

Matt Cross

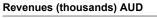
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(NASDAQ: IMMP)

Price	\$1.81
52 Week Range	(\$0.53 - \$3.10)
Price Target	\$5.00
Market Cap (mil)	\$100.70
Exchange rate	1US\$ = 1.39 AUD
Shares out (mil)	55.64
3-Mo Avg Vol	1,423,815
Cash per share	AUD0.37
Total Debt (mil)	AUD8.21
Observes and (mill) 40.4 Comments	Ohana ta ADO Datia

Shares out (mil): 10:1 Common Share to ADS Ratio



2019A	2020E	2021E
Actual	Curr	Curr
0A	7A	0E
0A	7E	0E
D		
2019A	2020E	2021E
Actual	Curr	Curr
(0.28)A	(0.16)A	(0.32)E
(0.57)A	(0.49)E	(0.71)E
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	Actual 0A 0A 2019A Actual (0.28)A	Actual Curr OA 7A OA 7E D 2019A 2020E Actual Curr Curr (0.28)A (0.16)A 0.16)A

Immutep Ltd.

Buy Volatility: 5

Eclipsing the LAG-3 Laggards in the Running for the Next Breakthrough in Immunotherapy

Immutep is an emerging biotech domiciled in Australia (with operations also in the U.S. and Europe) that has been at the forefront of development surrounding an intriguing immunotherapy target that we believe has the potential for extensive therapeutic utilization on par with PD-1 and CTLA-4. Lymphocyte Activation Gene-3 (LAG-3) was functionally characterized by Immutep's current CMO/CSO Frédéric Triebel in 1990, leading to the development of a recombinant LAG-3 Ig fusion protein (coined effilagimod alfa or IMP321) that entered clinical development in 2006. Since that time, at least fifteen prominent large pharma and biotech companies have acquired LAG-3 assets and initiated clinical testing of these product candidates. Bristol-Myers Squibb (BMY; not rated) has put particularly concerted efforts into developing its LAG-3 antagonist antibody relatlimab, since 2013, and subsequently clinical interest in LAG-3 has exploded. We believe Immutep is in an ideal position to capitalize upon this momentum given its expertise in LAG-3 functioning and therapeutic development, as well as its farreaching pipeline and partnerships with large pharma in this increasingly competitive space. Thus, we are initiating coverage of Immutep with a BUY rating and 12-month price target of \$5.00/ADS.

Immutep's early identification of LAG-3 promise and ties to efti's rapidly advancing peer group. We believe it is fair to say that there is a long road ahead for the clinical development of Immutep's LAG-3 assets, but we can also envision a very broad usage of this mechanism – akin to what has been seen for immunotherapies targeting PD-1 and CTLA-4 – that we feel justifies the wait if trial results live up to the theoretical hype. Immutep and its collaborators have begun evaluation of LAG-3 assets in a collection of trials across solid tumor indications including metastatic and triple-negative breast cancer, advanced melanoma, head and neck cancers, non-small cell lung cancer (NSCLC), and autoimmune diseases. In the context of oncology, the predominant approach to targeting LAG-3 has been via antagonistic antibodies, such as the LAG525 asset that Novartis has licensed from Immutep; however, Immutep's a different approach through APC activation. Efti binds to MHC Class II, including on the surface of APCs such as monocytes and dendritic cells. This increased APC activation and consequent dendritic cell maturation is only meaningful if relevant tumor antigens

are available for presentation to cytotoxic CD8 T cells, and thus Immutep opted to combine effi with chemotherapy in its latest-stage AIPAC trial in metastatic breast cancer patients. The effi clinical pipeline has since expanded considerably and is now comprised of four key combination trials with immunotherapy and chemotherapy agents (including anti-PD-1 given synergies reported from a host of pre-clinical studies and Bristol-Myers Squibb' ongoing work combining their anti-LAG-3 antibody with Opdivo), which are discussed separately in the following report.

Valuation:

Our valuation assumes that Immutep will successfully develop and commercialize effilagimod alfa in the U.S. and E.U. for metastatic breast cancer and advanced melanoma. We assume that the market opportunity will be modestly larger in metastatic breast cancer, with potential future revenues for melanoma staggered approximately one year behind those associated with metastatic breast cancer. We assume that pricing will remain consistent across all indications for effilagimod alfa. Based on our projected revenues and expenses for Immutep (subject to revision following the filing of formal FY2020 financial results), we expect the company to reach profitability by fiscal 2024, following our forecasted U.S. launches in metastatic breast cancer in fiscal 2023, and in advanced melanoma in fiscal 2024. At the end of fiscal 1H:20, the company held \$20.5M AUD of cash, equivalents, and securities with an annualized cash burn rate of \$12.8M AUD.

We derive our 12-month price target of \$5.00/ADS using a standard discounted cash flow (DCF) analysis valuation methodology in which we calculate cash flows out to fiscal 2028 with an assumed 2% terminal growth rate, discounted back at 39% over seven years. Our overall 39% blended discount rate is a weighted average of discount rates applied for each indication or revenue source we expect Immutep to pursue, with individual discount rates ranging from 37% to 40%. We assume a 2% terminal growth rate to account for other sources of revenue generation Immutep may develop that the company has not yet disclosed, as well as our expectation of continued cash flow growth beyond fiscal 2028. A sensitivity table is provided with our DCF breakdown, in the Financial Tables section of this report, for investors who wish to assume an alternative discount or terminal growth rate in their calculations.

Risks to achievement of target price:

Clinical/regulatory risk: Though Immutep has already presented encouraging initial data in metastatic breast cancer and advanced melanoma, this does not guarantee future clinical outcomes will prove positive. Should Immutep successfully complete all required clinical work sufficient to file for marketing approval of one or more product candidates the FDA, and regulatory agencies in any other pursued geographies, may choose not to approve Immutep's eftilagimod alfa or other product candidates, or may approve them with a label that is not ideal for the company's commercialization strategy. Additionally, any negative outcomes associated with ongoing or future clinical trials for candidates in Immutep's pipeline, including delays to expected clinical timelines or study protocol modifications resulting from the COVID-19 global pandemic, could have a materially detrimental effect on the company's stock price.

Commercial/competitive risk: Assuming that Immutep receives regulatory approval for eftilagimod alfa and/or other product candidates in one or more indications, the company may not be able to achieve the favorable pricing and market penetration needed to meet our revenue estimates. Though we believe eftilagimod alfa may have broad applicability in the treatment of oncology and autoimmune indications if clinical outcomes continue to prove favorable, Immutep still has significant clinical work ahead to confirm the potential benefits of its LAG-3 based therapeutics relative to existing treatment options. If ultimately approved, displacing existing treatment patterns may also prove more difficult than anticipated by current data and company estimates alone.

Financial risk: Immutep is well capitalized through fiscal 1H:21 by our estimates, but future capital demands may exceed our current expectations. The company may require additional sources of capital to fund the clinical development of eftilagimod alfa or other clinical pipeline projects depending on clinical and pre-clinical trial outcomes. Failure to secure needed financing to complete this work through the capital markets, partnerships, or grants may have significant consequences for company revenue estimates and the stock. Should the company choose to raise capital through future public offerings, investors may face dilution of their holdings.

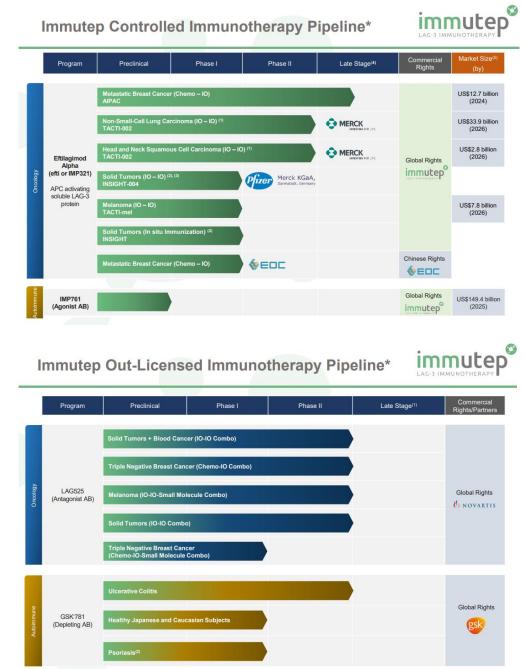
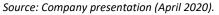


Exhibit 1: Immutep Clinical Pipeline



INVESTMENT THESIS

We are initiating coverage of Immutep Limited with a BUY rating and a 12-month price target of \$5.00/ADS based on the following key factors that make up our investment thesis:

- Immutep offers an opportunity to buy into a potential next-gen immune checkpoint treatment modality at a much more attractive valuation than peers. Many hypotheses about the functioning of Lymphocyte Activation Gene-3 (LAG-3) in human oncology and autoimmune settings remain to be tested (with only early clinical trial results available among the collection of product candidates in clinical development targeting LAG-3), but data to-date, in our view, suggest the potential for LAG-3 to follow in the footsteps of PD-1 and CTLA-4, as the next inhibitory checkpoint receptor with broad market applications. Large pharma players including Bristol-Myers Squibb (BMY; not rated), Merck (MRK; not rated); Novartis (NVS; not rated), GlaxoSmithKline (GSK; not rated), and several others have all bought into this hope with LAG-3 assets of their own, after Immutep introduced the first such candidate to the clinic in 2006. Immutep remains one of the leading LAG-3 innovators, with the company focused entirely on approaches to modulating this target. Furthermore, we believe the company offers a significantly more attractive opportunity to profit from future potential successes in the LAG-3 space, due to its approachable market valuation relative to that of the aforementioned peers. Our current cumulative and peak annual revenue assumptions for eftilagimod alpha through fiscal 2028 are as follows:
 - 1. Metastatic breast cancer- \$1.40B (peak of \$422M in fiscal 2028) USD
 - 2. Advanced melanoma- \$894M (peak of \$303M in fiscal 2028) USD
- Not one, but a handful of paths to added value from Immutep's existing pipeline. We believe Immutep has positioned itself well from a strategic standpoint, if hypotheses surrounding the potential applications of LAG-3 assets as immunotherapies for cancer and autoimmune conditions are confirmed. The company possesses a broad IP portfolio established during its role in characterizing LAG-3 functioning (including several composition of matter patents across its pipeline) which we believe will serve Immutep well as it seeks additional collaboration opportunities. Though subject to change, Immutep has also stated that it does not intend to seek commercialization of its LAG-3 assets alone if clinical results ultimately lead to one or more approvals, implying the potential for lucrative licensing agreements or M&A - particularly given the long list of competitors that may see value in Immutep's LAG-3 expertise and multiple unpartnered assets. Thus, whether it is through the success of its reputable development partners, an unlikely but possible commercial launch by Immutep itself, or some form of M&A arrangement, we believe Immutep has a broad array of potential routes to profitability and see now as an opportune time to buy in ahead of key developments expected over the next 12 months.
- * Immutep stock was hit hard following the somewhat disheartening topline readout of its Phase 2b AIPAC trial in March, but also provides a buying opportunity in wake of increased U.S. investor attention. Though sleepy over the last few years, IMMP stock has begun to count noteworthy U.S. institutional investors among its top holders and now shows signs of waking as data from several pipeline trials matures over the coming quarters. Following a surge of anticipatory buying in January and February, PFS data from the Phase 2b AIPAC trial of lead candidate eftilagimod alpha (soluble LAG-3) in HR+, HER2metastatic breast cancer missed the mark and resulted in a significant sell-off; however, we believe the reaction was overblown (full analysis in the body of this report), and did not reflect the asset's data and revenue potential in other indications, nor that of Immutep's much larger pipeline. Since that time, investors appear to have reached a similar conclusion, with shares once again trading near their early January levels. As such, we believe IMMP shares are poised for further recovery as forthcoming data, particularly from the ongoing Phase 2 TACTI-002 study of eftilagimod alpha (efti), serves up additional catalysts we expect will be positive given Immutep's overall clinical track record.

Pipeline Overview: LAG-3 is Gaining Prominence in the Immunotherapy Arena, but Immutep Made the Introduction

Inhibitory checkpoint receptors (PD-1 and CTLA-4) and the therapeutics that target them have undoubtedly become an intrinsic part of the oncology treatment landscape around which countless other modalities have positioned themselves (with checkpoint inhibition pioneers earning a Nobel Prize in the process). This is evidenced by the ever-growing collection of approved indications and treatment contexts for Merck's pembrolizumab (brand name Keytruda), and Bristol-Myers Squibb's nivolumab (brand name Opdivo) and ipilimumab (brand name Yervoy), as well as the ceaseless number of clinical trials combining these products with other mechanisms. However, the ubiquitous nature of combination regimens utilizing anti-PD1/PD-L1 and anti-CTLA-4 modalities also highlights a shortcoming of these approaches. It is estimated that only 20-30% of patients respond to these agents as monotherapies, and though this is not reflective of their real world utilization, it does suggest 1) The metaphorical immune system "brake" is likely not fully lifted by these agents; 2) Not all patients will exhibit expression of these receptors sufficient to achieve adequate response; and 3) A need exists for alternate mechanisms which build upon the breakthrough understandings of immune checkpoint biology to improve patient outcomes.

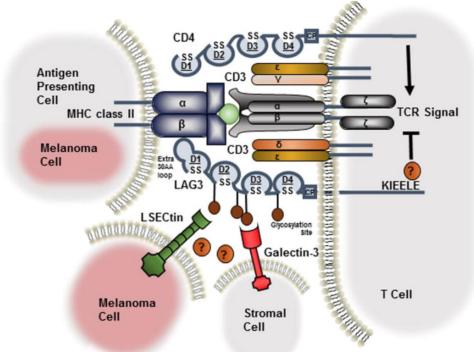


Exhibit 2: Ligand Interaction and Structural Similarities between LAG-3 and CD4

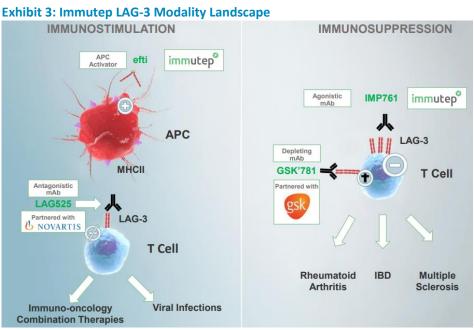
Source: Andrews, L. et al., "LAG3 (CD223) as a Cancer Immunotherapy Target." Immunol Rev. 2017 Mar; 276(1): 80–96.

Enter LAG-3, a unique inhibitory receptor that may further elucidate the immune response mediation equation. Without making undue connections between the discrete functioning of these targets, LAG-3 is, like CTLA-4 and PD-1, theorized to be an inhibitory receptor that modulates the activity of numerous immune cell types. The Lymphocyte Activation Gene-3 (LAG-3, a.k.a. CD223) receptor is expressed on activated CD4⁺ and CD8⁺ T cells, antigen-specific T cells acting in a regulatory capacity (Tregs), as well as NK cells, and appears to play a number of roles in mediating immune exhaustion and autoimmunity. Though understanding of LAG-3's functionality is still ongoing since its discovery by Immutep's current CMO/CSO Frédéric Triebel in 1990, it has been demonstrated that LAG-3 is a negative regulator of T cell activation and function which binds to MHC Class II. Furthermore, while limited amino acid homology remains between LAG-3 and CD4 today, the LAG-3 gene itself is located adjacent to CD4 on chromosome 12. Both bind MHC Class II, though LAG-3's binding affinity for MHC Class II is notably greater than that of CD4's. Despite the fact that LAG-3's MHC Class II binding affinity is roughly 100x greater than CD4's, LAG-3 does not compete with CD4 for binding, and it has been suggested that LAG-3 transmits inhibitory signals by way of its cytoplasmic domain. Interestingly, LAG-3 has been shown to downregulate $CD4^{+}$ and $CD8^{+}$ T cell function to similar degrees, but only the former is accomplished through established interactions with LAG-3's primary ligand MHC Class II. CD8⁺ T cell function is likely impacted by LAG-3's ability to activate antigenpresenting cells (APCs) such as monocytes and dendritic cells, increasing antigen presentation to CD8⁺ T cells. LAG-3 is also highly expressed on activated, tumor-infiltrating Tregs, but to a significantly lesser extent on resting Tregs in the periphery. The impact of LAG-3 on Tregs is less consistently characterized but may aid in T cell homeostasis, as LAG-3 imbalance has been implicated with worsening of disease in autoimmune conflict models.

Immutep's pipeline encompasses several aspects of LAG-3 biology, positioning the company and large pharma collaborators to benefit in many possible clinical scenarios.

The LAG-3 clinical development field has become increasingly active in just the last seven years, as additional insights into the potential of this target to be another key inhibitory checkpoint in the immunotherapy armamentarium have become available. As Immutep was the first company to characterize LAG-3 and enter clinical development (in 2006) with a recombinant, soluble LAG-3 Ig fusion protein (Eftilagimod alpha a.k.a. IMP321), it has well-canvassed the potential market surrounding LAG-3, and we believe stands to benefit most via one of many prospective avenues – should more theoretical benefits of targeting LAG-3 continue to play out in clinical trials. Immutep remains, in our view, the company most focused on LAG-3 clinical development, but it also comes as no surprise to us that some of the largest immunotherapy players in the market have tossed their hats into the ring as well (several in collaborations or licensing agreements with Immutep) to assess whether LAG-3 could follow in the footsteps of PD-1 and CTLA-4. As described in more detail below, Pfizer and Merck are collaborating with Immutep on trials of lead candidate eftilagimod alpha (efti) for which Immutep owns global rights, while Novartis and GlaxoSmithKline own global rights to LAG-3 antagonistic and depleting antibodies LAG525 and GSK'781, respectively, from which Immutep is entitled to milestones and royalties.

Eftilagimod alpha (IMP321) – soluble LAG-3 protein as APC activator for solid tumors. We believe it is fair to say that there is a long road ahead for the clinical development of Immutep's LAG-3 assets, but we also can envision a very broad usage of this mechanism – akin to what has been seen for immunotherapies targeting PD-1 and CTLA-4 - that we feel justifies the wait if trial results live up to the theoretical hype. Immutep and its collaborators have begun evaluation of LAG-3 assets in a collection of trials across solid tumor indications including metastatic and triple-negative breast cancer, advanced melanoma, head and neck cancers, non-small cell lung cancer (NSCLC), and autoimmune diseases. In the context of oncology, the predominant approach to targeting LAG-3 has been via antagonistic antibodies, such as the LAG525 asset that Novartis has licensed from Immutep; however, Immutep's lead candidate eftilagimod alpha (efti) pre-dated these efforts in the clinic and employs a different approach through APC activation. As previously discussed, efti binds to MHC Class II, including on the surface of APCs such as monocytes and dendritic cells. This increased APC activation and consequent dendritic cell maturation is only meaningful if relevant tumor antigens are available for presentation to cytotoxic CD8⁺ T cells, and thus Immutep opted to combine efti with chemotherapy in its lateststage AIPAC trial in metastatic breast cancer patients. The efti clinical pipeline has since expanded considerably and is now comprised of four key combination trials with immunotherapy and chemotherapy agents (including anti-PD-1 given synergies reported from a host of pre-clinical studies and Bristol-Myers Squibb' ongoing work combining their anti-LAG-3 antibody with Opdivo), which are discussed separately below.



Source: Company presentation (April 2020).

Phase 2b AIPAC trial in HR+, HER2- metastatic breast cancer. The program furthest along in Immutep's pipeline is the Phase 2b AIPAC trial of efti comparing the combination of efti + taxane chemotherapy paclitaxel to paclitaxel + placebo in hormone-positive metastatic breast cancer (MBC) patients. The trial is composed of two stages: 1) A safety run-in phase to establish a recommended Phase 2 dose; and 2) A randomized, two-arm phase comparing the efti + paclitaxel combo to paclitaxel + placebo to evaluate efficacy as measured by PFS (as well as ORR and OS). Data presented to date from a prior Phase 1 trial, as well as the 15-patient safety run-in portion of the Phase 2b AIPAC trial, proved highly consistent and encouraging compared to historical paclitaxel monotherapy response findings. In a Phase 1 trial of 30 patients, an overall response rate (ORR) of 47% was reported (all of which were partial responses), as well as a disease control rate (DCR) of 83%. Initial outcomes from the 15 patients in the Phase 2b AIPAC safety run-in included the same 47% ORR (again, all attributable to partial responses) and a marginally higher DCR of 87%. It is worth noting that these responses often took several months to be fully realized, with two of the seven partial responses seen in the AIPAC safety run-in coming after roughly six months, and marked tumor reductions reported between months three and six in the earlier Phase 1. These results appear to compare guite favorably to the approximately 20-30% response rates observed for paclitaxel monotherapy across a basket of trials, though durability of these responses and overall long-term outcomes remain to be determined.

Exhibit 4: Eftilagimod Alpha Results in Metastatic Breast Cancer Through AIPAC Run-In

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AIPAC – Safety Ru	ın Phase (n=15)
Response Parameter	Paclitaxel + IMP321 (n = 15)
Complete Response (CR)	0/15 (0%)
Partial Response (PR)	7/15 (47%)
Stable Disease (SD)	6/15 (40%)
Progressive Disease (PD)	2/15 (13%)
Overall Response Rate (ORR)	7/15 (47%)
Disease Control Rate (DCR)	13/15 (87%)

ORR of 47% and DCR of 87%
Two of the responses occurred

relatively late (after ~6 months)

Source: Company presentation (October 2018).

shrinkage between months 3 and 6

By contrast, the headline takeaway that prompted the March sell-off was inarguably the fact that the efti + paclitaxel arm of AIPAC did not demonstrate a statistically significant improvement in median PFS relative to paclitaxel monotherapy (HR=0.93), despite showing a numerical improvement of 7.16-7.29 months median PFS compared to 6.70-7.16 months for paclitaxel monotherapy. This result seemingly ruled out the hope that AIPAC could serve as a registrational trial for the efti combination (as far as the EMA is concerned), but closer examination of survival curves presented in Immutep's subsequent webcast (Exhibit 5) does justify Immutep's decision to continue clinical development of efti in MBC, in our view. Per AIPAC's protocol, all patients received paclitaxel for six months in addition to either efti or placebo every two weeks, followed by the removal of paclitaxel at this timepoint and reduced dosing of efti or placebo every four weeks for the remainder of the trial. We believe it is very telling that this reduction in the dosing frequency of efti coincided almost exactly with the survival curves of both arms coming together and ultimately determining their respective median PFS results.

This suggests to us that, outside of the higher than expected ORR of 38% for paclitaxel monotherapy (ORR for efti + paclitaxel was 48%, marginally higher than the previously observed 47%), reduced dosing of efti at this timepoint may have contributed to a prematurely short median PFS for the investigational arm. Further justification for this hypothesis may come from the fact that, with continued dosing of efti every four weeks, survival curves eventually began to separate again between months eight and ten of treatment. These observations indicate to us that, given the largely tolerable safety profile of efti across trials, Immutep may be able to explore higher doses of efti in combination with paclitaxel, or remove the tapering of dosing at six months in subsequent testing. Overall survival (OS) data is yet to be reported from AIPAC (and is expected later this year), but we believe odds of demonstrating a statistically significant improvement on this measure remain somewhat feasible based upon these longer-term trends in PFS. In addition to potential dosing protocol tweaks in planned Phase 3 testing of efti in MBC, adjustments to the number of patients enrolled in this next phase of testing may increase the odds of success on PFS and OS assessments from a powering perspective, taking into consideration that AIPAC enrolled a mid-stage population of approximately 113 patients per arm.

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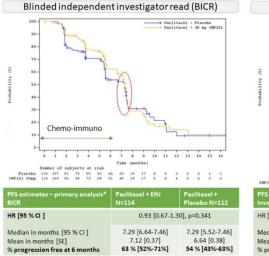


Exhibit 5: Kaplan-Meier PFS Curves for Phase 2b AIPAC Trial Total Population

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Probability (%)	40 -								1									
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Plac		112	107	92	87	66	66	49	43	19	18	11		4	3	2	1	1
IMP321 3	10mg	114	108	94	90	77	74	55	51	24	21	11		5	4	2	2	2
PFS est nvesti				mai	ry ar	haly	vsis*		acli N=11		el + 6	fti				itax	el + N=1	112
HR [95	% C	1]								(0.92	[0.6	9-1.	23]	, p=(0.30	5	
Median	n in i	mon	ths [95	% CI]			7.1	16 [5	5.65	-7.3	9]		6.70	0 [5.	52-7	7.33]
Mean i	n me	onth	s [St	E]						6.81	[0.	33]			6	.30	[0.3	1]
% prog	ress	ion t	free	at 6	mo	nth	s		57	% [4	47%	-669	61		54 9	% [4	3%-1	63%]

Investigator Read

Paclitaxel + Placebo Paclitaxel + 30 mg IM

Exhibit 6: Baseline Patient Characteristics for Phase 2b AIPAC Trial

	Paclitaxel + Efti N=114	Paclitaxel + Placebo N=112
Median age (range)	58 yrs (24-87)	61 yrs (35-79)
ECOG 0	60.5 %	62.5 %
% visceral disease	90.4%	92.9%
% pre-treated with CDK4/6 for met disease	43.9%	42.9%
One or more systemic therapies for metastatic disease	68.4%	71.4%
Tumor type (central pathology) Luminal A Luminal B	34.1%* 48.8%*	36.7%* 49.4%*
Monocytes at baseline < 0.25 x 10°/L	21.9%	19.8%

Source: Company presentation (March 2020).

Source: Company presentation (March 2020).

Company management did emphasize that select patient subgroups exhibited greater PFS improvements relative to the overall study population: those with low monocyte counts at baseline (7.29 months median PFS for the combo vs. 5.45 for paclitaxel monotherapy, HR=0.61), those with luminal B immunogenic tumors (7.29 months median PFS for the combo vs. 5.45 for paclitaxel monotherapy, HR=0.65), and those with lower performance status (7.13 months median PFS for the combo vs. 6.67 for paclitaxel monotherapy, HR=0.76). Though Immutep may opt to focus on one or more of these subgroups in Phase 3 testing and potential commercialization, we believe results for the broader population show significant enough merit to avoid positioning efti as a more niche adjuvant for patients treated with paclitaxel, that would reduce the addressable market for efti by roughly 50% (per company estimates and observed patient characteristics in AIPAC [Exhibit 6]. While prior guidance suggested that the combined datasets from AIPAC and a planned bridging study in the US would be sufficient for FDA approval, we have incorporated expenses and timelines for US approval hinging upon a larger Phase 3 study in our current estimates. We believe these results have no discernible impact on the development of efti for other indications pursued internally or with large pharma partners, including melanoma, head and neck carcinomas, and NSCLC (we do not currently include revenues or expenses for development in head and neck or NSCLC in our estimates) and that the MBC program appears likely to advance with the aforementioned modifications.

Phase 1 TACTI-mel trial in advanced melanoma. Despite being an earlier-stage trial, periodic updates from the Phase 1 TACTI-mel trial of efti in unresectable or advanced melanoma patients proved to be markedly positive catalysts for Immutep's stock historically. The trial originally consisted of three cohorts of six patients each, testing three different dosages of efti (1, 6, and 30mg given subcutaneously) in combination with pembrolizumab (Keytruda) at its approved dosage for melanoma. In these three initial cohorts (Part A), administration of efti began at cycle five of pembrolizumab and was given every two weeks. Final efficacy results of this combination were announced in October 2019, suggesting effects in this indication are quite durable. After reporting an update on Part B of the study (a six-patient cohort in which pembro and efti were given together from the start of treatment, rather than introducing efti at cycle 5 of pembro as done in Part A) in May 2019, responding patients retained their level of tumor reduction through at least 48 weeks. Updated Part B efficacy included a 50% ORR and 66% DCR (n=6), compared to a 33% ORR and 66% DCR in Part A (n=18). We believe it is worth highlighting that in addition to a single metabolic CR confirmed by PET scan, six additional patients saw complete disappearance of all target lesions (which we believe effectively constitute CRs from a practical standpoint), whether or not they meet RECIST criteria due to lymph node infiltration (a hypothetical 29% CR rate). These results, and particularly those in Part B (despite rather small patient numbers), compare favorably to what was observed in pembro's KEYNOTE-006 and KEYNOTE-002 registrational studies in ipilimumab(ipi)-naïve and ipi-refractory metastatic melanoma, respectively. In ipi-naïve patients treated with pembro monotherapy, a 33-34% ORR was achieved (for Q3W and Q2W dosing, respectively), while a 21-25% ORR was reported in ipi-refractory patients. Of these response rates, we believe the 21% ORR is likely the most appropriate bar to compare to the efti + pembro combo considering 1) Of the 24 patients enrolled in TACTI-mel, 12 did not respond to pembro monotherapy and may therefore be IO-refractory to some extent; and 2) the 25%, 33%, and 34% ORRs alluded to here were accomplished utilizing a 10mg/kg dose of pembro (with very minimal impact from Q2W vs. Q3W dosing) compared to the 2 mg/kg dose of pembro that resulted in the 21% ORR in KEYNOTE-002, and which was used in combination with pembro in TACTI-mel. Thus, we believe Immutep succeeded on at least two fronts in TACTI-mel, confirming the importance of dosing efti with pembro from the start (as is being done in the ongoing TACTI-002 study) and demonstrating a 33-50% ORR with efti + pembro that is a notable improvement over the 21% ORR for pembro monotherapy in ipi-refractory patients - a result accomplished with a 5x higher dose of pembro relative to the efti + pembro combo in TACTI-mel.

Phase 2 TACTI-002 combination trial of efti + Keytruda in multiple tumor types. Separate from Immutep's combination trial of efti + pembrolizumab in melanoma (the TACTI-mel study), Merck and Immutep announced a collaboration to test this same combination the Phase 2 TACTI-002 trial in three treatment settings: 1st- and 2nd-line NSCLC (Parts A and B, respectively) and 2nd-line head and neck cancer. As of the latest interim update at SITC this past weekend, response rates in both Part A and Part C of TACTI-002 improved marginally from a previously reported ORR of 47% (now 53%, n=17) in the former cohort and 33% (now 39%, n=18) in the latter, driven by one additional response (first recorded at ASCO 2020) in each cohort – which was a CR in Part C (a snapshot of competitive benchmarks are provided in Exhibit 7). Responding patients saw greater degrees of tumor reduction overall with greater follow-up, and the observation of two responses at 8 and 11 months in Part A further suggests that the efficacy of efti + pembro is not only durable but also improves over time. Additionally, a median PFS of 11.8 months (updated from 9+ months as of June) was reported in frontline NSCLC patients treated with the combo, which compares favorably to the median PFS of 8.8 months observed for pembro in combination with pemetrexed and platinum chemo in KEYNOTE-189, and median PFS of 6.4 months for pembro in combination with carboplatin and paclitaxel in KEYNOTE-407 (both of which enrolled frontline NSCLC patients regardless of PD-L1 expression status). These competitive outcomes were achieved with no discernible decrease in tolerability since last report and continue to markedly improve upon the AE profile of these aforementioned pembro combos and other IO/chemo combos as a whole (Exhibit 8). Immutep communicated that it expects to provide further interim readouts regularly, with the next look planned for later this year. We do not project revenues for efti in indications stemming from TACTI-002 at this time, until greater detail is available on the path forward for these collaborative efforts.

Exhibit 7: Results of IO and Chemo in 2nd-Line Head and Neck Cancers

Regimen	ORR ^(1;3)	Median DoR (months)	Median PFS (months) ^(1, 3)	PFS rate at 3 / 6 months ⁽²⁾	Median OS (months) ⁽¹⁾	Main downside/limitations ^(1,2,3)
Chemo	10.1%	5.0	2.3	45% / 20%	6.9	Not effective in >> 50% of patients
Pembro	14.6%	18.4	2.1	40% / 25%	8.4	Not effective in >> 50% of patients
Pembro ≥ 1% CPS	17.3%	18.4 (vs 9.6)	2.3	45% / 30%	8.7	Not effective in >> 50% of patients
Nivo	13.3%	9.7	2.0	37% / 21%	7.7	Not effective in >> 50% of patients

Source: Company presentation (June 2020).

regimens		
Regimen ⁽²⁾	Treatment related adverse events leading to discontinuation	Treatment related adverse events leading to death
Double Chemo	8-22%	1-6%
lpi + Nivo	20%	< 2%
Chemo + Pembro	23-33%	3-8%
Pembro alone	10-15%	< 2%
Efti plus pembro	4%	0%

Exhibit 8: Comparative Discontinuation and Grade 5 AE Rates for IO and Chemo Regimens

Source: Company presentation (June 2020).

Phase 1 INSIGHT/INSIGHT-004 program – intratumoral injection and anti-PD-L1 combo. The INSIGHT clinical program was cleared for initiation in July of 2017 and was originally envisioned as an investigator-sponsored Phase 1 trial to explore the potential of alternative routes of administration for efti – most prominently intratumoral injection.

alternative routes of administration for efti – most prominently intratumoral injection. Investigators hypothesized that intratumoral injection of efti into a tumor site may lead to responses in more distant tumors as a result of efti's ability to increase levels of circulating, activated APCs. In September 2018, Immutep announced that it would be amending this protocol in a clever way in order to accommodate another combination trial for efti, this time with the anti-PD-L1 antibody avelumab. By amending the study into the INSIGHT protocol (under the name INSIGHT-004), Immutep was able to begin clinical study of this combination much sooner than if it had gone through a separate regulatory filing process (by about 6 months) and was consequently advised to take this approach by regulators. Early clinical findings for efti + avelumab in a basket of advanced solid tumors were reasonably strong, with a 42% ORR (up from 33% at ASCO 2020) reported for both dose cohorts (800mg avelumab + 6mg or 30mg efti, n=6 per cohort) in a collection of different indications. Each of the five responses among these initial twelve patients occurred in a unique indication, including adenocarcinoma of the right colon, pleural mesothelioma, squamous cell anal carcinoma, gastroesophageal junction adenocarcinoma, and squamous cell cervical carcinoma. It is relevant to note that nearly half of these patients were unlikely to respond to a PD-L1 combination regimen given their disease characteristics (two had PD-L1 expression status ≤1%) and we believe these early assessments could remain consistent or improve as refining of dosing scheme and time on therapy continues. Rates of severe AEs (Grade 3+) appear to be fairly balanced across these two dose levels of efti, but longer-term follow-up may determine whether a dose of efti on the higher end of this range (or still greater) may be carried forward into subsequent testing.

IMP761 – non-licensed pre-clinical foray into LAG-3's autoimmune prospects. Details on Immutep's other wholly-owned asset IMP761 are fairly limited, as the candidate is still in pre-clinical testing, but it could represent the company's first efforts into developing a LAG-3 therapeutic in the context of autoimmunity (excluding GSK'781 for which GSK now holds global rights). Immutep has disclosed that IMP761 is a humanized, IgG4 monoclonal antibody that may transiently downregulate the activity of chronically activated LAG-3⁺ T cells. GMP manufacturing preparation is now underway, such that we would expect a Phase 1 could begin in 2021 depending upon company bandwidth.

GSK'781 – GSK out-licensed LAG-3 depleting antibody for autoimmune indications. In contrast to IMP761 above, global rights to Immutep's IMP731 (now GSK'781) have been out-licensed to GlaxoSmithKline for potential use in a collection of autoimmune indications currently under consideration (ulcerative colitis and psoriasis for a start). Unique to IMP761's proposed transient LAG-3⁺ T cell downregulation mechanism, GSK'781 is a depleting antibody designed to target and kill LAG-3⁺ T cells that become auto-reactive. GSK has initiated a Phase 2 trial for GSK'781 in ulcerative colitis and a Phase 1 trial in psoriasis, but timelines for data readout and possible transition into later-stage testing are difficult to predict at this time. Immutep is entitled to up to £64M and tiered single-digit royalties on GSK'781 if approved.

LAG525 – Novartis out-licensed antagonist LAG-3 antibody in mix of oncology combos. Since Immutep handed over global rights to IMP701 (now LAG525) to Novartis, an antagonistic antibody targeting LAG-3, it has proven to be one of Immutep's most active licensing arrangements. Novartis initiated a Phase 1 study of LAG525 in combination with PDR001 (an anti-PD-1 antibody) in a laundry list of tumor types in 2015 and has since moved into a number of Phase 2 trials. In December of 2017, it was announced that the LAG525 + PDR001 combination would move forward into a 160-patient Phase 2 study in advanced solid and hematological cancers. Additionally, three more Phase 2 studies in triple-negative breast cancer (a separate Phase 1b chemo + LAG525 trial in triple-negative breast cancer is also ongoing), metastatic melanoma, and further solid tumors were subsequently disclosed. Given Novartis' broad utilization of LAG525 from an indication perspective, we do not currently forecast revenues (in the form of potential milestones and royalties to Immutep) for this asset currently, but may opt to do so should Novartis continue to move these programs along and define a clearer path to market.

Competitive Landscape: LAG-3 Clinical Trials Are Beginning to Crop Up Like Weeds, but Immutep has the Green Thumb Likely Needed to Yield the Greatest Harvest

Though Immutep's Frédéric Triebel first described the functioning of LAG-3 in 1990 and eftilagimod alpha (efti) began its clinical voyage in 2006 (followed by Bristol-Myers Squibb's anti-LAG-3 antibody in 2013), it is only in the last seven years that development interest has taken off, seemingly in tandem with Bristol-Myers' entry into the clinic. Over that time period, the number of clinical trials revolving around a LAG-3 mechanism has risen ten times over, and at least 15 companies that we are aware of have a clinical trial in the works. Many of these companies (depicted in Exhibit 9) are much larger entities than Immutep with more resources at their disposal; however, we believe Immutep is the most attractive of these options from an investment perspective and also stands to gain the most from any positive developments in the LAG-3 clinical development landscape for the following key reasons: 1) Immutep has the longest track record of experience with the biology of LAG-3 and broad IP around its assets that reflects this; 2) Of the manifold companies developing a LAG-3-targeted asset, Immutep is the second furthest in clinical testing based upon the number and stage of ongoing studies (behind Bristol-Myers); 3) As a much smaller company by effectively all measures compared to the majority of competitors in the LAG-3 space, Immutep's pipeline is entirely focused on progressing this therapeutic modality and the company stands to see the greatest appreciation in its valuation should one or more of its LAG-3 assets reach market approval, relative to diffuse large pharma players; and 4) While early, preliminary data from Immutep's trials of efti is strikingly competitive compared to results of Bristol-Myers' LAG-3 blocking antibody in the advanced melanoma setting where both assets are being evaluated in combination with anti-PD-1 antibodies (as described in greater detail below).



Exhibit 9: Immutep is Punching Well Above Its Market Cap in LAG-3 Clinical Development

Source: Company presentation (April 2020).

LAG-3 immunotherapy market has room for multiple participants, and Immutep may reap added benefits by casting a wide net. As alluded to throughout this report, clinical development of assets targeting LAG-3 is still rather early despite the recent surge in interest over the last several years. In light of this, there are very few well-established participants in a position to dominate this potentially substantial market if targeting LAG-3 indeed offers a broadly applicable benefit to patients with cancer in a manner similar to that of PD-1 and CTLA-4 checkpoint inhibitors. We anticipate that if these prior checkpoint inhibitors serve as any guide, the market for LAG-3 therapeutics is large enough to support multiple approved products, and only Bristol-Myers is defensibly ahead of Immutep in the clinic (albeit with mechanisms that engage LAG-3 functionality in distinct ways that are not directly comparable). Through its collaborations with and out-licenses to Merck, Novartis, and GSK, Immutep is positioned to benefit from LAG-3 successes produced by any of these companies, in our view, and we do not perceive the abundance of companies, with Phase 1 assets in one or two ongoing trials each, as substantive threats to Immutep's more extensive and mature LAG-3 pipeline. Thus, for now we believe the only meaningful comparisons that can be made between clinical data on LAG-3 therapeutics at this time are between Immutep's efti and Bristol-Myers' relatlimab (a.k.a. BMS-986016).

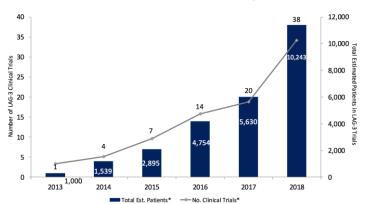


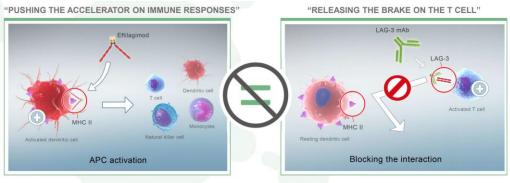
Exhibit 10: LAG-3 Clinical Interest is a Rising Tide that Could Lift Focus on All LAG-3 Assets

Combo agent monotherapy and LAG-3 peers offer most relevant reference points. As for comparisons between Immutep's LAG-3 assets and current treatment standards already on the market, given that targeting of LAG-3 will almost certainly be combined with other mechanistic approaches (potentially with a broad range of chemotherapies and immunotherapies), we believe at this stage it is more prudent to focus on the degree of synergies between LAG-3 agents and combination therapies relative to the monotherapy outcomes of these established treatments. The comparison made previously in this report between the monotherapy activity of paclitaxel and paclitaxel + efti from early results of the AIPAC trial serves as one such example. Ultimately, clinical data for LAG-3 is limited at this time, and it is consequently difficult to make applicable juxtapositions between datasets until more conclusive results are available. As data from players in the LAG-3 space matures we intend to draw further conclusions, but for now we turn to assessing Immutep's LAG-3 pipeline alongside that of peers with assets targeting this emerging immunotherapy mechanism.

Source: GlobalData, Company websites, Clinicaltrials.gov, Sec.gov, and Immutep company presentation (October 2018).

Bristol-Myers Squibb's relatlimab response rates in advanced melanoma fall well short of efti's in preliminary findings. Bristol-Myers' LAG-3 antagonist antibody relatlimab (BMS-986016) is the most directly comparable clinical comparator to Immutep's efti (and to the LAG525 asset out-licensed to Novartis), in our view, as it is being explored in numerous oncology indication trials, some of which overlap with those of efti. That said, it is important to point out that the mechanisms of efti and relatlimab are rather different from one another (depicted in Exhibit 11). Relatlimab is a monoclonal antibody designed to block LAG-3 interaction with tumor-infiltrating T cells, allowing them to retain more of their cytotoxic activity that may have been downregulated by expressed LAG-3, while efti is a soluble form of the LAG-3 Ig protein itself that supports greater T cell engagement and activity by activating APCs and inducing dendritic cell maturation. Both efti and relatlimab are being evaluated in unresectable/advanced melanoma patients that have not responded to anti-PD-1/PD-L1 therapy (some of which have also received anti-CTLA-4, BRAF, or MEK inhibitors) to see if these refractory patients can be re-sensitized to anti-PD-1 therapy when it is combined with a LAG-3 agent. Efti activity is being assessed in combination with Merck's anti-PD-1 pembrolizumab (Keytruda), while Bristol-Myers is combining relatlimab with its anti-PD-1 nivolumab (Opdivo).

Exhibit 11: Eftilagimod Alpha and Relatlimab Both Implicate LAG-3 but with Altogether Different Mechanisms



LAG-3Ig, an MHC II agonist (eftilagimod alpha): Source: Company presentation (August 2018). LAG-3 antagonist antibodies:

Patients, n (%)		м	el Prior IO (n = 48ª)					
BOR CR PR ^e SD PD Clinical progressions ^e			0 6 (13) 20 (42) 16 (33) 6 (13)					
ORR , 95% CI° LAG-3 \geq 1% (n = 25) LAG-3 < 1% (n = 14)	6 (13), 4.7, 25 5 (20), 6.8, 41 1 (7.1), 0.2, 34							
$ \begin{array}{l} \text{DCR} (\text{CR} + \text{PR} + \text{SD})^{\circ} \\ \text{LAG-3} \geq 1\% \ (n = 25) \\ \text{LAG-3} < 1\% \ (n = 14) \end{array} $		26 (54) 16 (64) 5 (36)						
			Mel Prior IO (n = 48ª))				
			0	RR				
		n	n (%)	95% CI				
LAG-3 expression ≥ 1% < 1%		25 14	5 (20) 1 (7.1)	6.8, 41 0.2, 34				
PD-L1 expression ≥ 1% < 1%		16 19	2 (13) 4 (21)	1.6, 38 6.1, 46				

Exhibit 12: Phase 1/2a Relatlimab Response Breakdowns Presented at ASCO 2017

Source: Ascierto, P. et al., "Initial Efficacy of Anti-Lymphocyte Activation Gene-3 (anti–LAG-3; BMS-986016) in Combination With Nivolumab in Patients With Melanoma Previously Treated With Anti–PD-1/PD-L1 Therapy." ASCO 2017.

Bristol-Myers reported initial findings from this Phase 1/2a combination trial of relatlimab + nivolumab at the ASCO and ESMO medical conferences in 2017, which included response rates and safety findings for the combination. At ASCO 2017 (results above in Exhibit 12), data indicated an ORR of 12.5% (all PRs) in 48 evaluable patients, and further stratified data to show that response rates for the 14 patients with >1% LAG-3 expression were notably higher (ORR of 20%) compared to the 25 patients with <1% LAG-3 expression (ORR of 7.1%). It is worth noting that LAG-3 expression on activated T cells may be less critical to the efficacy of efti, as it acts by binding MHC Class II on APCs as a recombinant LAG-3 protein itself, rather than directly blocking the binding of LAG-3 protein to LAG-3⁺ T cells and other LAG-3⁺ cell types. An update on the relatlimab findings at ASCO 2017 was provided later the same year at ESMO 2017, where results proved largely consistent with the prior findings. The relatlimab + nivolumab combo produced an ORR of 11.5% in 61 patients, which was again markedly higher in >1% LAG-3 expressers with an ORR of 18% compared to an ORR of 5% for patients with <1% LAG-3 expression. Due to the characteristic secrecy of large pharma clinical development, relatlimab has quietly advanced considerably, and is now scheduled to report potentially registrational data for relatlimab + Opdivo in frontline melanoma later this year or in early 2021. Pending comparison of this combination to Phase 1 efti + pembro results in melanoma, we anticipate positive results from relatlimab + Opdivo (and potential NDA filing thereafter) may provide significant validation for efti in melanoma and beyond.

FINANCIAL TABLES

IMMP Income Statement, with	Project	ions				Projection	s are shaded i	light gray										
(\$ AU, in thousands; FY end June)	2017A	2018A	1H:19A	2H:19A	2019A	1H:20A	2H:20E	2020E	1H:21E	2H:21E	2021E	2022E	2023E	2024E	2025E	2026E	2027E	2028E
Operating Revenue																		
Product Sales	-	-	-	-	-	-	-	-	-	-	-	-	49,630	237,066	474,702	701,085	910,422	1,095,935
Milestone Revenues	-	2,630	-	140	140	7,366	-	7,366	-	-	-	-	-	-	-	-	-	-
TOTAL Revenue	-	2,630	-	140	140	7,366	-	7,366	-	-	-	-	49,630	237,066	474,702	701,085	910,422	1,095,935
Operating costs and expenses						Ī				-								
Cost of products sold	-	-	-	-	-	-	-	-	-	-	-	-	7,444	35,560	71,205	98,152	118,355	131,512
Depreciation and amortization	1,702	1,809	943	936	1,879	965	1,373	2,338	1,530	1,695	3,226	4,099	5,369	6,917	8,818	11,166	14,078	17,698
Research and development	7,526	9,990	7,582	9,009	16,591	11,899	8,924	20,823	9,817	13,743	23,560	28,272	24,879	27,367	34,209	47,893	71,840	104,167
Selling, general and administrative	4,347	7,242	3,254	3,112	6,366	3,088	2,625	5,714	2,835	3,402	6,237	6,861	10,292	13,379	16,055	22,477	33,716	50,574
TOTAL Operating Expenses	14,326	20,098	11,544	13,328	24,872	15,905	13,576	29,480	14,860	19,669	34,529	41,082	50,258	86,017	133,721	183,907	243,172	310,322
TOTAL Operating Income (Loss)	(14,326)	(17,467)	(11,544)	(13,188)	(24,732)	(8,538)	(13,576)	(22,114)	(14,860)	(19,669)	(34,529)	(41,082)	(628)	151,049	340,982	517,178	667,250	785,613
Other income (expense):																		
Grant income	3,316	3,214	2,124	2,218	4,342	2,152	-	2,152	-	-	-	-	-	-	-	-	-	-
Interest income	104	177	198	199	397	137	170	307	254	149	403	1,249	422	272	2,714	8,629	18,105	29,798
Interest expense	-	-	-	-	-	(6)	-	(6)	-	-	-	-	-	-	-	-	-	-
Miscellaneous income	800	1,009	157	998	1,155	79	-	79	-	-	-	-	-	-	-	-	-	-
Total Other Income (Expenses)	4,222	4,723	2,871	3,518	6,388	2,159	170	2,329	254	149	403	1,249	422	272	2,714	8,629	18,105	29,798
Profit or Loss Before Taxes	(10,105)	(12,744)	(8,674)	(9,670)	(18,344)	(6,379)	(13,406)	(19,785)	(14,606)	(19,520)	(34,126)	(39,833)	(206)	151,321	343,696	525,807	685,355	815,410
Income tax (expense) / gain	(737)	2	5	(5)	-	0	-	-	-	-	-	-	-	-	65,301	144,597	188,473	224,238
Net Profit or Loss	(9,367)	(12,746)	(8,678)	(9,665)	(18,344)	(6,379)	(13,406)	(19,785)	(14,606)	(19,520)	(34,126)	(39,833)	(206)	151,321	278,395	381,210	496,882	591,172
Basic weighted average common shares	2,072,450	2,608,328	3,099,461	3,351,691	3,225,576	388,798	418,798	403,798	455,530	490,039	483,307	516,962	542,810	569,951	598,448	628,371	659,789	692,779
Diluted weighted average common shares	2,072,450	2,608,328	3,099,461	3,351,691	3,225,576	388,798	418,798	403,798	455,530	490,039	483,307	516,962	542,810	569,951	598,448	628,371	659,789	692,779
Basic net (loss) / income per common share \$	(0.00)	(0.00)	(0.00)	(0.00)	(0.01)	(0.02)	(0.03)	(0.05)	(0.03)	(0.04)	(0.07)	(0.08)	(0.00)	0.27	0.47	0.61	0.75	0.85
Diluted net (loss) / income per common share \$	(0.00)	(0.00)	(0.00)	(0.00)	(0.01)	(0.02)	(0.03)	(0.05)	(0.03)	(0.04)	(0.07)	(0.08)	(0.00)	0.27	0.47	0.61	0.75	0.85
Basic net (loss) / income per ADR \$	(0.45)	(0.49)	(0.28)	(0.29)	(0.57)	(0.16)	(0.32)	(0.49)	(0.32)	(0.40)	(0.71)	(0.77)	(0.00)	2.65	4.65	6.07	7.53	8.53
Diluted net (loss) / income per ADR \$	(0.45)	(0.49)	(0.28)	(0.29)	(0.57)	(0.16)	(0.32)	(0.49)	(0.32)	(0.40)	(0.71)	(0.77)	(0.00)	2.65	4.65	6.07	7.53	8.53

Source: Company reports and Alliance Global Partners projections.

IMMP Balance Sheet, with Projecti	ons				Projection	s are shaded	light gray								
(\$ AU, in thousands; FY end June)	2017A	2018A	1H:19A	2019A	1H:20A	2020E	1H:21E	2021E	2022E	2023E	2024E	2025E	2026E	2027E	2028E
ASSETS															
Cash and cash equivalents	12,237	23,476	26,002	16,568	20,516	30,581	17,971	52,036	17,577	11,343	113,049	359,372	754,031	1,241,014	1,835,5
Short-term investments	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
Restricted cash	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
Prepaid and other current assets	1,488	1,736	669	1,780	1,596	2,089	2,423	2,608	3,103	3,796	6,479	10,100	13,891	18,367	23,3
Accounts receivable	2,194	3,432	3,665	5,194	4,745	4,335	5,448	6,561	9,930	16,317	58,295	91,039	96,039	124,715	149,7
Inventories	-	-	-	-	-	-	-		-	4,895	17,488	27,312	26,891	32,426	35,9
TOTAL current assets	15,919	28,643	30,336	23,542	26,858	37,005	25,842	61,205	30,610	36,350	195,312	487,823	890,851	1,416,523	2,044,6
Property and equipment, net	24	26	38	53	57	61	69	78	105	137	177	226	286	361	4
Intangibles	19,020	18,329	17,865	16,947	15,782	14,697	13,686	12,745	11,053	9,585	8,312	7,209	6,252	5,421	4,7
Long-term investments	-	-	-	-	-	-	-		-	-	-	-	-	-	-
Restricted cash	-	-	-	-	-	-	-		-	-	-	-	-	-	-
Other assets	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
TOTAL long-term assets	19,045	18,356	17,903	17,000	15,838	14,757	13,755	12,823	11,158	9,723	8,490	7,435	6,538	5,782	5,1
TOTAL assets	34,964	46,999	48,239	40,541	42,955	52,022	39,857	74,288	42,028	46,333	204,061	495,517	897,649	1,422,565	2,050,0
LIABILITIES															
Accounts payable	2,589	3,664	3,970	5,060	3,615	3,120	3,378	4,545	5,407	6,615	11,291	17,600	24,206	32,007	40,7
Employee benefits	43	190	161	239	225	242	248	254	267	280	294	309	324	340	3
TOTAL current liabilities	2,632	3,853	4,131	5,299	3,966	3,488	3,752	4,925	5,800	7,021	11,711	18,035	24,656	32,473	41,2
Convertible note liability	5,779	6,646	7,143	7,643	8,214	8,830	9,493	10,205	11,793	9,434	3,774	-	-	-	-
Warrant liability	-	2,945	3,393	3,164	2,545	2,672	2,805	2,946	3,248	2,598	1,559	935	234	-	-
Employee benefits	20	32	42	48	55	55	55	55	55	55	55	55	55	55	
Other liabilities	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
TOTAL liabilites	8,431	13,477	14,709	16,154	14,924	15,189	16,248	18,274	21,039	19,252	17,242	19,169	25,089	32,672	41,4
TOTAL stockholders' equity (deficit)	26,532	33,522	33,530	24,388	28,032	36,834	23,609	56,014	20,989	27,081	186,819	476,348	872,560	1,389,893	2,008,6
Total liabilities and stockholders' equity	34,964	46,999	48,239	40,541	42,955	52,022	39,857	74,288	42,028	46,333	204,061	495,517	897,649	1,422,565	2,050,0
End of period shares used in computation (thousands)	2,072,450	2,608,328	3,099,461	3,225,576	388,798	448,798	462,262	504,353	529,571	556,049	583,852	613,044	643,697	675,881	709,67
SELECTED METRICS															
Current ratio	6.05x	7.43x	7.34x	4.44x	6.77x	10.61x	6.89x	12.43x	5.28x	5.18x	16.68x	27.05x	36.13x	43.62x	49.0
Norking capital	\$13,287	\$24,790	\$26,205	\$18,243	\$22,892	\$33,518	\$22,091	\$56,281	\$24,810	\$29,329	\$183,601	\$469,788	\$866,195	\$1,384,050	\$2,003,4
Book value per share	\$0.01	\$0.01	\$0.01	\$0.01	\$0.07	\$0.08	\$0.05	\$0.11	\$0.04	\$0.05	\$0.32	\$0.78	\$1.36	\$2.06	\$2
Cash, cash equivalents and current investment	\$12,237	\$23,476	\$26,002	\$16,568	\$20,516	\$30,581	\$17,971	\$52,036	\$17,577	\$11,343	\$113,049	\$359,372	\$754,031	\$1,241,014	\$1,835,
Cash, cash equivalents and all investment	\$12,237	\$23,476	\$26,002	\$16,568	\$20,516	\$30,581	\$17,971	\$52,036	\$17,577	\$11,343	\$113,049	\$359,372	\$754,031	\$1,241,014	\$1,835,
Cash, cash equivalents/common share	\$0.01	\$0.01	\$0.01	\$0.01	\$0.05	\$0.07	\$0.04	\$0.10	\$0.03	\$0.02	\$0.19	\$0.59	\$1.17	\$1.84	\$2
Debt															
Debt to (stockholder's) equity ratio															

Source: Company reports and Alliance Global Partners projections.

IMMP Cash Flow Statement, with Proje	ections		Projections	are shaded	light gray							
(\$ AU, in thousands; FY end June)	2017A	2018A	2019A	2020E	2021E	2022E	2023E	2024E	2025E	2026E	2027E	2028E
OPERATING ACTIVITIES												
Net Profit / (Loss)	(9,367)	(12,746)	(18,344)	(19,785)	(34,126)	(39,833)	(206)	151,321	278,395	381,210	496,882	591,172
Reconciliation of net loss to net cash:												
Depreciation and amortization	1,702	1,809	1,879	2,685	3,226	4,099	5,369	6,917	8,818	11,166	14,078	17,69
Stock-based compensation expense	862	2,264	1,582	1,829	2,053	2,421	2,424	2,808	3,464	4,849	7,274	10,663
Change in fair value of convertible note liability	752	867	997	1,188	1,374	1,588	(2,359)	(5,660)	(3,774)	-	-	-
Change in fair value of warrants	-	190	(961)	(493)	274	302	(650)	(1,039)	(624)	(701)	(234)	-
Changes in operating assets and liabilities:												
Account receivables	(2,026)	(1,238)	(1,762)	859	(2,226)	(3,369)	(6,387)	(41,978)	(32,744)	(5,000)	(28,676)	(25,00
nventories	-	-	-	-	-	-	(4,895)	(12,593)	(9,823)	421	(5,535)	(3,50
Prepaid expenses and other current assets	(865)	(247)	(44)	(309)	(519)	(495)	(693)	(2,683)	(3,621)	(3,791)	(4,476)	(5,00
Accounts payable	1,377	1,075	1,397	(1,941)	1,425	863	1,208	4,676	6,310	6,606	7,801	8,72
Change in employee benefits	(7)	158	64	10	12	13	13	14	15	15	16	1
NET OPERATING CASH FLOWS	(8,507)	(7,777)	(15,286)	(15,957)	(28,507)	(34,411)	(6,175)	101,781	246,416	394,774	487,129	594,76
NVESTING ACTIVITIES												
Purchase of property and equipment	(7)	(12)	(41)	(30)	(38)	(48)	(59)	(74)	(93)	(116)	(145)	(18
Purchases of investments	-	-	-	-	-	-	-	-	-	-	-	-
Maturities of investments	-	-	-	-	-	-	-	-	-	-	-	-
NET INVESTING CASH FLOWS	(7)	(12)	(41)	(30)	(38)	(48)	(59)	(74)	(93)	(116)	(145)	(18
FINANCING ACTIVITIES												
Net proceeds from the issuance of common stock and options	0	16,968	4,871	30,000	50,000	-	-	-	-	-	-	-
Share issue transaction costs	(9)	(1,319)	(773)	-	-	-	-	-	-	-	-	-
Dthers	-	-	-	-	-	-	-	-	-	-	-	-
NET FINANCING CASH FLOWS	(9)	18,405	8,013	30,000	50,000							
let increase (decrease) in cash and cash equivalents	(8,522)	10,616	(7,315)	14,013	21,455	(34,459)	(6,235)	101,707	246,323	394,658	486,984	594,57
Cash and cash equivalents at beginning of year or period	20,880	12,237	23,476	16,568	30,581	52,036	17,577	11,343	113,049	359,372	754,031	1,241,01
CASH AND CASH EQUIVALENTS AT THE END OF PERIOD	12,237	23,476	16,568	30,581	52,036	17,577	11,343	113,049	359,372	754,031	1,241,014	1,835,59

Source: Company reports and Alliance Global Partners projections.

(\$ AU, in thousands; FY end June)	2020E	2021E	2022E	2023E	2024E	2025E	2026E	2027E	2028E	Terminal Value
EBIT	(22,114)	(34,529)	(41,082)	(628)	151,049	340,982	517,178	667,250	785,613	
Effective Tax Rate	0%	0%	0%	0%	0%	19%	28%	28%	29%	
Гах	-	-	-	-	-	65,301	144,597	188,473	224,238	
EBIT after tax	(22,114)	(34,529)	(41,082)	(628)	151,049	275,681	372,581	478,777	561,375	
Add: Depreciation and amortization	4,514	5,279	6,520	7,793	9,724	12,281	16,015	21,351	28,361	
dd: Changes in working capital	(1,381)	(1,308)	(2,989)	(10,754)	(52,565)	(39,864)	(1,749)	(30,871)	(24,773)	
.ess: Capex	30	38	48	59	74	93	116	145	181	
Free cash flow to the firm (FCFF)	(19,011)	(30,596)	(37,598)	(3,649)	108,134	248,006	386,731	469,113	564,781	1,564,028
lime period (years)	-	1	2	3	4	5	6	7	8	8
PV Factor	1.000	0.720	0.519	0.374	0.269	0.194	0.140	0.101	0.072	0.072
Discounted FCFF	(19,011)	(22,038)	(19,507)	(1,364)	29,107	48,084	54,008	47,188	40,921	113,320

Terminal Value and NPV Worksheet (\$ AU, thousands)		Sensitivity Table			Те	rminal Growth	Rate	
Discounted FCFF (Fiscal 2021-2028)	176,398		_	0.0%	1.0%	2.0%	3.0%	4.0%
Terminal Value	113,320	Discount	29%	\$9.50	\$9.75	\$10.00	\$10.00	\$10.25
Implied Enterprise Value	289,718	Rate	34%	\$6.75	\$6.75	\$7.00	\$7.00	\$7.25
Less: Net Debt \ (Cash)	(21,751)		39%	\$5.00	\$5.00	\$5.00	\$5.25	\$5.25
Add:Investments	-		44%	\$3.75	\$3.75	\$3.75	\$3.75	\$4.00
Implied Market Cap (\$ USD)	205,570		49%	\$2.75	\$3.00	\$3.00	\$3.00	\$3.00
NPV per ADR (target price)	\$5.00			(Rounded to nearest \$0.25)				
Current Market Price per ADR (Last Closing Price)	\$1.27							
Upside/(Downside)	293.7%							
Common shares outstanding (est. at fiscal year-end 2020)	403,797,604							
Common share to ADR ratio	10:1							
Discount Rate	39%							

2%

Source: Company reports and Alliance Global Partners projections.

Terminal Growth Rate

Important Research Disclosures



Created by: BlueMatrix

Distribution of Ratings/IB Services

			IB Serv./Past 12 Mos.			
Rating	Count	Percent	Count	Percent		
BUY [BUY]	70	83.33	29	41.43		
HOLD [NEUTRAL]	11	13.10	2	18.18		
SELL [SELL]	0	0.00	0	0		
NOT RATED [NR]	3	3.57	0	0		
UNDER REVIEW [UR]	0	0.00	0	0		

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2 (Low to medium): Modest changes in stock price in a 12 month period

3 (Medium): Average fluctuation in stock price in a 12 month period

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