.6	0.0%
EPS:	FY22F	-2.0	-2.0	0.0%
norm	FY23F	-0.3	-0.3	0.0%
(cps)	FY24F	-3.1	-3.1	0.0%
DPS:	FY22F	0.0	0.0	0.0%
(cps)	FY23F	0.0	0.0	0.0%
	FY24F	0.0	0.0	0.0%
Price target:		0.63	0.63	0.0%
Rating:		O/W	O/W	

Growth rates

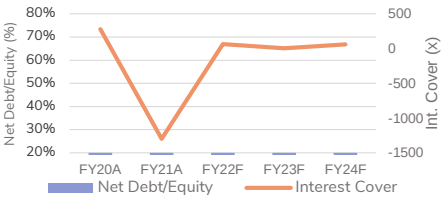


Returns

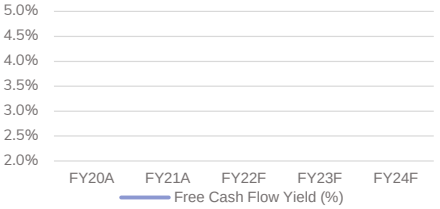


Margin trends

Solvency



Free cash flow yield



Interims ($m)

	1H21A	2H21A	1H22E	2H22E
Sales revenue	0.0	0.0	0.0	0.0
EBITDA	-2.1	-6.1	-5.7	-7.6
EBIT	-2.0	-6.0	-5.8	-7.7
Net profit	**-2.0**	**-7.7**	**-5.7**	**-7.6**
Norm EPS	**-0.4**	**-1.1**	**-1.0**	**-1.0**
EBIT/sales (%)				
Dividend (c)	0.0	0.0	0.0	0.0
Franking (%)	0.0	0.0	0.0	0.0
Payout ratio (%)	0.0	0.0	0.0	0.0
Adj payout (%)	0.0	0.0	0.0	0.0

Key assumptions

	FY17A	FY18A	FY19A	FY20A	FY21A	FY22F	FY23F	FY24F
Revenue Growth (%)	-0.6	-0.4	1.2	0.2	-0.2	4.5	0.3	-0.6
EBIT Growth (%)	0.0	-0.2	0.3	1.0	0.4	0.7	-0.8	6.8
NPAT Growth (%)	0.1	-0.2	0.3	1.0	0.4	0.6	-0.8	7.9
EPS Growth (%)	**0.2**	**-0.3**	**-0.4**	**0.7**	**0.4**	**0.1**	**-0.8**	**7.9**
Tax Rate (%)	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
R&D Expenditure	**-1.1**	**-1.0**	**-1.8**	**-1.9**	**-10.0**	**-13.0**	**-5.0**	**-22.0**

Financial ratios

	FY17A	FY18A	FY19A	FY20A	FY21A	FY22F	FY23F	FY24F
PE (x)	-12.3	-17.5	-27.6	-16.2	-8.5	-10.4	-61.2	-6.9
EV/EBITDA (x)	-41.8	-49.5	-39.2	-19.8	-14.1	-8.6	-40.2	-5.0
Dividend yield (%)	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
FCF yield (%)	-2.4	-1.9	-2.4	-3.3	-4.8	-8.3	1.8	-17.7
Payout ratio (%)	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Adj payout (%)	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0

Profit and loss ($m)

	FY17A	FY18A	FY19A	FY20A	FY21A	FY22F	FY23F	FY24F
Sales revenue	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
EBITDA	-2.7	-2.3	-2.9	-5.8	-8.1	-13.4	-2.9	-22.9
Depn & amort	0.0	0.0	0.0	0.1	0.1	0.1	0.1	0.1
EBIT	**-2.7**	**-2.3**	**-2.9**	**-5.9**	**-8.0**	**-13.5**	**-3.0**	**-23.0**
Net interest expense	-0.1	0.0	-0.1	0.0	0.0	-0.2	-0.4	-0.4
Tax	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Minorities/pref divs	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Equity accounted NPAT	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Net profit (pre-sig items)	**-2.8**	**-2.3**	**-2.9**	**-5.9**	**-8.1**	**-13.3**	**-2.5**	**-22.6**
Abns/exts/signif	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Reported net profit	**-2.8**	**-2.3**	**-2.9**	**-5.9**	**-8.1**	**-13.3**	**-2.5**	**-22.6**

Cash flow ($m)

	FY17A	FY18A	FY19A	FY20A	FY21A	FY22F	FY23F	FY24F
EBITDA	-2.7	-2.3	-2.9	-5.8	-8.1	-13.4	-2.9	-22.9
Interest & tax	-0.1	0.0	-0.1	0.0	0.1	0.2	0.4	0.4
Working cap/other	-0.1	0.0	0.1	1.9	2.3	3.1	4.6	1.2
Operating cash flow	**-2.9**	**-2.3**	**-2.9**	**-3.9**	**-5.8**	**-10.0**	**2.1**	**-21.3**
Maintenance capex	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Free cash flow	**-2.9**	**-2.3**	**-2.9**	**-3.9**	**-5.8**	**-10.0**	**2.1**	**-21.3**
Dividends paid	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Growth capex	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Invest/disposals	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Oth investing/finance flows	-0.1	-2.7	2.3	-0.4	-0.7	-1.8	0.0	0.0
Cash flow pre-financing	**-3.0**	**-5.0**	**-0.6**	**-4.3**	**-6.5**	**-11.8**	**2.1**	**-21.3**
Funded by equity	0.1	5.0	1.6	5.5	8.5	30.0	0.0	0.0
Funded by debt	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Funded by cash	2.9	0.0	-1.0	-1.2	-2.0	-18.2	-2.1	21.3

Balance sheet summary ($m)

	FY17A	FY18A	FY19A	FY20A	FY21A	FY22F	FY23F	FY24F
Cash	1.9	1.9	2.9	4.1	6.0	41.1	43.3	22.0
Current receivables	0.4	0.3	0.6	0.7	0.6	0.8	0.8	1.1
Current inventories	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Net PPE	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Intangibles/capitalised	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Total assets	**2.6**	**4.8**	**3.7**	**5.4**	**7.0**	**42.7**	**44.8**	**23.8**
Current payables	0.4	0.3	0.6	0.3	0.5	0.5	0.7	0.6
Total debt	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Total liabilities	**0.7**	**0.6**	**0.9**	**0.8**	**1.3**	**1.1**	**1.3**	**1.2**
Shareholder equity	**1.9**	**4.2**	**2.8**	**4.5**	**5.7**	**41.6**	**43.5**	**22.6**
Total funds employed	**1.9**	**4.2**	**2.8**	**4.5**	**5.7**	**41.6**	**43.5**	**22.6**

New data supports future indication expansion

Genetic modifier changes identified driven by ATL1102 treatment. Antisense have presented new data at the World Muscle Society (WMS) meeting garnered from an analysis of patient plasma samples from their completed Phase II study in DMD.

Two key genetic modifiers, THBS1 and LTGFB4, that are related to the loss of ambulation (ability to walk) in DMD boys have been shown to be positively affected by ATL1102 treatment in their Phase II trial (**Table 1**). Simply, this data highlights that ATL1102 treatment could potentially be efficacious in the ambulant DMD population also (increasing their current DMD TAM by 100%), in addition to being applicable in other fibrotic indications (i.e. other muscular dystrophies, diabetic nephropathy etc). Antisense have filed a patent to protect these claims.

Loss of Ambulation (LoA) genetic modifiers. There are several known genetic modifiers of rate of ambulation loss; which include Latent TGF-β binding protein 4 (LTBP4) and thrombospondin-1 (THBS1). LTBP4 and THBS1 have been shown to be positively affected by ATL1102 treatment over a 6-month period (**Table 1**). These two modifiers act on tissue-growth factor β (TGF-β), which is a central mediator of fibrosis. Fibrosis is the process of creating fibrous connective tissue which in a healthy person can be a reparative response to injury. In DMD patients' fibrosis is excessive and detrimental (e.g. reduced muscle health; lung fibrosis affects breathing). Reducing fibrosis in DMD is a common drug target. Excessive fibrosis is also linked to loss of ambulation. Simply, the observed changes in THBS1(-49%) and LTBP4 (+20.7%) reflect positive effects on fibrotic mechanisms that have been shown to modify (i.e. slow) the rate of loss of ambulation. This may support benefit of ATL1102 treatment in ambulatory DMD.

Table 1. Key changes identified from Phase II sample analysis, relative to healthy controls (directional changes)

	DMD baseline realtive to healthy control	DMD ATL1102-treated relative to healthy control	Mean change from baseline with ATL1102 treatment	Meaning of change
DMD genetic modifiers of ambulation loss via TGF-beta modulation				
THBS1	1.04	0.71	**-49%**	Positive effects on TGF-beta
LTBP4	0.70	0.87	**21%**	Positive opposing effect on TGF-beta
Ligands associated with inflammatory processes and muscle regeneration				
sVCAM-1	0.77	0.93	**18%**	Supports mechansim, reducing CD49d binding of ligand sVCAM-1 (ergo elevated circulating levels)
sCXCL16	0.91	1.18	**30%**	Positive - CXCL16 associated with muscle regeneration.

All values shown are medians normalised to a healthy control population (1.0) to simplify the relative movements of these markers. A value below 1 shows a lowered relative level compared to a healthy control population.

Source: Antisense, Wilsons.

See page 5 for commentary on CXCL16 and VCAM1 changes.

Ambulant DMD population expands TAM by 100%. Expansion of the ATL1102 addressable market to include ambulant DMD populations doubles the patient pool, given that ambulant boys comprise ~50% of all DMD patients. We currently model ATL1102 potential market penetration of 25-30% between US and EU5 markets based on a non-ambulant population only; this represents total (ambulant + non-ambulant) DMD peak market penetration of 8-12% in our current model. There is future scope to expand this should ATL1102 progress to Phase II studies and show meaningful benefit. Noting that the approvable endpoint in ambulant populations is different as it focuses on walking ability (NSAA vs PUL2.0).

PIP includes scope for ambulant DMD patient clinical studies. Antisense have already included scope for evaluation of ATL1102 in ambulant populations within their PIP, just to ensure the PIP is comprehensive of future potential trial activities. The view that ATL1102 could be a relevant treatment for ambulant populations is not a new one, however this new data does support the potential of ATL1102 to modify or delay ambulation loss and therefore eventually moving it into trials of boys who still have some degree of ambulatory ability is a relevant step.

Does not affect current EU trial plans. This does not affect the current Phase IIb trial plans in Europe. We understand this trial is still to be focused on non-ambulant boys exclusively given the high unmet clinical need in this population and the positive Phase II data needed to support its progression to a pivotal Phase IIb study.

ATL1102 mechanism further supported

We preface the below commentary with the caveats of small (n=9), single arm, trial results from the Phase II study of ATL1102 upon which this analysis is based.

Lymphocyte changes correlate to clinical outcomes = proof of concept. When we look at absolute change to PUL2.0 scores from Baseline to W24 and compare to absolute change in CD4+CD49d+ lymphocyte number from W28 (as a proxy for baseline given no significant differences) and W24, we see a correlation (R^2=0.8, **Figure 1**) with the exception of two patients (**Table 2**).

In the majority of patients (7 of 9) we saw a negative correlation between CD4+CD49d+ lymphocyte number and PUL2.0 improvement (**Table 2; Figure 1**) which is supportive of the drug's proposed mechanism of action. 5 of 9 patients saw reductions in lymphocytes (14-45%) that correlated to stabilised or improved PUL2.0 scores. Keeping in mind that a zero point change in PUL2.0 over a six month period reflects ~2 point relative benefit when we compare to historical controls and placebo arms of other trials, where a 2 point loss in PUL2.0 score (i.e. -2) over 6 months is typical of disease progression.

Non-responders identified. In 2 of 9 patients we saw a correlation still however with detrimental outcomes (as expected). These two patients (006 & 011) had an elevation in CD4+CD49d+ lymphocyte number and worsening in PUL2.0 (at levels similar to historical controls and other placebo cohorts) suggesting these patients were potential ATL1102 treatment non-responders.

Variance suggests other mechanisms at play. In two patients (008 & 010) we saw no lymphocyte change (-2% and 0% respectively) associated with PUL2.0 improvements. These patient responses suggest other mechanisms may also be responsible, (or simply that this one biomarker doesn't accurately reflect complete inflammatory benefits), which is likely in a complex disease pathology. Patient 008 is a clear outlier in this Phase II study with a PUL2.0 improvement of +7 over the 6-month treatment period. All other patients fell within the -3 to +2 range. As a reminder (-2 is average loss in PUL2.0 over a 6-month timeframe observed in other DMD placebo cohorts and historical controls)[1,2].

Table 2. Lymphocyte changes and PUL2.0 changes from Phase II dataset

Patient ID	Baseline proxy CD4+CD49d+ lymphocytes ^ (cells 10^9/L)	End of treatment CD4+CD49d+ lymphocytes ^ (cells 10^9/L)	Change in CD4+CD49d+ lymphocytes^ (cells 10^9/L)	Change in PUL2.0	Lymphocyte change	Correlation#
01-001	1.50	0.83	-0.68	2	-45%	Yes
01-002	1.95	1.40	-0.55	2	-28%	Yes
01-003	0.83	0.60	-0.23	0	-27%	Yes
01-004	1.40	1.13	-0.28	2	-20%	Yes
01-006	0.55	0.73	0.18	-3	32%	Yes
01-008	1.50	1.48	-0.02	7	-2%	No
10-009	1.63	1.40	-0.23	0	-14%	Yes
01-010	0.38	0.38	0.00	0	0%	No
01-011	1.08	1.18	0.10	-2	9%	Yes

^This change is based on assessment of Week 24 (end of treatment) versus Week 28 (4 weeks post-treatment) changes, given that there are no significant differences in lymphocytes between W28 and baseline levels. This is due to the individual baseline values not being publically available (using W28 as baseline proxy following post-treatment reversion).

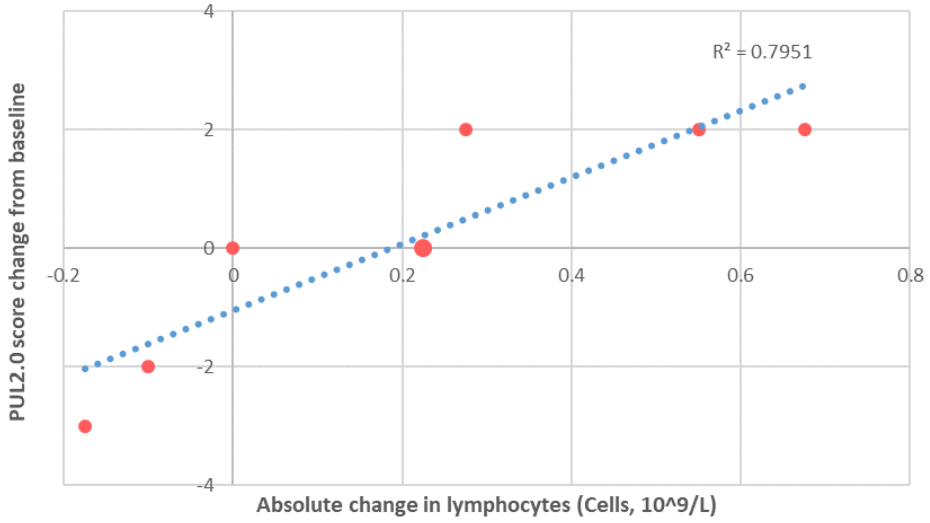
#Correlation is a negative correlation that reflects a relationship between lympocytes moving in a direction opposite to PUL2.0 score change.

Source: Antisense, Wilsons.

Reminder: PUL2.0 is the approvable primary endpoint in non-ambulant DMD. A change of 1.0 point is considered clinically meaningful.

[1] Tachas et al. 2020. ATL1102 treatment improves PUL2.0 in non-ambulant boys with Duchenne Muscular Dystrophy compared to a natural history control. *Neuromuscular Disorders*. 30(S1): S129-S130.
[2] Capricor Therapeutics NASDAQ Announcement: 7 Oct 2019. https://www.irdirect.net/prviewer/release/id/4066915

Figure 1. Positive correlation between lymphocyte changes and PUL2.0 changes in Phase II. One outlier patient has been removed.



The larger data point represents two patients (003 & 009). n=8 total shown.
Source: Wilsons.

Caveat of this being a small sample size (n=9) with one outlier patient. This outlier is not shown in **Figure 1** *to left. Patient 008 saw the most marked improvement in PUL2.0 score (+7) however had de minimis change in in CD49d lymphocyte number (-2%) suggesting other mechanisms were responsible for their improvement.*

A reminder on MRI data that was supportive of mechanism. Briefly, in DMD muscle mass is slowly transformed into fat mass which leads to the subsequent loss of ambulation and upper body muscle function The MRI data from their Phase II trial shows a positive link between ATL1102 treatment and stabilisation/improvement of lean muscle mass and fat mass ratios within patient limbs. This shows at a gross level that ATL1102 treatment is aiding in preventing the conversion from muscle to fat in these patients, and thus prolonging their upper limb muscle function.

Inflammatory signalling pathways positively affected by ATLL102 treatment in Phase II trial. New data presented by Antisense shows a modulation of soluble VCAM-1, an inflammatory molecule that is a known binding ligand of CD49d (the target of ATL1102). Increased levels of soluble VCAM-1 were measured at end of treatment (W24) compared to baseline (+18%) in patients treated with ATL1102 for 6 months. This is supportive of ATL1102's mechanism of action, in that reduction of CD49d on the surface of cells means there are less targets for soluble VCAM-1 to bind, ergo the increased circulating levels of this marker in the blood (unbound). Perhaps more importantly, the levels of soluble VCAM-1 in DMD boys were brought closer to a healthy external control range, showing normalisation of the inflammatory signalling processes (**Table 1**).

CXCL16 increases support muscle regeneration hypothesis. Similar outcomes were seen with increased soluble CXCL16 levels (+29.9% vs baseline) after ATL1102 treatment (**Table 1**). CXCL16 is a chemokine that has been shown preclinically to be linked to promotion and support of muscle regeneration and anti-fibrotic outcomes[3]. Levels of CXCL16 at study baseline were below external healthy controls however ATL1102 treatment appears to have boosted them to above healthy control levels, which in the context of a muscle degenerative disease is positive. It is postulated that CXCL16 increases could underly the reduced fat fraction percentage and retained muscle mass that was observed via MRI in the Phase II trial.

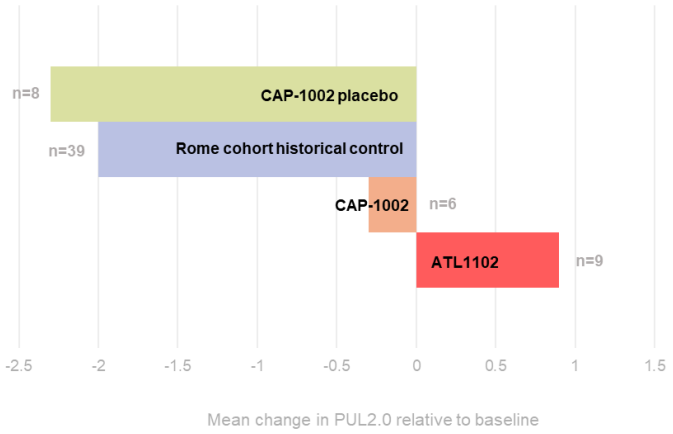
[3] Zhang et al. 2009. Chemokine CXCL16 Regulates Neutrophil and Macrophage Infiltration into Injured Muscle, Promoting muscle regeneration. *Am J Pathol.* 175(6): 2518-2527.

ATL1102 vs CAP-1002

Here we take the time to remind and refresh on ATL1102 relative efficacy versus peers, notably in comparison to Capricor Therapeutics' CAP-1002 cell therapy also being trialled in non-ambulant DMD boys. Complete Phase II trial data for the Capricor HOPE-2 study was presented at the WMS meeting last week which included the full 12-month treatment dataset, an update from the interim 6-month data we had previously evaluated. ATL1102's relative efficacy remains superior based on comparisons.

Figure 2. 6-month data comparison for ATL1102 and CAP-1002 in Phase II non-ambulant DMD boys.

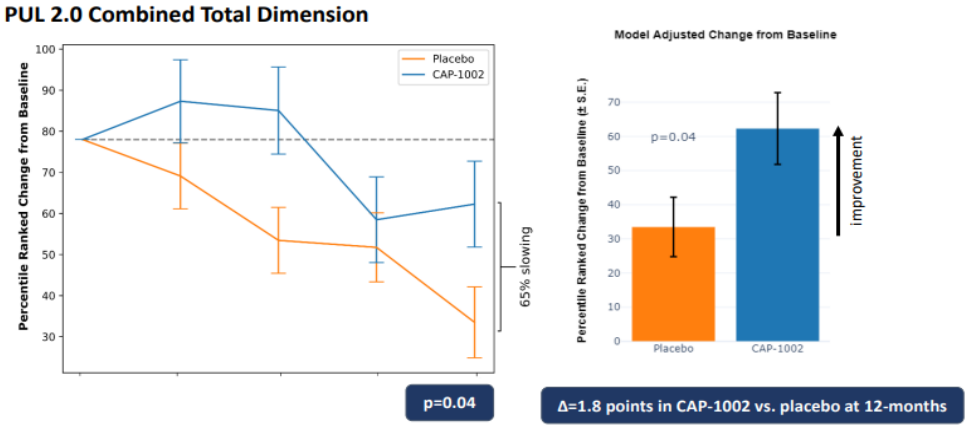
Include Capricor interim 6-month data set from WMS Oct 2019. We note an updated interim 6-month dataset showing 1.8 point difference at May 2020 (Placebo n=10; Capricor n=6).



Source: Wilsons, Capricor, Antisense.

Differences between Phase II programs. The key differences between the ATL1102 and CAP-1002 Phase II studies was the presence of a placebo arm in the Capricor trial and the longer 12-month dosing timeframe, both features of Antisense's Phase IIb design. Additionally, 20% of Capricor Phase II patients were ambulant as opposed to all non-ambulant patients in the ATL1102 study. Sample size were similar.

Figure 3. Final 12-month CAP-1002 PUL2.0 data from Phase II trial



Source: Capricor.

CAP-1002 maintained PUL2.0 benefit seen at 6 months out to 12-month timepoint. The incremental update from Capricor (**Figure 3**) showed that they achieved a 1.8 point benefit over placebo (p=0.04) with their treatment over a 12 month period. At the 6-month timepoint (**Figure 2)** Capricor reported a 2.0 point PUL2.0 advantage vs placebo in Oct 2019 (-2.3 in placebo vs -0.3 in CAP-1002 treated) which was updated to 1.8 at the May 2020 analysis (-2.1 placebo vs -0.3 CAP-1002). This compares to a potential 2.9 point advantage for ATL1102 when using a proxy historical matched control cohort in absence of a placebo arm. Even with the +7 outlier removed we assess a superior advantage for ATL1102 (2.13 point). We also note one outlier in the CAP-1002 data where a patient on treatment had a significant deterioration (-10 change in PUL2.0 score) over the treatment period. 2

Antisense Therapeutics Limited (ANP)

Business description

Antisense Therapeutics is a clinical stage biopharmaceutical company focused on development of antisense oligonucleotides targeting rare diseases. Their primary asset, ATL1102, is currently in Phase II trials for the treatment of Duchenne Muscular Dystrophy (DMD) with positive results thus far in the more advanced, non-ambulant disease population. Antisense have also conducted some advanced clinical work on ATL1102 as a treatment for multiple sclerosis (MS) and with another asset ATL1103, for the growth disorder, Acromegaly.

Investment thesis

We maintain our OVERWEIGHT recommendation on Antisense Therapeutics (ANP) and risked PT of $0.63 per share. Recent presentations at the World Muscle Society (WMS) meeting (20-24 Sept) from those working in DMD have reminded us that ATL1102 is positioned well versus peer approaches (i.e. Capricor) and that DMD gene therapies continue to struggle with safety setbacks (e.g. recent Pfizer narrowing of Phase III trial inclusion due to serious adverse events). Importantly, we have a growing understanding and comfort in the mechanistic basis of ATL1102's effects in DMD boys from the Phase II trial data but also from the recently presented proteomics analysis that has shown potential for ATL1102 to expand into ambulant DMD cohorts in the future. In this note we summarise these developments, outline the mechanistic data supporting ATL1102 thus far and provide a head to head update on ATL1102 efficacy vs CAP-1002, a competitor in the non-ambulant space that has just released final Phase II data. As a reminder, the key catalyst we await is feedback from the European regulator to bed down ANP's Phase IIb trial design and plans which is due at any point. This paves the way for CTA submission, approval and trial initiation, still planned for CY21.

Revenue drivers

Underlying growth in DMD market driven by greater diagnosis rates

Partnering transactions related to ATL1103 or ATL1102 assets with upfront payments/milestones and royalties

Balance sheet

- Cash of $6M as at 30 June 2021.

Margin drivers

- Not applicable.

Board

- Dr Charmaine Gittleson (Chairman)
- Mark Diamond (Managing Director)
- William Goolsbee (Non-executive Director)
- Robert Moses (Non-executive Director)
- Dr Graham Mitchell (Non-executive Director)
- Dr Gary Pace (Non-executive Director)

Key issues/catalysts

Clinical trial results

Regulatory interactions with EMA and FDA including CTA and/or IND approval of Phase IIb/III trials

Competitor development progress in DMD market

Partnering opportunities

Management

Mark Diamond (Chief Executive Officer)

Dr George Tachas (Director – Drug Discovery & Patents)

Phillip Hains (Chief Financial Officer)

Nuket Desem (Director of Clinical & Regulatory Affairs)

Dr Gil Price (Consultant Medical Director)

Alicia Mellors (Company Secretary)

Risk to view

Failure of ATL1102 to show adequate efficacy in DMD to achieve regulatory approvals

Development of superior disease modifying/curative drugs by competitors

Availability of capital to fund intensive period of R&D in near term with limited catalysts

Ability of management to deliver on commercialisation outcomes

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Definitions at wilsonsadvisory.com.au/disclosures.

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