



Immutep (IMM)

Leading not LAGging

Our View

Immutep is developing therapies based on LAG-3 through in-house programs and licensing deals with big pharma. Its share price fell last month when the progression free survival benefit from adding its antigen presenting cell activator 'efti' to chemo in the AIPAC Phase IIb breast cancer study failed to reach statistical significance. However, the higher tumour response rate in AIPAC (48% vs 38%) indicates that efti activates the immune system. We view this as a strong positive indicator for the continued success of ongoing studies of efti plus the immune checkpoint inhibitors (ICI) Keytruda and Bavencio – response rates for melanoma, lung and head and neck cancers have been at least 50% higher than the rates reported for Keytruda alone. ICI combos are where the real value in efti lies, in our view. IMM's value is further supported by Phase II LAG-3 programs out-licensed to Novartis and GSK. We initiate coverage with a risk adjusted DCF valuation of A\$327m or \$0.53/sh fully diluted (\$0.83/sh undiluted).

Key Points

Efti plus chemo not sufficiently active in metastatic breast cancer – Immutep's lead in-house product, efti, delivered only modest, non-significant improvements in tumour response rates and progression free survival when to chemotherapy in breast cancer patients in AIPAC. We did not believe that combining eft with chemo was the optimum therapeutic approach, as the immunosuppressive local environment within tumours can shut down anti-tumour immune responses. On the other hand, efti's mechanism of action of activating antigen presenting cells to initiate an immune response (pushing on the accelerator) is complementary to checkpoint inhibitors which 'take the brakes off' anticancer immune responses. We do not expect IMM to invest further funds in the efti/chemo combo in breast cancer unless new data come to light.

A promising checkpoint inhibitor combo – ICI drugs such as Keytruda and Opdivo have transformed the treatment of a number of cancers in recent years. However, only a minority of patients respond to ICI therapy, and treatments which can be combined with ICI drugs to improve response rates are being urgently sought. Efti has reported high response rates from ICI combo studies which should attract attention from potential pharma partners. The inclusion of IMM's update on the ICI combo study in the **high impact paper session** at the AACR conference on **27/28 April** suggests the data could be very encouraging.

By year-end data set will be comparable to Viralytics at time of \$500m Merck deal. Immutep's strategy for efti is similar to that pursued by Viralytics in its development of the Cavatak immuno-oncology therapy, which led to its acquisition by Merck for ~\$500m in 2018. Within the next 12 months Immutep should have data on efti plus Keytruda in 120-130 patients with melanoma, lung and head and neck cancer. This should be enough data for Merck to get a good understanding of how well the drug works. If efti is an effective therapy, in our view it should be worth more than Viralytics because it can be used in a much wider range of cancer types. We do not believe that the potential for a significant deal to occur in the next 12-18 months is reflected in the current share price.

Out-licensed LAG-3 programs in Phase II with big pharma - Immutep has two additional LAG-3 programmes that that are partnered with big pharma and could create significant value; both of these programs have recently progressed to Phase II (mid-stage) trials which represent significant investments by the pharma partner. These programs are an anti-LAG-3 antibody in cancer partnered with Novartis and a LAG-3 depleting antibody in inflammatory disorders partnered with and GSK.

22 April 2020

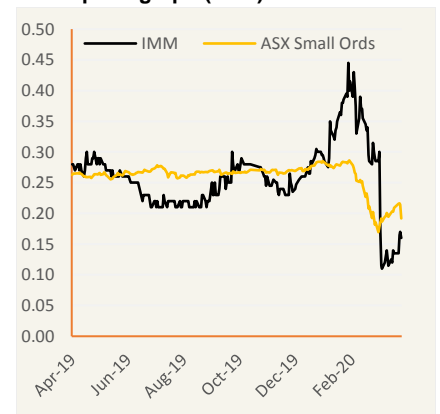
Speculative Investment

Recommendation: Outperform

Summary (AUD)

Market Capitalisation	\$59M
Share price	\$0.15
52 week low	\$0.11
52 week high	\$0.49
Cash as at 31 March 2020	\$16.1m

Share price graph (AUD)



Key Financials (AUD)

	FY19A	FY20E	FY21E
Revenue	6.6	13.0	5.6
R&D	(16.6)	(19.8)	(14.0)
SG&A	(7.4)	(7.1)	(7.3)
EBITDA	(17.4)	(13.9)	(15.7)
Reported NPAT	(18.3)	(13.8)	(15.6)
NPAT Adj.	(18.3)	(13.8)	(15.6)
EPS Adj. (c)	(5.7)	(3.8)	(3.2)
PE ratio (x)	n/a	n/a	n/a
DPS (c)	0.0	0.0	0.0
EV/Sales	n/a	n/a	n/a
EV/EBITDA (x)	n/a	n/a	n/a
ROE	n/a	n/a	n/a

Immutep - Summary of Forecasts

IMM \$ 0.15

PROFIT & LOSS SUMMARY (A\$m)

Year end June	FY19A	FY20E	FY21E	FY22E
Sales, royalties, milestones	0.1	7.4	0.0	9.5
Other (includes R&D tax rebate)	6.5	5.6	5.6	5.6
Total Revenue	6.6	13.0	5.6	15.1
Growth (pcp)	-3.7%	96.4%	-56.8%	169.7%
R&D Expenses	(16.6)	(19.8)	(14.0)	(14.0)
SG&A expenses	(7.4)	(7.1)	(7.3)	(7.5)
EBITDA	(17.4)	(13.9)	(15.7)	(6.4)
Dep'n/Other Amort'n	(1.9)	(0.0)	(0.0)	(0.0)
EBIT	(19.2)	(14.0)	(15.7)	(6.5)
Net Interest	0.4	0.2	0.1	0.3
Pre- Tax Profit	(18.3)	(13.8)	(15.6)	(6.2)
Tax Expense	0.0	0.0	0.0	0.0
Minorities	0.0	0.0	0.0	0.0
NPAT Adj.	(18.3)	(13.8)	(15.6)	(6.2)
Growth (pcp)	n/a	n/a	n/a	n/a
Adjustments	0.0	0.0	0.0	0.0
NPAT Reported	(18.3)	(13.8)	(15.6)	(6.2)

PER SHARE DATA

Year end June	FY19A	FY20E	FY21E	FY22E
EPS (c) - Reported	(5.7)	(3.8)	(3.2)	(1.1)
Growth (pcp)	n/a	n/a	n/a	n/a
EPS (c) - Adjusted	(5.7)	(3.8)	(3.2)	(1.1)
Growth (pcp)	n/a	n/a	n/a	n/a
Dividend (c)	0.0	0.0	0.0	0.0
Franking	0.0	0.0	0.0	0.0
Gross CF per share (c)	(4.7)	(3.5)	(3.0)	(0.9)
NTA per share (c)	2.2	1.0	3.0	2.1

KEY RATIOS

Year end June	FY19A	FY20E	FY21E	FY22E
Net Debt : Equity (%)	-67.9%	-62.1%	-76.9%	-72.7%
Net Debt: EBITDA (x)	1.0	0.9	1.7	3.3
Current ratio (x)	4.4	3.8	6.3	5.3
ROE (%)	-63.4%	-60.9%	-56.4%	-19.5%
ROIC (%)	n/a	n/a	n/a	n/a
Dividend Payout Ratio (%)	n/a	n/a	n/a	n/a

VALUATION MULTIPLES

Year end June	FY19A	FY20E	FY21E	FY22E
PE Ratio (x)	n/a	n/a	n/a	n/a
Dividend Yield (%)	0.0%	0.0%	0.0%	0.0%
EV/Sales (x)	n/a	n/a	n/a	n/a
EV/EBITDA (x)	n/a	n/a	n/a	n/a
EV/EBIT (x)	n/a	n/a	n/a	n/a

CAPITAL RAISING ASSUMPTIONS

Year end June	FY19A	FY20E	FY21E	FY22E
Shares Issued (m)	26.0	47.8	187.5	0.0
Issue Price (A\$)	0.34	0.21	0.16	
Cash Raised (A\$m)	8.8	10.0	30.0	

BALANCE SHEET SUMMARY

Year end June	FY19A	FY20E	FY21E	FY22E
Cash	16.6	13.0	26.5	21.4
Receivables	5.2	5.2	5.2	5.2
Inventories	0.0	0.0	0.0	0.0
Other	1.8	1.8	1.8	1.8
Total Current Assets	23.5	19.9	33.5	28.3
Inventories	0.0	0.0	0.0	0.0
Property Plant & Equip	0.1	0.1	0.2	0.3
Intangibles	16.9	16.9	16.9	16.9
Other	0.0	0.0	0.0	0.0
Total Current Assets	17.0	17.1	17.2	17.2
TOTAL ASSETS	40.5	37.0	50.7	45.5
Accounts Payable	5.1	5.1	5.1	5.1
Borrowings	0.0	0.0	0.0	0.0
Provisions	0.2	0.2	0.2	0.2
Other	0.0	0.0	0.0	0.0
Total Current Liab	5.3	5.3	5.3	5.3
Borrowings	0.0	0.0	0.0	0.0
Provisions	0.0	0.0	0.0	0.0
Other	10.9	10.9	10.9	10.9
Total Non-Current Liab	10.9	10.9	10.9	10.9
TOTAL LIABILITIES	16.2	16.2	16.2	16.2
TOTAL EQUITY	24.4	20.9	34.5	29.4

CASH FLOW SUMMARY

Year end June	FY19A	FY20E	FY21E	FY22E
EBIT (excl Abs/Extr)	(19.2)	(14.0)	(15.7)	(6.5)
Add: Dep'n & Amort'n	1.9	0.0	0.0	0.0
Other non- cash items	(4.5)	(1.4)	(1.4)	(1.7)
Less: Tax paid	0.0	0.0	0.0	0.0
Net Interest	0.4	0.2	0.1	0.3
Change in Rec.	(1.8)	0.0	0.0	0.0
Change in Inv.	0.0	0.0	0.0	0.0
Gross Cashflows	(15.3)	(12.7)	(14.5)	(5.1)
Capex	(0.0)	(0.1)	(0.1)	(0.1)
Free Cashflows	(15.3)	(12.8)	(14.6)	(5.2)
Share Issue Proceeds	8.0	9.2	28.2	0.0
Other	0.4	0.0	0.0	0.0
Dividends Paid	0.0	0.0	0.0	0.0
Net Cashflows	(6.9)	(3.6)	13.6	(5.2)
FX Effect on Cash	0.4	0.0	0.0	0.0

IMM base case valuation summary (undiluted)

	Probability/valuation (%)	Value (A\$m)	Value A\$/share
efti/ICI NSC lung cancer	15%	96.5	0.25
efti/ICI head & neck cancer	15%	46.4	0.12
efti/ICI melanoma	15%	11.4	0.03
efti/chemo breast cancer	2%	6.8	0.02
efti milestones - partner post TACTI-002	15-50%	80.5	0.21
LAG525 solid tumours (lung cancer)	20%	54.7	0.14
GSK'781- ulcerative colitis	20%	54.9	0.14
SG&A	-	(23.8)	(0.06)
Portfolio total	-	327.4	0.84
Net cash end FY20e (incl conv note face val)	-	(0.8)	(0.00)
Total Valuation	-	326.7	0.83

Overview

Immutep is an ASX-listed immunotherapy company that is focused on developing products based on the lymphocyte activation gene 3 (LAG-3) pathway, following the acquisition of a private French immunotherapy company in December 2014. Two of its pipeline products, efti and LAG525, enhance anti-cancer immune responses. Two other products, GSK'781 and IMP761, target LAG-3 to suppress undesirable immune responses in auto immune disease. Exhibit 1 summarises the 4 pipeline products and Exhibit 2 illustrates how they target the immune system.

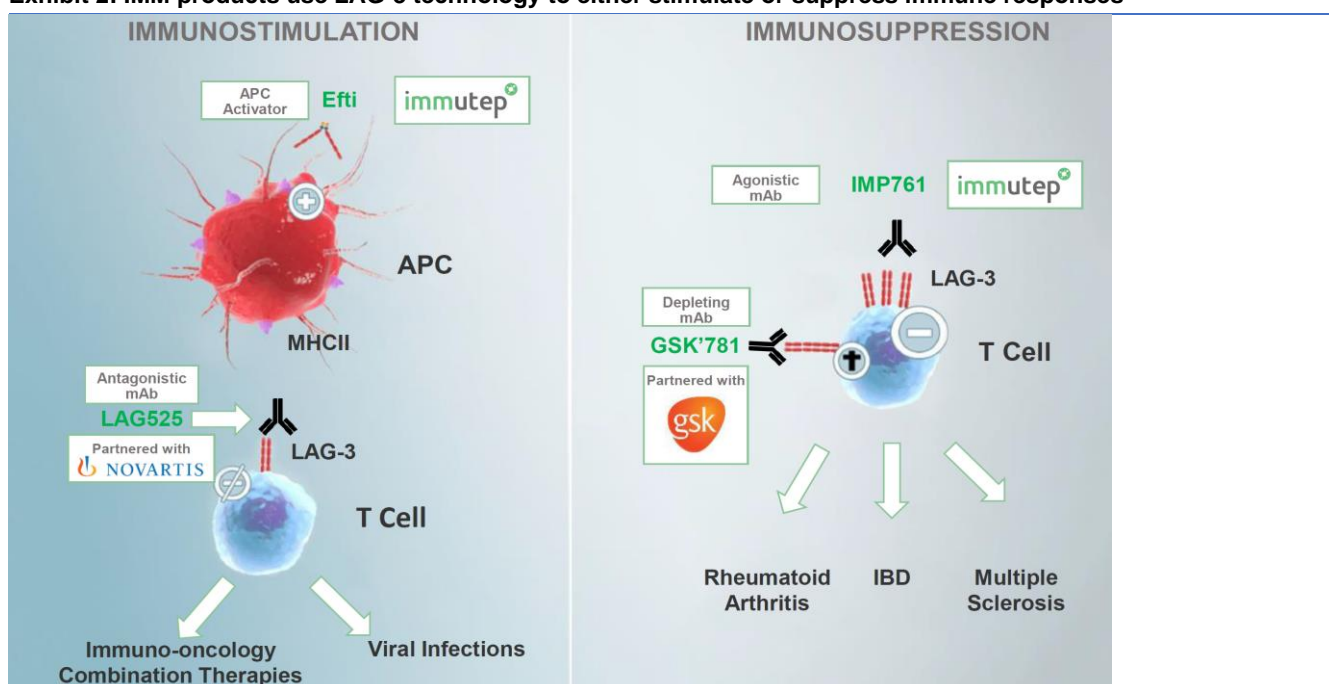
Its lead product, efti (eftilagimod alpha; IMP321), is in Phase II in combination with the checkpoint inhibitor Keytruda in lung and head and neck cancers. Immutep retains all the product rights to efti except for China (where it is partnered with EOC Pharma). LAG525, which is licensed to Novartis, is in Phase II studies for solid tumours and lymphoma in combination with a checkpoint inhibitor. GSK'781, which is licensed to GSK, progressed to a Phase II study for ulcerative colitis in 2019, earning Immutep a GBP4m (A\$7.4m) milestone payment. Its newest product candidate, IMP761 for autoimmune disease, is currently undergoing manufacture in preparation for a Phase I clinical trial. The company has facilities in Paris, France; Leipzig and Berlin, Germany; and its headquarters are in Sydney, Australia.

Exhibit 1: Immutep's pipeline of LAG-3 products

Product /Partner	Indication	Status	Notes
efti/EOC, (China)/ Merck & Co clinical trial collaboration/ Merck KGaA & Pfizer clinical trial collaboration	lung cancer, head and neck cancer & melanoma + Keytruda;	Phase II	Clinical trials underway of efti antigen-presenting cell activator combined with immune checkpoint inhibitors. Efti/Keytruda combo studies in lung cancer and head and neck cancer are being conducted in collaboration with Merck & Co (MSD). Efti/Bavencio combo study in solid tumours in collaboration with Merck KGaA and Pfizer. The Phase IIb AIPAC study of efti + chemo in metastatic breast cancer failed to significantly improve progression free survival. WuXi AppTec China produces efti under terms of partnership with EOC, to US European and Chinese GMP standards.
	Solid tumours + Bavencio;	Phase I	
	Metastatic breast cancer + chemotherapy	Phase IIb/	
LAG525/ Novartis (worldwide)	Cancer and chronic infectious disease	Phase II	Antagonist anti-LAG-3 antibody, activates T-cell proliferation, immune checkpoint blocker. Five Phase I/II or Phase II trials are underway in solid tumours including melanoma, breast, lung and neuroendocrine cancers as well as lymphoma.
GSK'781/ GSK (worldwide)	Autoimmune disease/ulcerative colitis	Phase II	Depleting anti-LAG-3 antibody, depletes activated T-cells. Phase I trial in patients with plaque psoriasis completed. Phase II in ulcerative colitis underway, topline data due 2022. Potential milestone payments of up to GBP64m + royalties.
IMP761	Autoimmune disease	Preclinical	First in class LAG-3 agonist antibody. Aims to help treat autoimmune disease by temporarily switching off activated LAG-3 ⁺ T cells.

Source: Immutep, Taylor Collison research.

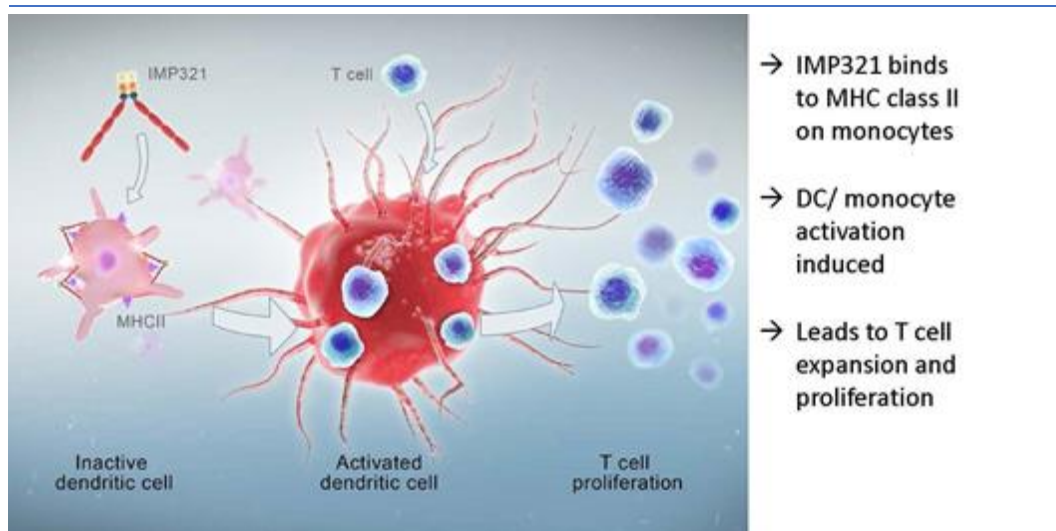
Exhibit 2: IMM products use LAG-3 technology to either stimulate or suppress immune responses



Source: Immutep.

Immutep's lead product, efti, is a LAG-3 Ig fusion protein that is based on the soluble form of LAG-3 and can activate antigen presenting cells (APCs) to stimulate the initial steps of the immune response. These activated APCs process tumour antigens, transport the antigens to lymph nodes and present the tumour antigens to T lymphocytes, thus activating and amplifying the immune response. Levels of CD4 and CD8 T cells and natural killer cells are all elevated in response to efti treatment.

Exhibit 3: Efti LAG-3 fusion protein activates APCs



Source: Immutep. Note: IMP321= efti

Immutep is currently collaborating with Merck & Co (known as MSD outside the US and Canada) to investigate efti in combination with Keytruda in lung cancer and head and neck cancer. Immutep recently reported the results of a Phase II study of efti combined with chemotherapy in breast cancer. It is also collaborating with Germany-based Merck KGaA and Pfizer in an earlier stage study investigating efti in combination with Bavencio (avelumab).

ICI drugs such as Keytruda and Opdivo have transformed the treatment of a number of cancers in recent years. Keytruda and Opdivo generated sales of US\$11.1bn and US\$7.2bn respectively in 2019. According to the market research group EvaluatePharma, they are expected to achieve sales of US\$17bn and US\$10.5bn respectively in 2024, so a successful ICI combination therapy would be expected to generate substantial sales.

Scientist who discovered LAG3 part of the Immutep team

The scientist who discovered the LAG-3 gene in 1990, Professor Frédéric Triebel, is Immutep's Chief Scientific Officer and Chief Medical Officer. Professor Triebel's deep understanding of LAG-3 and its potential applications in medicine has given Immutep a position of scientific leadership in the LAG-3 space. While a number of companies are developing anti-LAG-3 antibodies as checkpoint blockade therapies to enhance T-cell activity in cancer (including Immutep's partner Novartis) no other company can match the breadth of Immutep's LAG-3 pipeline, with two different therapeutic approaches to enhance immune responses in cancer (efti and LAG525), plus another two approaches to suppressing undesirable immune responses in autoimmune disease.

In addition to this scientific expertise, Immutep has an experienced management team with runs on the board. Chairman Dr Russel Howard is an experienced scientist, entrepreneur and manager; he was a cofounder and CEO of Maxygen when it was spun out of GSK and listed on NASDAQ. After leaving Maxygen in 2008 he started the cleantech company NovoNutrients Inc. Deputy Chairman Pete Myers is the CFO of Eagle Pharmaceuticals (NASDAQ: EGRX) and was previously CFO of NASDAQ-listed Motif Biosciences and TetraLogic Pharmaceuticals. CEO Marc Voight joined Immutep in 2011 and was appointed CFO in 2012 and CEO in 2014; before joining Immutep Marc worked for a number of years as an investment manager for a mid-sized VC fund base in Berlin, specialising in healthcare, and held management roles with Revotar Biopharmaceuticals, Caprotec Bioanalytics and Medical Enzymes AG.

AIPAC – evidence of activity but progression free survival benefit falls short

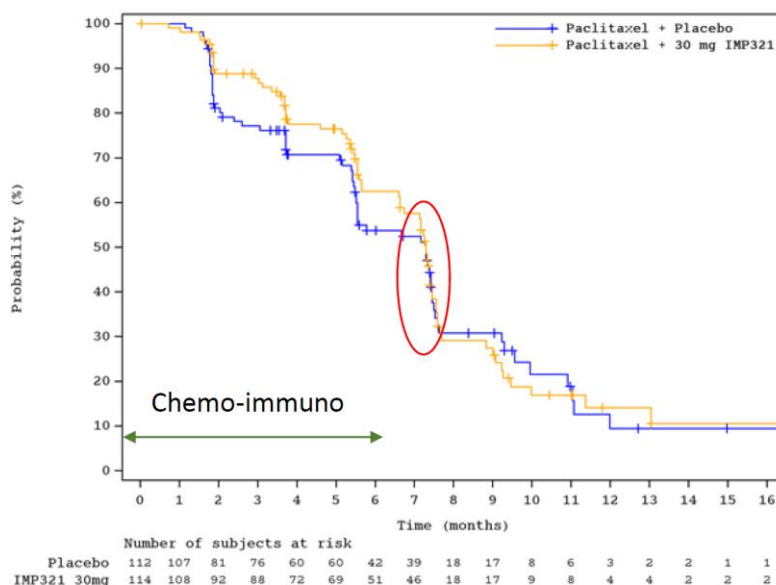
Last month Immutep announced results showing that its AIPAC study failed to deliver a statistically significant improvement in progression free survival (PFS) in breast cancer patients. The Phase IIb AIPAC trial compared efti plus paclitaxel chemotherapy to chemotherapy alone in 227 patients with metastatic breast cancer who were undergoing first-line chemotherapy. The study recruited women with hormone receptor positive, HER2 negative cancer, who comprise the largest subset of breast cancer patients.

There was a modest 7% reduction in the risk of disease progression in patients receiving the efti combo, but the effect was not statistically significant (hazard ratio=0.93, p=0.34). As the chart below shows, there was a separation of the progression free survival curves in favour of efti during the six month period when the patients received efti plus chemotherapy, but the curves come together after the chemotherapy treatment stops. The median progression free survival was the same in both treatment arms at 7.3 months.

The AIPAC study built on an earlier study published in 2010 which reported a response rate¹ (at least 30% tumour shrinkage) of 50% among 30 women with hormone receptor positive, HER2 negative breast cancer. That paper compared the response rate to response rate to paclitaxel monotherapy of 25%. A more recent study reported a response rate of 41% following paclitaxel monotherapy in a similar, but not identical patient population (80% hormone receptor positive cancer)².

The response rate in efti plus paclitaxel treatment arm of AIPAC in the was 48%, similar to the previous study, but the response rate of 38% in patients treated with chemo alone was at the upper end of the range reported in previous studies. No p-values were reported, but we suspect that the difference was not statistically significant.

Exhibit 4: Progression free survival curves for efti/chemo combo vs chemo alone in AIPAC



Source: Immutep. Note: efti is labelled as IMP321; data shown is for the blinded, centralised assessment of tumour progression by an independent investigator.

Immutep identified two patient subgroups where there was a large, but not statistically significant, improvement in progression free survival. In patients with a low innate immune response at baseline, expressed as a low monocyte count, the efti- treated group had a 39% reduction in disease progression (HR 0.61, p=0.08). The apparent benefit in this patient group makes sense, given efti’s mechanism of action in stimulating the innate immune system, but the low monocyte count group only represented around 21% subjects in the trial. Women with the more aggressive luminal B tumoural subtype also showed lower disease progression (HR=0.65, p=0.06). These two subgroups could both be interesting areas of future research.

Immutep’s Chinese partner for efti, EOC Pharma, intends to continue with its ongoing Phase I study of the efti/chemo combo in breast cancer patients, despite the disappointing AIPAC results. There could potentially be scope for EOC Pharma to investigate one or both of the breast cancer subgroups on future studies of the combo.

¹ Immutep reports objective tumour responses in AIPAC and TACTI-002 as defined by **irRECIST**, a set of published criteria designed to ensure consistent reporting of responses to treatment in clinical trials of anti-cancer drugs. Under irRECIST, up to 5 tumours are identified as target lesions and the longest diameter of each target lesion is recorded (the shortest diameter is recorded for metastatic lymph nodes selected as target lesions). The measure of overall tumour burden at baseline is defined as the sum of these target tumour diameters. Progressive disease (PD) is defined as a 20% increase in the sum of tumour diameters, or the appearance of one or more new lesions. A partial response (PR) is a 30% decrease in the sum of diameters, with no new lesions allowed. A complete response (CR) is the complete disappearance of all lesions. Stable disease is neither sufficient shrinkage to qualify as PR nor sufficient increase to qualify for PD, and no new lesions. If the response rate is the main efficacy endpoint of the study, then the responses should be confirmed at a second scan at least 4 weeks after the first.

² Martin et al, Lancet Oncol 2011; 12: 369–76

Positive implications from AIPAC for ongoing ICI combo studies

We were looking AIPAC to answer two key questions:

- Firstly, is eftri plus chemo an effective therapy in breast cancer?
- Secondly, is there evidence that eftri is an active anti-cancer agent that could provide meaningful benefit to patients when combined with immune checkpoint blockade?

The answer to the first question currently appears to be no, although the history of Provenge³ in prostate cancer means that it is still possible that there could be an overall survival benefit. Overall survival data is expected to be reported later in 2020.

However, we find the AIPAC data much more encouraging as a pointer to the potential benefit of eftri/ICI combo therapy. The numerically higher response rate (48% vs 38%) and the separation of the progression free survival curves indicates to us that eftri has immune stimulating activity, in line with the proposed method of action, although the activity was not sufficient to be a useful therapy in this treatment setting in breast cancer.

We have learnt over the past decade that many tumours are able to grow because of the immunosuppressive tumour microenvironment created by tumours expressing immune checkpoint inhibitor molecules such as PD-L1 and PD-L2. One possible explanation for the modest efficacy observed in AIPAC is that an immunosuppressive tumour microenvironment may have limited the benefit that could be obtained by eftri activating APCs to enhance the initial immune response.

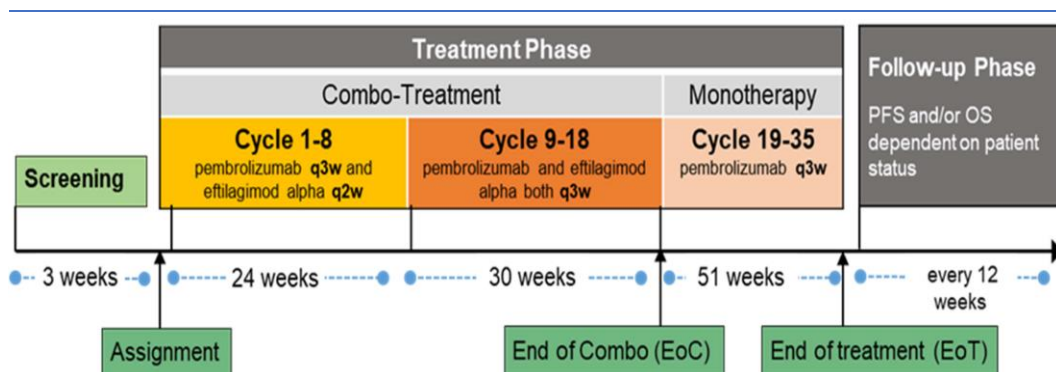
In our view, this points to the logical step of combining eftri with simultaneous blockade of immune checkpoint pathways by anti-PD1/L1 antibodies as Keytruda (pembrolizumab) or similar drugs to overcome the immunosuppressive tumour microenvironment. The company has already reported very encouraging response rates when eftri was combined with Keytruda in three different diseases, as described below. Based on the AIPAC results we believe that an eftri/Keytruda/chemo triple combo could be an effective therapy in metastatic breast cancer.

TACTI-002 reports high response rates from checkpoint inhibitor combo studies

Immutep has reported very encouraging results over the past six months from its TACTI-002 checkpoint inhibitor combo study. TACTI-002 is investigating the safety and efficacy of eftri plus Merck's anti-PD1 ICI drug Keytruda (pembrolizumab) in head and neck cancer and in first and second line lung cancer settings. The open label, single arm study (no placebo control group), which is being conducted in collaboration with Merck & Co, is enrolling up to 109 patients at up to 13 sites in Europe, the US and Australia.

Patients will receive 12 months of eftri/Keytruda combination therapy, followed by a further 12 months of Keytruda monotherapy (Exhibit 5). Treatment with eftri (30mg by subcutaneous (SC) injection) starts on the same day as Keytruda; eftri is administered every two weeks for the first 24 weeks (8 cycles of Keytruda) after which eftri is administered every three weeks, to align with the Keytruda treatment schedule. The primary efficacy assessment is the overall response rate (ORR; as per irRECIST).

Exhibit 5: TACTI-002 trial design



Source: Immutep. Note: One cycle: three weeks; q2w: every two weeks; q3w: every three weeks; pembrolizumab= Keytruda

³ Provenge, which like eftri targeted dendritic antigen presenting cells, produced a statistically significant improvement in overall survival despite only a non-significant 6% improvement in progression free survival (hazard ratio 0.94).

The three patient populations targeted in TACTI-002 are:

- Part A: first-line advanced/metastatic non-small cell lung cancer (NSCLC) patients, who are PD-1/L-1 naive and have not undergone systemic therapy for advanced/metastatic disease.
- Part B: second-line advanced/metastatic NSCLC patients who have experienced confirmed treatment failure (disease progression) following treatment with any PD-1/PD-L1 regimen.
- Part C: second-line squamous cell carcinoma of the head and neck (HNSCC) patients who are PD-1/L1 naive.

The TACTI-002 study is using Simon's two-stage design, in which the study only recruits the full number of subjects if the initial cohort shows promising results. For each of the three treatment indications, an initial cohort of 17–23 patients is being treated. For each indication, if the number of patients with tumour responses exceeds a pre-specified threshold, additional patients will be recruited to take the total to ~37 for that indication, as shown in Exhibit 6. Recruitment has already progressed to the expansion cohorts for parts A and C in first line lung cancer and head and neck cancer, respectively.

Exhibit 6: TACTI-002 trial design and summary interim data

Indication	Recruitment in initial cohort	Minimum number of responses required	Number of responders reported in initial cohort	Response rate in initial cohort	Recruitment in expansion cohort	Total patients	Keytruda monotherapy ORR
Part A: NSCLC 1st line	17/17	5	8	47%	17/19	36.0	25%*
Part B: NSCLC PD1/L1 refractory 2nd line	18/23	2	-	-	-/13	36.0	N/A
Part C: HNSCC PD1/L1 naïve 2nd line	18/18	3	6	33%	6/19	37.0	16-18%**

Source: Immutep, Taylor Collison research. Note: #to aid clarity we have expressed the threshold as the minimum number of responses to be achieved; *Keynote-001 study; **Keynote-012 study

The best overall response rates for efiti/Keytruda combo reported so far have been 47% (8/17) in first line lung cancer (vs ~25% ORR for Keytruda monotherapy) and 33% (6/18) in head and neck cancer (vs 16-18% ORR).

In the earlier Phase I TACTI-mel study of efiti plus Keytruda therapy in melanoma, the response rate was ~50% (14 out of 27 or 28), which compares to the response rate of ~33% to Keytruda monotherapy in melanoma trials. In these three disease settings the response rates to date have been at least 50% higher (melanoma) and up to 100% higher (head and neck cancer) than the response rates reported following Keytruda monotherapy in similar patient populations.

Efiti is well tolerated in combination with Keytruda, with no dose-limiting toxicities reported.

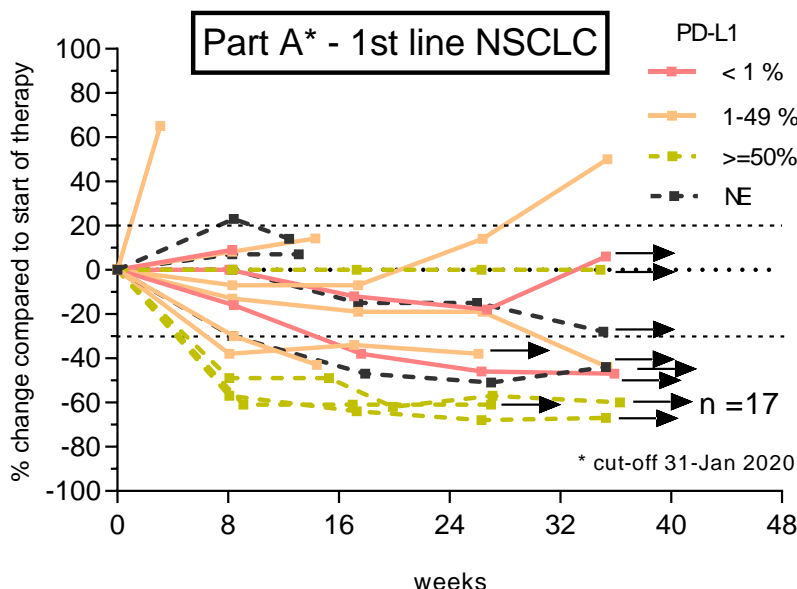
Lung cancer patients responded to efiti/Keytruda combo at all PD-L1 expression levels

The interim data from the initial cohort in Part A of the study showed that 8 out of 17 (47%) first-line NSCLC patients had achieved a partial response as of the data cut-off on 31 January. Ten patients were still receiving the combination therapy at least 7 months after commencing treatment. Exhibit 7, below, shows that one of the subjects with stable disease is experiencing ongoing tumour shrinkage which is approaching the 30% threshold that would qualify as a partial response; if this subject goes on to experience a partial response then that would push the response rate over 50%. The exhibit also shows that the tumour responses are long-lasting, with 7 of the 8 responders remaining on therapy with ongoing responses.

The expansion cohort in Part A of the study has recruited 17 out of the target of 19 additional subjects

Part A recruited all comers, regardless of the level of expression of the PD-L1 biomarker in their tumour.

Exhibit 7: Spider plot showing individual patient tumour responses in first line lung cancer (NSCLC) in TACTI-002



Source: Immutep. Note: the upper dashed line marks the 20% increase in tumour size that is the threshold for progressive disease and the lower dashed line marks the 30% decrease in tumour size that is the threshold for a partial response; NE = PD-L1 expression not evaluable in tumour tissue sample.

At the time of the data cut-off, Immutep was able to assess tumour samples from 13 of the 17 first-line NSCLC patients for expression of the PD1 biomarker, which is a predictor of response rates to Keytruda monotherapy, as shown in Exhibits 7 and 8. Exhibit 8 also shows the overall response rate reported for first line NSCLC patients with low, medium and high PD-L1 expression following Keytruda monotherapy treatment in the Keynote-001 study.⁴ We note that the Keynote-001 overall response rate (ORR) data only includes patients where the tumour shrinkage was confirmed in a second scan, whereas the best overall response rate (BORR) reported for the ongoing TACTI-002 study includes patients with tumour responses that have not yet been confirmed at a second scan, and so the data are not directly comparable. Despite this caveat, it is clear that the response rate following efiti/Keytruda combo therapy (BORR 47%) is much higher than the 25% ORR reported for a similar patient population treated with Keytruda monotherapy in the Keynote-001 trial.

Exhibit 8: TACTI-002 Part A first line lung cancer responses by PD-L1 expression category

PD-L1 expression category	Number of patients (% of evaluable) in Part A	Historical distribution#	No. of responders in Part A*	Best overall response rate to efiti/Keytruda combo	Historical Keytruda monotherapy ORR in first line treatment**
Low (<1%)	3 (23%)	32%	1	33%	14%
Medium (1-49%)	6 (46%)	44%	3	50%	19%
High (≥50%)	4 (31%)	25%	3	75%	47%
Not evaluable	4		1	25%	N/A
Overall	17		8	47%	25%

Source: Immutep, Taylor Collison research. Note: # Garon et al 2015 (Keynote-001) Supplementary Table S9; *Includes unconfirmed responses; ** Garon et al 2015, Supplementary Table S8 - ORR (overall response rate) includes only confirmed responses.

NSCLC represents a substantial market opportunity. Lung cancer is the leading cause of cancer death in the US, making up 22% of all cancer deaths, and is expected to account for ~229,000 new cases and 136,000 deaths in 2020. NSCLC makes up approximately 85% of lung cancers.

⁴ Garon et al, 2015. Pembrolizumab for the Treatment of Non-Small-Cell Lung Cancer. N Engl J Med; 372:2018-2028

While ICI drugs such as Keytruda and Opdivo have transformed the treatment of lung cancer recent years there is an urgent need to improve response rates and patient outcomes. For example, Keytruda as a single agent is approved for treating newly diagnosed (first line) NSCLC with expression levels of the PD-L1 tumour biomarker of at least 1%, but the response rate in this patient population in the pivotal Phase III study was only 27%. Keytruda is also approved for use in combination with chemotherapy in first line NSCLC patients regardless of PD-L1 expression therapies; the response rates in this setting ranged from 45% to 58% in different NSCLC subtypes.

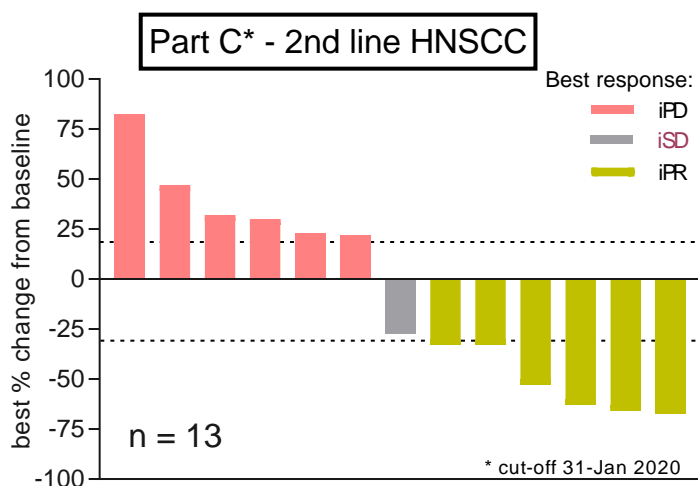
We see two Key opportunities for efti combo therapies in NSCLC. The first opportunity is for an ICI/efti combo that generates higher response rates than ICI single agent but without the unpleasant side effects of chemotherapy. The second key opportunity in our view is as an ICI/chemo/efti triple combo where it has the potential to generate a meaningful increase in response rates without adding to the side effect burden for patients.

Head and neck cancer response rate already 33% with more patients to come

Part C of the TACTI-002 study, in second-line HNSCC patients who are PD-1/L1 naive, has already surpassed the threshold response rate in the initial cohort, with 6 of the 18 subjects (33%) achieving a partial response as of the data cut-off point on 31 January, as shown in Exhibit 9. The 6 responders reported to date is well above then minimum of 3 responders required for cohort expansion. Importantly, at that point 3 of the subjects had been on treatment for less than 9 weeks and had not yet undergone an initial response assessment, so there is scope for the response rate to go higher when the additional 3 patients are scanned.

In Merck's Keynote-012 study of Keytruda monotherapy in second-line recurrent or metastatic HNSCC the confirmed ORR was reported as 16% (n=174) in the Keytruda prescribing [information](#), and in a subsequent publication as 18% among 192 patients at long-term follow-up (Mehra et al 2018).⁵ Even bearing in mind the caveat that some of the responses reported by Immutep from part C have not yet been confirmed at a repeat scan, the 33% response rate reported for HNSCC following efti/Keytruda combo therapy is approximately double the response rate reported for a similar patient population following treatment with Keytruda alone.

Exhibit 9: Waterfall plot patient tumour responses in second line head and neck cancer in TACTI-002



Source: Immutep.

Earlier TACTI-mel combo study reported similar increases in response rates in melanoma

Immutep had previously investigated efti/Keytruda combination therapy in melanoma patients in the TACTI-mel study. As this was the first time efti had been administered in combination with a checkpoint inhibitor in humans, in Part A of the study efti therapy commenced 12 weeks after administration of Keytruda began. This allowed patients to be monitored for adverse events that coincided with efti therapy. Subjects were injected with 1, 6 and 30 mg of efti every 2 weeks, starting with the fifth cycle of Keytruda. Subjects were assessed after 3 cycles of Keytruda monotherapy and only subjects who had a suboptimal response to initial treatment with Keytruda were enrolled into the combination therapy study.

⁵ Mehra et al, 2018. British Journal of Cancer 119:153–159

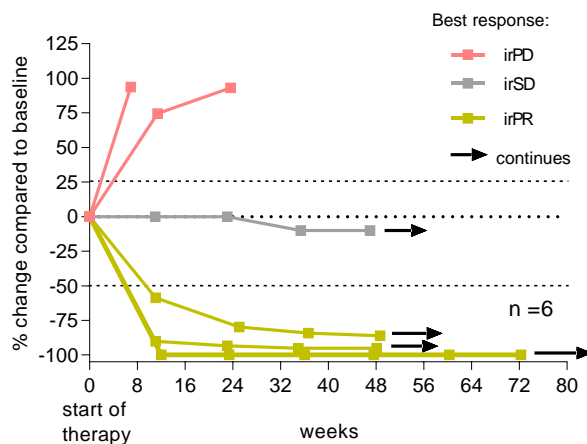
The response rate measured from the start of efTi/Keytruda combo therapy (at the start of the fifth cycle of Keytruda therapy) was 33% (6 out of 18 subjects). An exploratory analysis showed that the response rate measured from the first day of Keytruda therapy was 61% (11 out of 18). However, the design of TACTI-mel Part A meant that the results could not be directly compared to previous Keytruda monotherapy studies in melanoma patients.

In Part B of the TACTI-mel study subjects were administered with 30 mg of efTi by subcutaneous injection every 2 weeks, starting with the first cycle of Keytruda. The confirmed overall response rate was 50% (3/6), which confirms favourably to the 33% response rate in pivotal studies of Keytruda monotherapy in melanoma patients.

Management has indicated that approximately 22 subjects commenced Keytruda monotherapy during TACTI-mel Part A. Based on this, we estimate that the response rate among all participants in TACT-mel Part A was also approximately 50% (11 out of approximately 22 participants).

EfTi had a favourable safety profile, with no dose limiting toxicities and no new safety signals observed.

Exhibit 10: Spider plot of tumour responses in TACTI-mel Part B



Source: Immutep. Note: Responses are evaluated according to irRC criteria, rather than irRECIST; irPD= progressive disease; irSD= stable disease; irPR= partial response

Multiple studies indicate that efTi combination therapy delivers higher response rates

While indirect comparisons between clinical trials should always be treated with caution, response rates reported for immunology studies tend to fall within a much narrower range than the response rates from studies of chemotherapy agents. This gives us more confidence that the high response rates following efTi/Keytruda combo therapy in melanoma, lung and head and neck cancers are likely to be due to activation of the immune response by efTi and are unlikely to be chance observations.

Furthermore, we believe the fact that the response rate following efTi/chemo combo therapy in the AIPAC breast cancer study was 10% higher than the response rate to chemo alone (48% vs 38%), also points to efTi being an active anticancer agent. Even though the difference in response rates was probably not statistically significant, it represents evidence of activity from a randomised controlled study, rather than relying on cross trial comparisons.

Taking all of this evidence together, we believe that combining efTi with an ICI such as Keytruda is likely to lead to a real and reproducible increase in tumour response rates. Given the responses seen so far in TACTI-002 have been long lasting, we expect the increased response rates to translate into improved patient survival. If subsequent studies confirm this belief, the convenient subcutaneous administration and favourable side effect profile could potentially see efTi combo therapy used in a wide range of different cancer types, opening up a very large addressable market.

By year-end the efTi data set could be comparable to Vialytics at the time of the A\$500m Merck deal

Immutep currently has response data on almost 60 patients who have been treated with efTi plus Keytruda in TACTI-mel and the first two cohorts of TACTI-002. A further 27 subjects have already been recruited in TACTI-002 cohorts for which no response data has been reported yet. TACTI-002 is currently planned to recruit at least 96 subjects, and a total of 109 subjects if TACTI-002 Part B in PD1 refractory lung cancer proceeds to recruit the expansion cohort.

The ongoing investigator-initiated Insight study could also report additional data on efTi in combination with Merck KGaA and Pfizer's checkpoint inhibitor Bavencio (avelumab), which would add to the data set.

If recruitment in the studies is able to continue despite the Covid-19 pandemic, then by the end of the year Immutep could have data on over 100 patients treated with efti/Keytruda combo therapy.

This would give Immutep a similar amount of data on patients treated with checkpoint inhibitor combination therapy to the data set that Viralytics had when it was acquired by Merck for ~A\$500m in early 2018. The acquisition price was at a 160% premium to the one-month VWAP of Viralytics shares.

Furthermore, we note that the response rates when efti is added to Keytruda is comparable to the efficacy of Cavatak plus Keytruda. While the 59% (16/27) response rate following intralesional injection of Cavatak was higher than the ~50% ORR for efti plus Keytruda in melanoma, the response rate following iv Cavatak plus Keytruda in second and third line NSCLC (31%, 5/16) was considerably lower than the 47% response rate (8/17) reported for efti plus Keytruda in first line NSCLC patients. In metastatic bladder cancer the response rate following iv Cavatak plus Keytruda was not much higher than the response rate in pivotal studies of Keytruda monotherapy (28% vs a 21%).

It is important to note that the convenient subcutaneous injections are used to administer efti for all of the different types of cancer, whereas the highest response rates to Cavatak required direct injection into the tumour, a delivery method that is quite cumbersome for most types of cancer. Response rates to Cavatak were lower when the intravenous delivery route was used.

Overall, the efficacy of efti plus Keytruda appears to be comparable to Cavatak plus Keytruda, although the efti combo may be more efficacious than intravenous Cavatak.

We believe efti could attract a similar deal to Viralytics, but Epcadostat failure creates uncertainty

We believe that there are two key unanswered questions for investors about efti/checkpoint inhibitor combination therapy.

1. Will efti combo continue to deliver high response rates in the ongoing study?
2. Would Immutep be able to do a Viralytics style deal on the back of continued high response rates in TACTI-002.

The first point reflects the ongoing clinical trial risk that applies to all drug development programs, and cannot be avoided, but the results so far are very encouraging, in our view.

On the second point, we believe that the failure of a once-promising immuno-oncology combination therapy a few months after the Viralytics deal has dented investor confidence that high response rates in single arm studies will translate into efficacy in a randomised controlled study. In April 2018 the Keynote-252 Phase III study showed that the addition of the IDO1 inhibitor epacadostat (Incyte; NASDAQ: INCY) to Keytruda therapy failed to stop cancer progression in first-line melanoma patients, compared with Keytruda alone. The overall response rate for patients treated with epacadostat plus Keytruda was almost the same as that for Keytruda alone (34% vs 32%). The low response rate in the Phase III study was marked contrast to a previous Phase I/II study where 56% of the 54 melanoma patients treated with the epacadostat/Keytruda combo achieved an objective response. A single arm study of epacadostat combined with a different checkpoint inhibitor, Opdivo, in melanoma patients also reported a high response rate (65%).

The lack of efficacy of epacadostat when added to Keytruda was confirmed in two subsequent randomised studies in lung cancer, which showed that adding epacadostat resulted in lower response rates compared to Keytruda alone. In the Phase II Keynote-654-05 [study](#), in lung cancer patients with high expression of the PD-L1 biomarker, the overall response rate in patients receiving the epacadostat/Keytruda combo was 33% vs 39% in patients given Keytruda plus placebo. Similarly, in the Keynote-715-06 study the response rate in the Keytruda/chemo/epacadostat arm was 26% vs 45% for Keytruda/chemo/placebo treated patients.

We don't believe that the failure of epacadostat has a negative readthrough for efti. Firstly, IDO1 inhibitors like epacadostat act in a different part of the immune system to efti. IDO1 inhibitors act on T cells which kill cancer cells, as do the anti-PD-1/L1 checkpoint inhibitors. In contrast, efti activates antigen presenting cells which are key actors in recognising tumour antigens and triggers the initial steps of the immune response.

Secondly, given that there was a 10% difference in response rates in favour of the efti/chemo combo in the randomised AIPAC study, it seems unlikely that efti would suffer the same fate as epacadostat and fail to show any benefit when added to checkpoint inhibitor therapy in a randomised study. However, what future trials will need to determine is whether the benefit would be large enough to make the combo an effective therapy with broad uptake in the clinic.

Another factor that may influence the prospects for attracting a pharma partner is that a large number of other therapies are being investigated for efficacy in combination with checkpoint inhibitors. Other immuno-oncology agents which aim to enhance anti-tumour immune responses include compounds targeting the STING (STimulator of INTERferon Genes), TLR9 (Toll-like Receptor 9), IL-12 (interleukin 12), TIGIT (T-cell immunoglobulin and ITIM domains) and PIN-2 pathways, as well as anti-LAG-3 checkpoint inhibitor antibodies. In addition to these combinations of two immuno-oncology agents, checkpoint inhibitors are also being combined with chemotherapy and targeted therapies such as BRAF and MEK inhibitors. EvaluatePharma reported that there were 765 ICI combo studies underway in 2017, and the number is likely to have grown since then.

Despite this competition, we believe that the high response rates reported for the efiti/Keytruda combo in a range of cancer types, its favourable side effect profile and the convenient subcutaneous route of administration mean that it is well placed to attract interest from big pharma. We believe that there is a realistic prospect that IMM could achieve a significant licence deal or M&A transaction in the next 12-18 months if efiti continues to generate high response rates in the TACT-002 study. We do not believe that the potential for a significant transaction in the next 12-18 months is reflected in the current share price, and therefore that IMM represents an attractive opportunity for investors.

Programs partnered with big pharma have progressed to mid-stage trials

On top of the in-house efiti program, Immutep has two additional LAG-3 programmes that are partnered with big pharma and could create significant value, both of which have already progressed to Phase II (mid-stage) trials. These programmes are partnered with Novartis (anti-LAG-3 antibody in cancer) and GSK (LAG-3 depleting antibody in inflammatory disorders).

Novartis: Five LAG525 combo studies underway

LAG525 is an antibody which blocks the LAG-3-mediated inhibitory signal given to tumour-infiltrating T-cells and thus activates T-cell proliferation and enhances the anti-tumour activity of anti-PD-1 ICI drugs. LAG525 is Novartis's humanised version of IMP701, which it in-licensed from Immutep. The total milestones and royalties payable to Immutep under the global license agreement with Novartis have not been disclosed.

Novartis has five Phase I/II or Phase II studies of LAG525 underway. In each of these studies, LAG525 is combined with Novartis's in-development anti-PD-1 antibody PDR001 (also known as spartalizumab).

The original Phase I/II study of LAG525 as a single agent and in combination with PDR001 that commenced in June 2015, has enrolled 490 subjects and is expected to reach final completion in July this year (Clinical trials.gov: [NCT02460224](https://clinicaltrials.gov/ct2/show/study/NCT02460224); Novartis protocol LAG525X2101C). The Phase II component of the study comprised cohorts with NSCLC, melanoma, renal cancer, mesothelioma and triple-negative breast cancer (TNBC) who are being treated with LAG525 plus PDR001.

A second Phase II study of LAG525 plus PDR001 commenced in January 2018 ([NCT03365791](https://clinicaltrials.gov/ct2/show/study/NCT03365791); Novartis protocol CPDR001XUS01). This adaptive, open label Phase II study enrolled 76 patients with solid or haematological cancers including small cell lung cancer, gastric/oesophageal, prostate, ovarian cancers or diffuse large B-cell lymphoma. Study completion is expected in October 2020.

Novartis is conducting a randomised Phase II study in TNBC patients in which LAG525 is being combined with either PDR001 or carboplatin chemotherapy, or with both PDR001 and carboplatin ([NCT03499899](https://clinicaltrials.gov/ct2/show/study/NCT03499899)). The trial, which commenced in July 2018, recruited 88 patients. Collection of top-line ORR data was completed in February 2020, and final study completion is expected in January 2021.

LAG525 is one of four drugs being randomly assigned for use in combination with PDR001 in 230 patients with advanced melanoma in the PLATforM Phase II study ([NCT03484923](https://clinicaltrials.gov/ct2/show/study/NCT03484923)). The study, which commenced in September 2018, was originally intended to recruit 135 subjects, but was expanded to 230 subjects in April 2019. Final topline data is expected to be collected in June 2021.

Lastly, Novartis has also initiated an open label triple combo Phase Ib study in TNBC patients in which LAG525 plus PRD001 will be combined with either the drugs NIR178 or capmatinib or the biologicals MCS110 or canakinumab ([NCT03742349](https://clinicaltrials.gov/ct2/show/study/NCT03742349)). The trial, which commenced in January 2019, will recruit up to 220 patients. The expected primary completion date is January 2022.

Novartis Phase II supported by efficacy signals in combo Phase I/II and Phase II studies

Novartis reported⁶ encouraging signs of efficacy from the LAG525 in combination with PDR001 from the Phase I component of its ongoing Phase I/II study ([NCT02460224](https://clinicaltrials.gov/ct2/show/study/NCT02460224)) in 2018. Among 121 patients with solid tumours treated with LAG525 plus PDR001 at a wide range of doses there were 13 durable responses, including 3/8⁷ mesothelioma patients and 2/5 TNBC patients. In TNBC tumour biopsies, there was a trend to conversion from immune-cold to immune-activated biomarker profiles. No responses were observed in 134 patients treated with LAG525 alone.

Novartis presented preliminary data on the [NCT03365791](https://clinicaltrials.gov/ct2/show/study/NCT03365791) Phase II study at ASCO in 2019. Efficacy was assessed based on the clinical benefit rate (CBR) which comprises either a tumour response or stable disease. Three cancers, small cell lung cancer; neuroendocrine tumours (NETs) and diffuse large B-cell lymphoma (DLBCL), entered the cohort expansion phase having met the CBR target threshold. The CBR reported for the 3 cancers were 27%, 86% and 43%, respectively. Assessment in three cancers which had not met the expansion thresholds was ongoing (prostate and ovarian cancer and soft tissue sarcoma), while the gastroesophageal cancer cohort was stopped for futility.

⁶ Hong et al 2018. <http://novartis.medicalcongressposters.com/Default.aspx?doc=e07a1>

⁷ 3/8 includes an additional partial response after the data cut-off date for the conference abstract.

GSK is conducting a Phase II study of GSK'781 in ulcerative colitis

GSK'781 (GSK2831781), which is based on the IMP731 antibody that GSK in-licensed from Immutep in 2011, is an anti-LAG-3 depleting antibody that will kill the few LAG-3 positive activated T-cells that infiltrate autoimmune disease sites. The licence deal involved potential milestone payments of approximately GBP64m (~A\$125m) plus undisclosed royalties payable to Immutep.

According to the clinicaltrials.gov registry entry ([NCT03893565](https://clinicaltrials.gov/ct2/show/study/NCT03893565)), GSK started a Phase II study of GSK'781 in May 2019. The study will investigate the safety, tolerability, efficacy and dose response of GSK'781 in up to 280 subjects with moderate to severe ulcerative colitis. The estimated primary completion date is April 2022.

Ulcerative colitis is a type of inflammatory bowel disease. Ulcerative colitis and Crohn's disease are the most common types of inflammatory bowel diseases. The US Centers for Disease Control and Prevention [estimates](#) that the prevalence of ulcerative colitis is 0.24% of the population, which is equivalent to 780,000 patients in the US.

The key drugs for ulcerative colitis are Humira (adalimumab), Entyvio (vedolizumab), Remicade (infliximab) and Simponi (golimumab). Allied Market Research forecasts the global ulcerative colitis market to grow to US\$7.5bn by 2023.

GSK completed a Phase I study of GSK'781 in psoriasis patients in March 2018. We believe that the Phase I study was conducted in psoriasis patients because it is easy to assess responses to treatment in psoriasis skin lesions, and because it is convenient to collect tissue biopsy samples from the skin lesions in order to assess the impact of the treatment on inflammatory markers in the tissue. We suspect that ulcerative colitis as the lead indication for GSK'781 because it represents a larger commercial opportunity than psoriasis.

IMP761 LAG-3 a potential treatment for autoimmune diseases

Immutep's novel LAG-3 agonist IMP761 inhibited immune responses, including infiltration by inflammatory lymphocytes, in a non-human primate study. This proof-of-concept (PoC) study confirmed that IMP761 has potential as a treatment for inflammatory autoimmune disorders.

The company's manufacturing partner for IMP761 has developed a high-yielding cell line as it prepares for the manufacture of GMP-grade IMP761 product for use in clinical studies.

IMP761 is the first known therapeutic antibody with agonist properties that enable it to activate the LAG-3 receptor on the surface of activated T cells, and thereby down-regulate T cell activation and proliferation. In contrast, LAG525 and the other known anti-cancer LAG-3 antibodies are antagonist antibodies that block LAG-3 signalling and thereby prevent the down-regulation of T cell immune responses.

The mechanism of action of IMP761 is also different to the company's partnered GSK'781 cytotoxic mAb, which aims to treat autoimmune disease by killing LAG-3-positive T cells. IMP761 offers the opportunity to fine-tune immune responses, which could benefit sufferers of autoimmune diseases by temporarily switching off activated LAG-3-positive T cells that are damaging tissue or causing inflammation.

Valuation

We initiate coverage of Immutep with a valuation of \$327 or 83c per share (undiluted), based on a risk-adjusted discounted cash flow model. On a fully diluted basis, our valuation is 53c per share, after taking into account the options, warrants and convertible notes on issue, and a \$30m capital raise that we model in FY21. Exhibit 11 summarises the constituent parts of our valuation, which is based on a discount rate of 12.5%.

We forecast the company's gross cash balance at end FY20 to be \$13.0m. For valuation purposes we deduct the \$13.75m face value of the Ridgeback Capital convertible note in calculating end-FY20e net debt of (\$0.8m) as shown in Exhibit 11. We note that this is different to the accounting treatment of the convertible note, which includes only the A\$7.6m estimated fair value of the convertible note as a non-current liability, with the remainder treated as equity.

We model Immutep outlicensing efti at the completion of the TACTI-002 study in a deal that includes an upfront payment of US\$85m and US\$540m of milestone payments. We model a licencing deal being finalized in FY22, although we see a realistic prospect of a deal in FY21. We assume that 50% (US\$270m) of the milestone payments are for the achievement of clinical and regulatory milestones, with the remaining 50% assumed to be based on sales hurdles. We risk adjust the US\$85m upfront and US\$270m clinical and regulatory milestones with a 15-50% probability (50% probability of signing a license deal, 15% for approval milestones). We do not include any potential sales-based milestones in our forecasts, and instead model a 15% royalty rate. The modelled deal terms are based on relevant benchmarks over the last few years (sourced from EvaluatePharma and a [report](#) produced by the industry group BIO).

Exhibit 11 shows our key assumptions for each product included in the valuation and the rNPV for each product. We have offset the risk-adjusted trial cost of a potential Phase IIb study of efti against potential efti milestone revenue.

Exhibit 11: Immutep risk-adjusted DCF base case valuation and assumptions

Value driver	Base case success likelihood (%)	rNPV (\$m)	rNPV/share (\$)	Assumptions
efti+anti-PD1 ICI NSCLC	15%	96.5	\$0.25	Global peak sales of US\$2.1bn. We assume 121,270 NSCLC deaths in the US each year, 75% eligible for therapy, 15% penetration; pricing US\$60k per patient; global sales double US sales; launch 2027; 15% net royalty.
efti+anti-PD1 ICI head and neck	15%	46.4	\$0.12	Global peak sales of US\$1.0bn. We assume 65,630 new cases; 10% penetration; pricing US\$60k per patient; global sales double US sales; launch 2027; 15% net royalty
efti+anti-PD1 ICI melanoma	15%	11.4	\$0.03	Global peak sales of US\$120m. We assume 100,350 new cases; 16% advanced or metastatic (Stage III&IV); 10% penetration; pricing US\$60k per patient; global sales double US sales; launch 2027; 15% net royalty
efti+chemo breast cancer	2%	6.8	\$0.02	Global peak sales of US\$1.1bn. We assume 276,480 new cases; 65% HR positive, HER2 negative. 40% advanced or metastatic; 15% penetration; pricing US\$60k per patient; global sales double US sales; launch 2027; 15% net royalty. We assume a 2% success probability as OS data is still pending.
efti milestones – assume partnered post TACTI-002	15-50%	80.5	\$0.21	R&D cost: Assumes A\$30m for randomised Phase IIb study if licence deal is delayed beyond FY22
LAG525 solid tumours (lung cancer)	20%	54.7	\$0.14	NSCLC as indicative indication. Global peak sales of US\$2.1bn. We assume 121,270 NSCLC deaths in the US each year, 75% eligible for therapy, 15% penetration; pricing US\$60k per patient; global sales double US sales; launch 2027; 5% net royalty
GSK*781- autoimmune disease (ulcerative colitis)	20%	54.9	\$0.14	Global peak sales of US\$1.1bn – 15% share of US\$7.5bn global market; launch 2027; 8% net royalty. GBP55m of total GBP64m potential milestones from GSK (risk adjusted to GBP17m)
SG&A to 2024		-23.8	-\$0.06	
Portfolio total		327.4	\$0.84	
Cash end FY20e (incl. conv. note at face value)		-0.8	\$0.00	
Enterprise total		326.7	\$0.83	

Source: Taylor Collison research. Note: NPV adjusted for tax at an effective tax rate of 20%. We assume that the addressable markets grow at 2% per year.

Potential dilution

We note that Viralytics raised ~\$30m in a placement to fund a pivotal study of Cavatak December 2017, approximately two months before the Merck transaction was announced. We model IMM similarly raising \$30m in FY21 so that it would be in a position to proceed with a randomised Phase IIb study of efti plus a checkpoint inhibitor if it does not reach agreement with a pharma partner. Viralytics had ~\$22m cash at the time the placement was announced.

Exhibit 12 shows that in addition to the 391.6m Immutep shares in issue, there are a further 345.3m potential shares that could be issued in the assumed FY21 placement and on the exercise of options, warrants, performance rights and convertible notes, all of which would be in the money at our 83c per share undiluted valuation. Exhibit 12 shows that after taking into account these potential shares, our diluted valuation is 53c per share.

Exhibit 12: Potential further dilution and value per share

	Average exercise price (A\$)	m
Current number of shares		391.6
Ridgeback convertible note potential shares	0.200	68.8
Ridgeback warrants*	0.235	38.0
2017, 2018 placement warrants	0.385	36.3
Other warrants	0.472	0.2
Performance rights**	0.000	14.5
A\$30m placement @ 16c in FY21	0.160	187.5
Total potential new shares		345.3
Total potential diluted number of shares		736.9
Net cash from placement and options exercise (A\$m)		\$51.2
Debt expunged on Conv note conversion (A\$m)		\$13.8
Valuation (above plus additional cash)		\$391.6
Diluted value per share		\$0.53

Source: Taylor Collison research. Note: *3.63m ADS warrants converted to ordinary shares at the long-term exchange rate.

**Both vested and unvested performance rights have been included.

We include risk-adjusted milestones payable by current partners GSK for GSK'781 and Novartis for LAG525, plus milestones from prospective deals for efti. The breadth of the LAG-3 pipeline means there could be further upside if Immutep or its partners launch additional products into the clinic or broaden the indications being studied.

Risks

Immutep is subject to clinical trial, regulatory and commercialisation risks common to all biotech companies. The key sensitivity is clinical progress of its pipeline of LAG-3 candidates, primarily the internally funded efti. While Immutep has funds to complete the Phase I/II TACTI-002 study of efti in combination with Keytruda, it would require a partnership or alternative forms of funding to advance efti/checkpoint inhibitor comb therapy into randomised studies (we model a \$30m capital raise to fund this). Existing partnerships with big pharma reduce the financial and execution risk for LAG525 and GSK'781; in addition, if the proof of concept study of GSK'781 in ulcerative colitis reveals evidence of efficacy, it could lead GSK to extend the study to additional indications including rheumatoid arthritis, psoriasis and multiple sclerosis, which could increase the potential peak sales and therefore the value of the product. The Covid19 pandemic is likely to slow down recruitment in clinical trials, especially in countries such as the UK and Spain. TACTI-002 has already recruited 70% of the total patients and INSIGHT has recruited 91% of the total.

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