

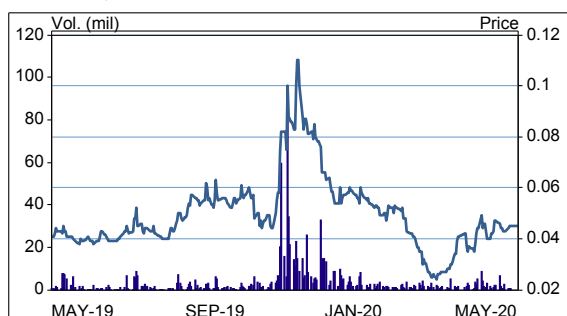
Prescient Therapeutics Limited (PTX.AX)
Rating: Buy

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Differentiated CAR-T Platform Technologies Licensed; Agnostic to Cell Type and Targets

Stock Data		05/25/2020		
Price		A\$0.05		
Exchange		ASX		
Price Target		A\$0.20		
52-Week High		A\$0.15		
52-Week Low		A\$0.02		
Enterprise Value (M)		A\$10		
Market Cap (M)		A\$18		
Public Market Float (M)		132.9		
Shares Outstanding (M)		394.3		
3 Month Avg Volume		1,957,227		
Balance Sheet Metrics				
Cash (M)		A\$8.20		
Total Debt (M)		A\$0.00		
Total Cash/Share		A\$0.02		
<i>General: U.S. to AUS exchange rate of 1.52901 on 5/25/20.</i>				
EPS (A\$) Diluted				
Full Year - Jun	2019A	2020E	2021E	
1st Half	(0.01)	(0.00)A	(0.01)	
2nd Half	(0.01)	(0.01)	(0.01)	
FY	(0.02)	(0.01)	(0.01)	
Revenue (A\$M)				
Full Year - Jun	2019A	2020E	2021E	
1st Half	0.0	0.0A	0.0	
2nd Half	0.0	0.0	0.0	
FY	0.0	0.0	0.0	

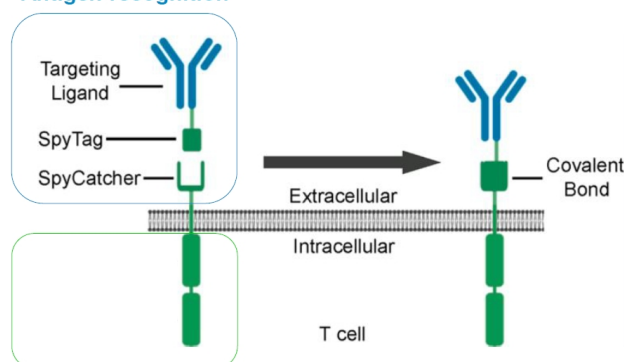
Quarterly EPS may not add to full year due to increases in share count and rounding.



OmniCAR could answer many questions for first-gen CAR-T approaches. Last night, Australian based Prescient hosted an investor call, and announced a licensing agreement with U. Penn and Oxford University for: (1) a universal immune receptor technology platform (U. Penn); and (2) a non-exclusive license with Oxford for the SpyTag/SpyCatcher molecular binding system, both with the goal of generating a potentially transformative approach to CAR-T therapy. The license encompasses an enabling platform called OmniCAR that could be beneficial for not only Prescient but potentially for multiple future licensing agreements. While Prescient continues to quickly manage its cash resources, this agreement is both non-dilutive, and importantly, has no immediate material financial impact for the company. U. Penn was looking toward a quick commercial path for initial use of the platform and therefore, the deal is more back-end loaded with typical milestones and royalties to be paid on potential revenue. The key differentiating feature of the OminCAR platform is that it is not only cell type agnostic (e.g., T-cell, NK cell, etc.) but is also completely agnostic to the target and targeting approach. This latter feature allows for broad adaptability to dose patients sequentially with a single target CAR-T or concomitantly with multi-targeting cells. The crux of the technology is shown in the figure below, but in short, the SpyTag linker is attached to any tumor-targeting ligand (e.g., scFv, antibody, aptamer, imaging labels), which then forms a covalent bond to SpyCatcher on the cell's membrane, creating the specific targeting cell. This covalent bond with any ligand(s) of choice decouples antigen recognition with signaling domains.

OmniCar Modular Characteristics Present Broad Applicability

Antigen recognition



Source: May 26, 2020 Prescient OmniCAR investor presentation.

Appears that safety could be better controlled. While physicians are becoming more knowledgeable in controlling the harsh side effects of CAR-Ts, OmniCAR could give docs more control post infusion by: (1) controlling the subsequent dose of the targeting ligand; and (2) could titrate the dose to safe and efficacious levels more efficiently. In this case, the proverbial "kill switch" is the removal of the targeting ligand at-will should any toxicity arise, which could then be subsequently be restarted.

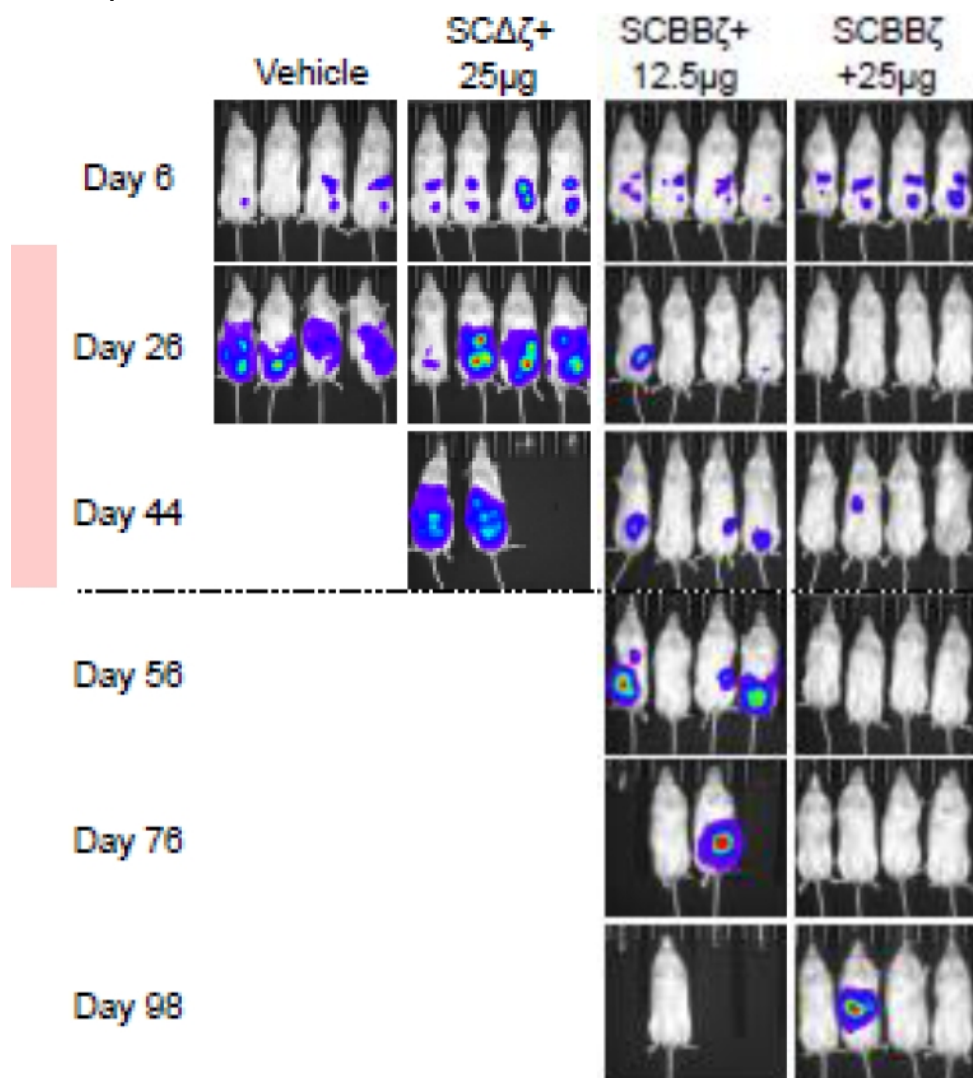


Solid tumor teaser. We are intrigued by the announcement of the OmniCAR platform for Prescient's future. One of the key factors we focused on with management is the utility in solid tumor cells. As most are aware, the effective use of CAR-T approaches in solid tumors has been elusive, so the question presents as to why OmniCAR could answer this ongoing problem. One of the leading hypotheses for why CAR-Ts do not work in solid tumors is complexity of the tumor microenvironment (TME), which could prevent access of CAR-Ts to the tumor. Management gave an important teaser regarding this issue. We look forward with great anticipation to further updates, but the approach could revolve around an OmniCAR approach that has a cell expressing both: (1) a targeting ligand; and (1) a chemokine receptor or ligand; the latter could be the key to gaining access into the TME and directing the CAR-T to the tumor cells.

Business development could be quite bright for Prescient and OmniCAR. As the company develops the technology and delivers proof-of-concept data, we believe business development could represent a bright future for the company. This could occur based on potential partners' underlying targeting ligands and the wish to link them to a powerful CAR-T approach. While the technology is early, we believe that Prescient could deliver on its first non-dilutive deal relatively quickly providing external validation for the technology, and deliver more lucrative deals as the technology begins to mature.

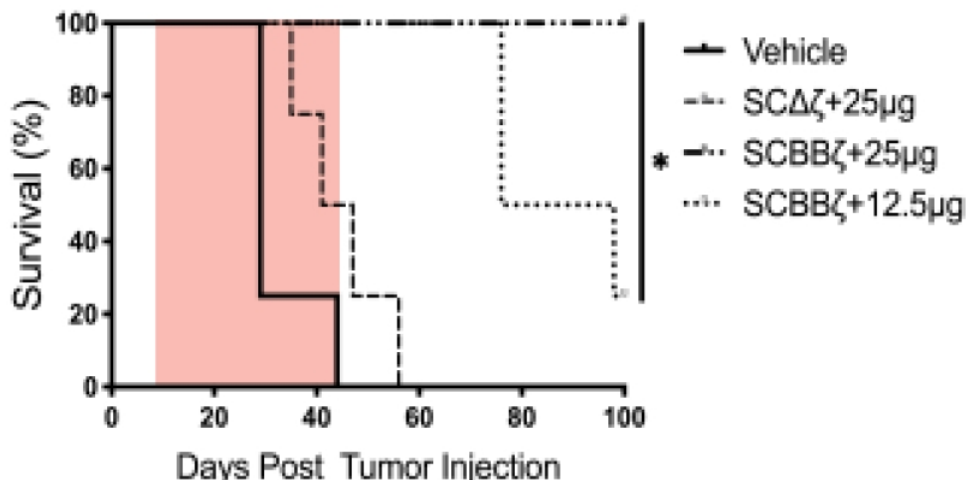
Early data show dose-dependent impact of OmniCAR approach; multi-targeting also shows specificity. Data have already been generated showing preclinical efficacy of the OmniCAR approach. One aspect of these data show a dose-dependent positive impact on an ovarian cancer model using an anti-HER2 OmniCAR. The first figure shows that loading more targeting ligand (binder) leads to proportionate killing in the animals; the second figure shows the Kaplan-Meier survival curves for the animals also showing the dose-dependent impact on survival. Importantly, the pink shading in both figures denotes when the animals were dosed; the survival curves show long-term survival even when dosing of the binder was ceased after Day 44.

Dose-Dependent Tumor Control With Anti-HER2 OmniCAR



Source: Powell, DJ et al., JACS; 2020.

Dose-Dependent Survival Impact With Anti-HER2 OmniCAR



Source: Powell, DJ et al., JACS; 2020.

PTX-200 path in breast cancer continues to make sense. In December 2019, Prescient announced the anticipated update from its ongoing Phase 2a study using PTX-200 in HER2 negative, locally advanced breast cancer. The data are described further below, but in short: (1) overall response rate (ORR) of 91%; (2) two patients had pathologic complete responses (pCR); (3) one patient with clinical complete response (cCR); and (4) durability continues to be encouraging with nine of 10 evaluable patients free of disease progression to date (up to 40 months of disease-free progression). The study enrolled 11 patients with HER2-negative tumors; nine with ER+ disease and two with triple negative disease. While the data are from a small group of patients, they are still encouraging, in our belief.

Intriguing responses. The patient that achieved the cCR was termed non-evaluable due death prior to scheduled surgery due to adverse events (cardiac complications) from doxorubicin, and unrelated to PTX-200. However, the patient had a large ER+ that had a complete response following PTX-200 and paclitaxel. The Simon two-stage design's goal was to demonstrate three pCRs in the first eleven patients; two pCRs were seen, and the cCR was confirmed by autopsy, which appears to have established proof-of-concept for the study. While it is difficult to compare data interstudy, the expected pCR rate from locally advanced ER+ and HER2 negative is approximately 16% (Green et al. *J Clin Oncol* 23; 2005). A summary of the responses is found in the table below.

Ongoing PTX-200 Positive Efficacy Results in Locally Advanced Breast Cancer

	ER positive	Triple negative	Total
pCR	2	0	2
pPR	6	2	8
SD	0	0	0
PD	0	0	0
NE (cCR)*	1	0	
ORR			90.9%

Source: December 23, 2019 PTX release.

Durability appears to be pointing in the right direction. Progression-free intervals in the study are encouraging thus far. The current PFS ranges from 6.7 to 40 months (average of 22 months), with nine of the 10 patients being progression-free, to date. Assessment of overall survival (OS) continues, and is currently 22.4 months. The benchmark for durability is the 24-month mark, with many women progressing within this period.

Next steps to add further personalization. According to management, the data point to moving the study into ER positive disease, which appears to be the most responsive group. The goal is to combine PTX-200 with hormone therapy (SoC for locally advanced ER positive tumors). Investigators have stated to management their expectation of a more favorable safety profile vs. the current combination with chemotherapy (paclitaxel, doxorubicin-cyclophosphamide). In consideration of costs, the company is seeking to conduct the study in Australia, potentially in conjunction with an investigator-sponsored study to help defer costs.

P-Akt is a promising pharmacological target in AML. Regarding the relevance of targeting Akt in AML, we highlight that, despite this disease being highly dependent on a wide range of oncogenic genetic aberrations, it is also subject to the activation of defined tumor niches, where survival factors including hyperactivated Akt may play a role. To this end, high p-Akt levels are correlated with inferior survival in AML (Nepstad et al., *Cancers* 2018; Liang et al., *Sci Rep* 2017)

Long-term focus is on novel targeted compounds for therapy resistant cancers devoid of treatment options. Prescient's assets share a key feature of being not only targeted, but also being able to tackle a wide spectrum of malignancies. The company's clinical focus is on those cancers or metastases that become addicted to Akt, RAS and downstream effectors of these two clinically relevant oncogenes (e.g., RhoA). Of note, market opportunities are significant and we think underappreciated as these two targets alone could trigger tumorigenesis in the vast majority of cancers spanning hematological and solid tumors (e.g., pancreatic cancer or PDAC). For example, regarding PDAC, KRAS is the major tumor driver, and there are currently no targeted therapies, representing a significant unmet clinical need, in our view. Thus, as the clinical programs mature, we think that Prescient could potentially deliver new therapies for key diseases including aggressive cancers. For a more detailed analysis of our investment thesis on Prescient, refer to our initiation document here referenced: ([*Targeted Oncology From Down Under: Initiating Coverage at Buy and A\\$0.20 PT*](#)).

PTX-200 rationale in breast cancer; program in HTR breast cancer represents the near-term value driver for the shares; 4Q19 data readout. There are multiple PI3K and Akt inhibitors in clinical trials; however, preliminary data, literature, and our due diligence suggest that PTX-200 therapy could be successful in HTR BCa, where others have faltered thus far. Importantly, we highlight the strong scientific rationale for targeting Akt signaling in HTR cancer, which includes: (1) increased PI3K/Akt genetic aberrations lead to higher tumor growth; (2) higher dependency on PI3K/Akt pathways in patients that do not respond to targeted therapy (ERa inhibitors) and/or chemo; (3) Akt drives increased estrogen receptor (ERa) activity following the inhibition of the pathway from hormonal therapies (HT); and (4) there is a ubiquitous and strong consensus from clinical and laboratory practices highlighting the need for targeting Akt in HTR tumors. Overall, we believe that PTX-200 may deliver promising data in HTR disease with the potential of becoming a new therapy for these patients.

Valuation and risks to price target achievement. We maintain our Buy rating and A\$0.20 price target. Our valuation is based on our clinical net present value (NPV) model, which allows us to flex multiple assumptions affecting a drug's potential commercial profile. Our valuation is currently based on PTX-200 in breast cancer: (1) 84% contribution from HTR-BCa; and (2) 16% contribution from TNBCa. We currently do not include AML and ovarian in our projections, as well as any indications for PTX-100, both of which we consider to be free call options currently in our valuation. As the programs progress and data are released, we would look to reassess potential contribution from these indications and assets. Factors which could impede reaching our price target include failed or inconclusive clinical trials or inability of the company to secure adequate funding to progress its drugs through the development pathway.

(AUD\$ in millions except per share data)

Profit & Loss - June fiscal	2016A	2017A	2018A	2019A	2020E	2021E	2022E	2023E	2024E	2025E	2026E	2027E	2028E	2029E
Licensing	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
R&D collaborations	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Product and Royalties	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	2.0	44.0	93.0	196.0	396.7	416.8
Other revenues	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Revenues	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	2.0	44.0	93.0	196.0	396.7	416.8
CoGS	0.0	0.0	0.0	0.0	0.0	0.0	0.3	6.7	14.0	29.3	46.2	64.7	68.9	72.7
Gross Profit	0.0	0.0	0.0	0.0	0.0	0.0	(0.3)	(6.7)	(12.0)	14.7	46.8	131.3	327.9	344.1
<i>Gross margin</i>	0%	0%	0%	0%	0%	0%	0%	0%	-598%	33%	50%	67%	83%	83%
SG&A	2.0	1.4	1.6	1.8	2.1	2.6	4.6	6.0	6.6	7.1	7.7	8.0	8.4	9.1
R&D	0.8	2.4	2.1	3.7	3.7	6.9	8.0	10.0	18.6	19.7	21.2	22.7	23.4	24.8
Other op ex	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
EBIT	(2.8)	(3.8)	(3.6)	(5.5)	(5.8)	(9.4)	(12.9)	(22.6)	(37.1)	(12.1)	17.9	100.6	296.0	310.2
<i>EBIT margin</i>	<i>nm</i>	<i>nm</i>	<i>nm</i>	<i>nm</i>	<i>nm</i>	<i>nm</i>	<i>nm</i>	<i>nm</i>	<i>nm</i>	<i>nm</i>	19%	51%	75%	74%
Depreciation	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Amortisation Intangibles	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
EBITDA	(2.8)	(3.8)	(3.6)	(5.5)	(5.8)	(9.4)	(12.9)	(22.6)	(37.1)	(12.1)	17.9	100.6	296.0	310.2
<i>EBITDA margin</i>	<i>nm</i>	<i>nm</i>	<i>nm</i>	<i>nm</i>	<i>nm</i>	<i>nm</i>	<i>nm</i>	<i>nm</i>	<i>nm</i>	<i>nm</i>	19%	51%	75%	74%
Non operating expenses	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Net Interest Income/Other	1.0	1.2	1.1	1.7	1.6	3.0	3.5	4.3	8.1	8.6	9.2	9.9	10.2	10.8
Interest expense	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
EBT	(1.8)	(2.6)	(2.6)	(3.8)	(4.2)	(6.5)	(9.5)	(18.3)	(29.0)	(3.5)	27.1	110.5	306.2	321.0
<i>EBT margin</i>	<i>nm</i>	<i>nm</i>	<i>nm</i>	<i>nm</i>	<i>nm</i>	<i>nm</i>	<i>nm</i>	<i>nm</i>	<i>nm</i>	<i>nm</i>	29%	56%	77%	77%
Provision for taxes	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	(1.1)	8.1	33.1	91.9	96.3
Net Income	(1.8)	(2.6)	(2.6)	(3.8)	(4.2)	(6.5)	(9.5)	(18.3)	(29.0)	(3.5)	27.1	110.5	306.2	321.0
Participation of preferred stock	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Net Income to common	(1.8)	(2.6)	(2.6)	(3.8)	(4.2)	(6.5)	(9.5)	(18.3)	(29.0)	(2.5)	19.0	77.3	214.3	224.7
<i>net margin</i>	<i>nm</i>	<i>nm</i>	<i>nm</i>	<i>nm</i>	<i>nm</i>	<i>nm</i>	<i>nm</i>	<i>nm</i>	<i>nm</i>	<i>nm</i>	20%	39%	54%	54%
NoSH	82.2	210.0	211.4	244.5	396.1	545.0	632.2	644.8	696.4	752.1	812.3	877.3	947.5	1,023.3
EPS - basic	(0.02)	(0.01)	(0.01)	(0.02)	(0.01)	(0.01)	(0.01)	(0.03)	(0.04)	(0.00)	0.02	0.09	0.23	0.22
EPS - diluted	(0.02)	(0.01)	(0.01)	(0.02)	(0.01)	(0.01)	(0.01)	(0.03)	(0.04)	(0.00)	0.02	0.09	0.22	0.21

Source: Company reports and H.C. Wainwright estimates

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Half-yearly P&L - June fiscal	Dec	June	June	Dec	June	June	Dec	June	June	Dec	June	June
AUD\$ in millions	FH1'18A	FH2'18A	FY'18A	FH1'19A	FH2'19A	FY'19A	FH1'20A	FH1'20E	FH1'20E	FH1'21E	FH1'21E	FH1'21E
Licensing	0.00	0.00	0.0	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
R&D collaborations	0.00	0.00	0.0	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
Product and Royalties	0.00	0.00	0.0	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
Other revenues	0.00	0.00	0.0	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
Revenues	0.00	0.00	0.0	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
CoGS	0.00	0.00	0.0	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
Gross Profit	0.00	0.00	0.0	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
Gross margin	100%	100%	0%	100%	100%	0%	100%	100%	0%	100%	100%	0%
SG&A	0.74	0.84	1.6	0.81	1.02	1.83	1.01	1.04	2.05	1.20	1.37	2.57
R&D	0.90	1.16	2.1	1.54	2.14	3.68	1.33	2.39	3.72	2.85	4.03	6.88
Other op ex	0.00	0.00	0.0	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
EBITDA	(1.6)	(2.0)	(3.6)	(2.3)	(3.2)	(5.5)	(2.3)	(3.4)	(5.8)	(4.1)	(5.4)	(9.4)
EBITDA margin			nm			nm			nm			nm
Non operating expenses	0.00	0.00	0.0	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
Net Interest Income/Other	0.46	0.61	1.1	0.75	0.97	1.72	0.64	0.98	1.62	1.50	1.49	2.99
Interest expense	0.00	0.00	0.0	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
EBT	(1.2)	(1.4)	(2.6)	(1.6)	(3.9)	(5.5)	(1.7)	(2.5)	(4.2)	(2.6)	(3.91)	(6.5)
EBT margin			nm									
Provision for taxes	0.00	0.00	0.0	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
Participation of preferred stock												
Net Income to common	(1.2)	(1.4)	(2.6)	(1.6)	(2.2)	(3.8)	(1.7)	(2.5)	(4.2)	(2.6)	(3.9)	(6.5)
net margin			nm									
NoSH - basic	209.96	211.37	211.37	211.88	310.50	244.55	394.26	398.00	396.13	515.00	575.00	545.00
NoSH - diluted	209.96	211.37	211.37	211.88	310.50	244.55	394.26	398.00	396.13	515.00	575.00	545.00
EPS - basic	(0.01)	(0.01)	(0.01)	(0.01)	(0.01)	(0.02)	(0.004)	(0.006)	(0.010)	(0.005)	(0.007)	(0.012)
EPS - diluted	(0.01)	(0.01)	(0.01)	(0.01)	(0.01)	(0.02)	(0.004)	(0.006)	(0.010)	(0.005)	(0.007)	(0.012)

Source: Company filings and H.C. Wainwright estimates

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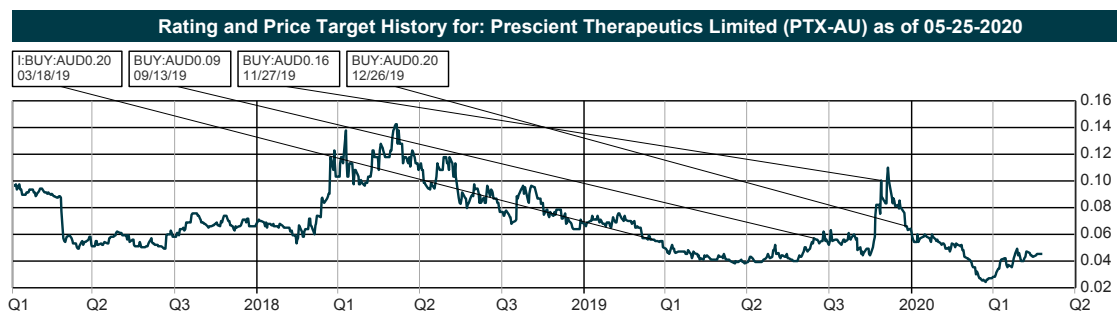
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RETURN ASSESSMENT

Market Outperform (Buy): The common stock of the company is expected to outperform a passive index comprised of all the common stock of companies within the same sector.

Market Perform (Neutral): The common stock of the company is expected to mimic the performance of a passive index comprised of all the common stock of companies within the same sector.

Market Underperform (Sell): The common stock of the company is expected to underperform a passive index comprised of all the common stock of companies within the same sector.



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Distribution of Ratings Table as of May 25, 2020				
Ratings	Count	Percent	IB Service/Past 12 Months	
			Count	Percent
Buy	380	90.26%	130	34.21%
Neutral	38	9.03%	7	18.42%
Sell	0	0.00%	0	0.00%
Under Review	3	0.71%	3	100.00%

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