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COMPANY NOTE

Immutep Limited (IMM-AU)

Unlocking the value of LAG-3 in autoimmunity

OUTPERFORM

Target Price AUD0.078
Current Price AUD0.030

KEY TAKEAWAY

The biopharma industry is racing to develop combination therapies with approved immune checkpoint ("IC") inhibitors targeting mainly PD-1 / L1 to augment response rates. These include agents targeting other ICs such as LAG-3, TIM-3 and TIGIT. Discovered by Immutep's CSO / CMO in 1990, LAG-3 has been gaining increasing attention in the last few years, with c.50 trials involving >10,400 patients ongoing. Immutep has the broadest LAG-3 targeting pipeline, which includes four molecules with different mechanisms of action, and to our knowledge is the only company (with partner GSK) to have LAG-3 targeted molecules for autoimmune disease. In this note, we review the industry's overall LAG-3 pipeline and provide a detailed analysis of Immutep's autoimmune assets, which are not yet adequately reflected in our valuation. Hence, we see room for upside to our target price ("TP") of A$0.078, which largely reflects the value of Immutep's oncology assets. Reiterate OUTPERFORM.

Clinical trials for LAG-3 targeted compounds expanding fast

An analysis of clinicaltrials.gov reveals that there are approx. 50 ongoing or recently completed clinical trials involving LAG-3 targeted assets, up from one in 2013. 2018 was a particularly busy year with 24 trial starts. All trials except one target cancers, predominantly skin, lung, colorectal, gastric and breast, as well as lymphomas. Most trials test combination therapies, mainly with PD-1 / L1 inhibitors, but also chemotherapy. BMS is by far the most active company: its anti-LAG-3 mAb relatlimab is undergoing around 24 trials involving >5,300 patients, of which a few are expected to read out this year. Immutep's pipeline includes four LAG-3 targeting assets, all with different mechanisms of action. Two compounds are in clinical development for solid tumours (eftilagimod alpha / IMP321, LAG525 / IMP701) and two focus on autoimmune diseases (GSK2831781 [based on Immutep's IMP731], IMP761).

Tackling the root cause of autoimmune disease

To our knowledge, GSK2831781 and IMP761 are the only LAG-3 targeted product candidates in development for autoimmune diseases. Both assets aim to silence autoreactive T cells, therefore acting on the root cause of autoimmune disease and upstream of current therapies, which block inflammatory molecules to provide symptomatic relief. GSK2831781, partnered with GSK since 2011, has successfully completed a Phase I / Ib trial in healthy volunteers and patients with plaque psoriasis. It is due to enter a Phase II trial in ulcerative colitis ("UC") this year, with GSK recently confirming that proof of concept data is expected in H2/2020E. Unencumbered asset IMP761 should start first-in-human studies in H2/2020E (cell line development for GMP manufacturing is ongoing) following encouraging *in vivo* studies recently presented at the European Crohn's and Colitis Organisation ("ECCO") meeting.

Autoimmune assets not yet reflected in our valuation

Our valuation for Immutep consists almost entirely of risk-adjusted net present values ("rNPVs") for the cancer assets plus net cash, including negligible value for GSK2831781 and IMP761. Hence, there could be upside to our TP in the coming year as we get more visibility on the continued development of these assets. We remind investors that the key catalyst for Immutep is Phase IIb data for lead asset eftilagimod alpha from the event-driven AIPAC trial in metastatic breast cancer ("mBC"), now expected in Q4/2019E. A positive outcome would increase our fair value to A$0.110.

FINANCIAL SUMMARY

Net Cash/Debt (M):	13.88

MARKET DATA

Current Price:	AUD0.030
Target Price:	AUD0.078
52 Week Range:	AUD0.060 - AUD0.020
Total Enterprise Value:	72
Market Cap (M):	91
Shares Out (M):	3,384.0
Float (M):	3,210.0
Average Daily Volume:	3,135,217

**Target Price: AUD 0.078 / USD 5.8 (ADR)*

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PRICE PERFORMANCE

Source: Factset

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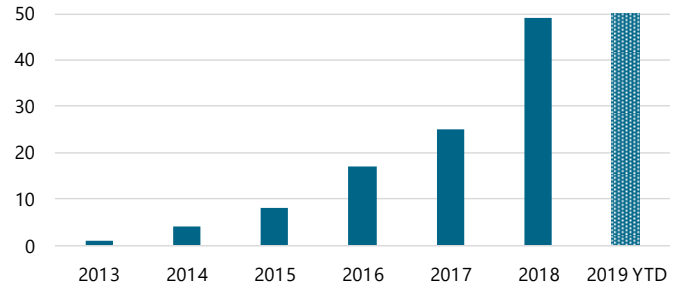
Expansion of clinical trials involving LAG-3 agents

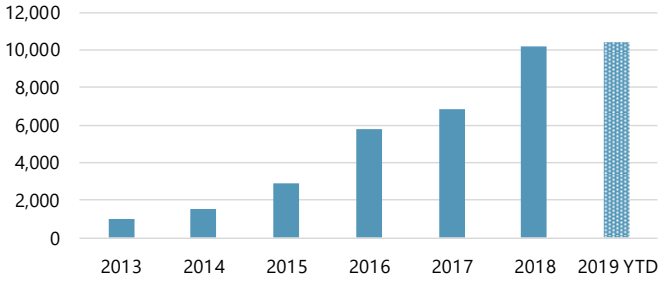
An analysis of clinicaltrials.gov reveals that as of 12 March 2019, there were around 50 ongoing or recently completed clinical trials for LAG-3 targeted assets (CHART 1) involving >10,400 patients (CHART 2). 2018 was a particularly busy year with 24 new trial starts, most of which were related to relatlimab.

CHART 1: Cumulative no. of clinical trials involving LAG-3 targeted molecules



Source: Clinicaltrials.gov as of 12 March 2019, goetzpartners Research

CHART 2: Cumulative no. of patients enrolled in clinical trials for LAG-3 molecules



Source: Clinicaltrials.gov as of 12 March 2019, goetzpartners Research

Anti-LAG-3 antagonistic mAbs the most common approach

Most innovators have chosen to follow the same rationale as that of approved ICIs, namely, blocking LAG-3 with an antagonistic mAb to release the breaks on inactivated immune cells (CHART 3). This category includes Novartis's / Immutep's LAG525 / IMP701. A few companies are also pursuing bispecific antagonistic mAbs blocking two ICs. The other three approaches are exclusively related to assets emanating from Immutep's pipeline: LAG-3 Ig fusion protein eftilagimod alpha, an immune cell activator, and two assets designed to dampen the immune response in the context of autoimmune indications, GSK2831781 / IMP731 and IMP761.

CHART 3: LAG-3 pipeline: 5 different mechanisms of action for cancer and autoimmune disease

Company	Molecule	MoA	Indications being tested	Combinations	Phase
BMS	Relatlimab	Anti-LAG-3 antagonistic mAb	Melanoma, chordoma, gastric, colorectal, glioblastoma, NSCLC, MSI-H tumours, other solid and haematological tumours	Nivolumab (anti-PD-1), ipilimumab (anti-CTLA-4), chemo (FOLFOX, XELOX, SOX, paclitaxel), other mAbs	2/3
Immutep	Eftilagimod alpha	Anti-LAG-3-Ig-fusion-protein	Breast, NSCLC, HNSCC, melanoma, other advanced solid tumours	Pembrolizumab (anti-PD-1), paclitaxel	2b
Novartis / Immutep	LAG525	Anti-LAG-3 antagonistic mAb	Melanoma, SCLC, gastric, prostate, ovarian, NET, DLBCL, TNBC, other advanced solid tumours	Spartalizumab (anti-PD-1), capmatinib (anti-C-MET), canakinumab (anti-IL-1 beta), carboplatin, AA2A R antagonist, anti-M-CSF	2
BI	BI 754111	Anti-LAG-3 antagonistic mAb	NSCLC, HNSCC, other advanced solid tumours	BI 754091 (anti-PD-1)	2
Merck & Co	MK-4280	Anti-LAG-3 antagonistic mAb	NSCLC, NHL, HL, BCL, other advanced solid tumours	Pembrolizumab, chemo (oxaliplatin, irinotecan, leucovorin, 5-FU)	2
GSK / Immutep	GSK2831781	Anti-LAG-3 depleting mAb	Ulcerative colitis	-	2
Symphogen	Sym022	Anti-LAG-3 antagonistic mAb	Advanced solid tumours and lymphomas	Sym021 (anti-PD-1)	1
GSK	TSR-033	Anti-LAG-3 antagonistic mAb	Advanced solid tumours	Dostarlimab / TSR-042 (anti-PD-1)	1
Incyte / Agenus	INCAGN 02385	Anti-LAG-3 antagonistic mAb	Advanced solid tumours and lymphomas	-	1
Sanofi / Regeneron	REGN3767	Anti-LAG-3 antagonistic mAb	Advanced malignancies	Anti-PD-1	1
Merck KGaA / F-Star	FS118	Bispecific anti-(LAG-3 x PD-L1) mAb	Advanced malignancies	-	1
Macrogenics	MGD013	Bispecific anti-(LAG-3 x PD-L1) DART	Advanced solid and haematologic tumours	-	1
Xencor	XmAb22841	Bispecific anti-(LAG-3 x CTLA-4) mAb	Advanced solid tumours	-	1
Immutep	IMP761	Anti-LAG-3 agonistic mAb	Autoimmune disease	-	Precl.

Abbreviations: AA2A R: adenosine A2A receptor; (DL)BCL: (diffuse large) B cell lymphoma; HNSCC: head and neck squamous cell carcinoma; LAG-3: lymphocyte activation gene-3; MSI-H: microsatellite instability-high; NET: neuroendocrine tumour; (N)HL: (non)-Hodgkin lymphoma; (N)SCLC: (non)-small cell lung cancer; TNBC: triple negative breast cancer.
Source: Clinicaltrials.gov as of 12 March 2019, GSK FY2018 results presentation, goetzpartners Research

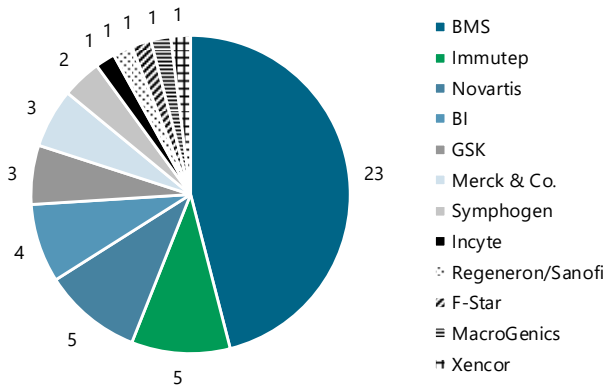
Oncology the primary therapy area of focus

All clinical-stage LAG-3 targeted compounds are in development for different types of advances cancers, predominantly solid tumours, although some innovators are also looking at select lymphomas. Since the focus in the immune-oncology space is to increase response rates to established ICIs, most trials – excluding those for bispecific molecules and a few others – test combination regimens with a marketed or developmental anti-PD-1 / L1 or anti-CLTA-4, or an established chemotherapy regimen.

BMS dominates in oncology with anti-LAG-3 relatlimab

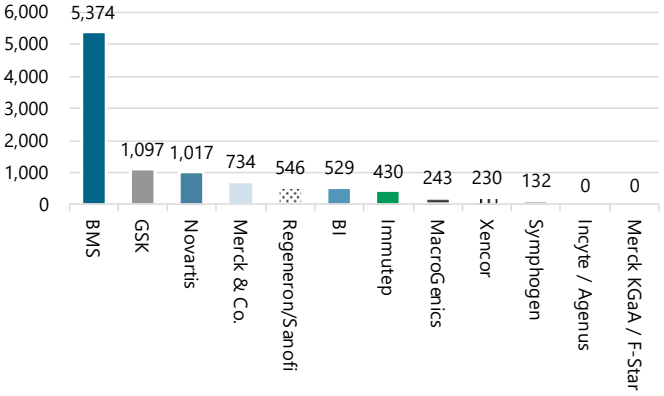
BMS is by far the most active company. Its anti-LAG-3 mAb relatlimab (BMS 986016) is currently undergoing around 23 trials (CHART 4) involving >5,300 patients (CHART 5), of which a few are expected to read out this year. The antibody is being tested predominantly with the company's marketed anti-PD-1 ICIs nivolumab (Opdivo) and ipilimumab (Yervoy) in all major solid tumours, but there are also a few trials testing combinations with chemotherapy (XELOX, FOLFOX and SOX) for gastric cancers and with anti-CD38 daratumumab for colorectal cancer.

CHART 4: BMS dominates with regard to number of ongoing trials…



Source: Clinicaltrials.gov as of 12 March 2019, goetzpartners Research

CHART 5: … and number of patients

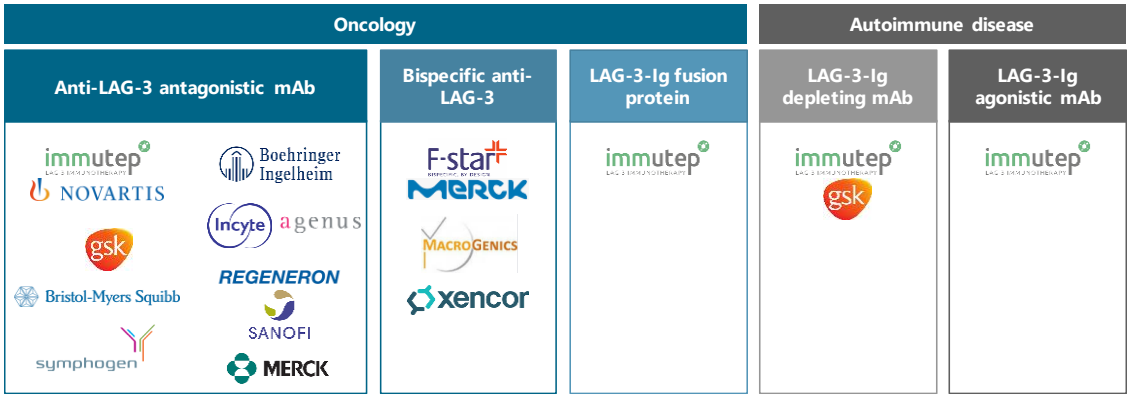


Source: Clinicaltrials.gov as of 12 March 2019, goetzpartners Research

Immutep has the broadest LAG-3 pipeline…

CHART 6 below shows that Immutep is the company with the broadest pipeline with respect to mechanisms of action.

CHART 6: Five LAG-3 targeted approaches are currently being pursued



Source: Company websites

...and leads the way in autoimmunity

To our knowledge, GSK2831781 (LAG-3 depleting mAb based on Immutep's IMP731) and IMP761 (LAG-3 agonistic mAb) are the only LAG-3 targeted product candidates being studied for autoimmune diseases. The only other molecule targeting an ICI in the context of autoimmune disease is AnaptysBio's CC-90006 (CHART 7). GSK2831781 is due to enter a Phase II trial in ulcerative colitis ("UC") this year. IMP761 is being prepared for first-in-human studies following positive preclinical data from a primate animal model that were recently presented at the ECCO medical meeting.

CHART 7: Immune checkpoint targeting agents in development for autoimmune diseases

Company	Molecule	Mechanism of action	Indication	Status
GSK / Immutep	GSK2831781	LAG-3 depleting mAb	Ulcerative colitis	Phase II start imminent, data in H2/2020E
Celgene / AnaptysBio	CC-90006	anti-PD-1 agonistic mAb	Psoriasis	Phase I recruiting, primary completion in Aug-2019
Immutep	IMP761	LAG-3 agonistic mAb	TBD	Cell line development for GMP manufacturing ongoing

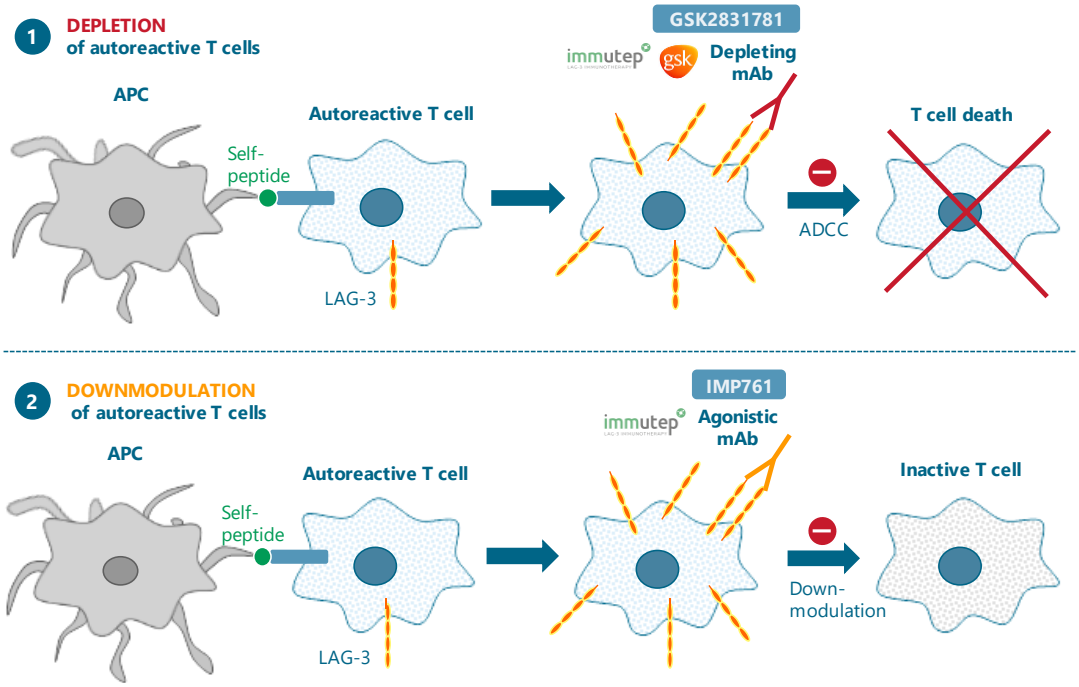
Source: Company websites, goetzpartners Research

Silencing autoreactive T cells rather than fighting symptoms

Most available treatments for autoimmune diseases focus on symptom alleviation by reducing the inflammation with corticoids and methotrexate as well as anti-TNF alpha, IL-6, IL-17 and IL-23 mAbs

Current approaches to treat autoimmune diseases focus on reducing the symptoms by removing pro-inflammatory mediators, most commonly TNF-α. Immutep's approach aims to tackle the root cause by silencing autoreactive T cells in two ways: (1) Depletion (IMP731 / GSK compound) or (2) downmodulation (CHART 8). It is not yet known what mechanism is best suited for which indication, and the approaches could be complementary with, individual use depending on e.g. disease severity or anatomical location. Depletion is an irreversible effect, while downmodulation is more subtle and can be fine-tuned. For example, GSK discovered that there is a high frequency of LAG-3+ T cells in the lamina propria within the colonic mucosa of patients who have active UC, and that the number of cells decrease in those responding to mAb therapy, prompting the company to pursue UC as lead indication.

CHART 8: Two distinct mechanisms of action to silence autoreactive T cells in autoimmune disease



Abbreviations: ADCC: antigen-dependent cellular cytotoxicity; APC: antigen-presenting cell.
Source: goetzpartners Research

LAG-3 is a marker for activated effector T cells and upregulated during inflammation

It has been established that LAG-3 is a marker for recently activated effector T cells, which are of major importance in many autoimmune diseases (Poirier *et al.* 2011). It is expressed by activated CD4+ and CD8+ T cells in inflamed secondary lymphoid organs or tissues and is upregulated strongly during inflammation. Deficiencies in the LAG-3 pathways have been linked to the development of autoimmune diseases (Angin *et al.* 2019). Finally, auto-immune memory T cells are chronically stimulated by the same self-peptide and acquire an exhausted phenotype, expressing LAG-3.

GSK2831781 due to start Phase II in ulcerative colitis

In January 2011, GSK and Immutep entered a licensing deal for LAG-3 antibodies worth up to £64m plus single-digit tiered royalties on sales. GSK is responsible for all development and commercialisation costs

GSK2831781 is a highly potent, humanised monoclonal antibody developed by GSK based on Immutep's IMP731 (itself derived from murine mAb A9H12) that was engineered to be afucosylated in order to have enhanced antibody-dependent cell cytotoxicity ("ADCC"). The effect is exerted through the depletion of recently activated T cells that accumulate at the diseased organ site by binding to LAG-3 on their cell surface. A first-in-human Phase I / Ib trial in Europe that enrolled both healthy volunteers and patients with plaque psoriasis was completed in March 2018, showing a clean safety profile and encouraging clinical improvements (Ellis *et al.* 2019). GSK also conducted compelling *in vitro* studies using colonic biopsy samples from UC patients, which provided further evidence suggesting that the depletion of LAG-3+ cells could be a promising therapeutic strategy in UC, paving the way for progression of GSK2831781 into Phase II. At its FY2018 results presentation, GSK confirmed proof-of-concept data in UC is expected in H2/2020E, suggesting a trial is due to start soon.

Selective depletion of pathogenic activated LAG-3+ CD4+ and CD8+ T lymphocytes

The rationale behind depleting LAG-3+ T cells is that this might lead to selective immunosuppression that would eliminate only pathogenic activated T cells specific for auto-antigens without affecting resting T cells necessary for the body to fight infections. Other potential targets include CD40 and CD25, although the former has been associated with thromboembolic complications due to the presence of CD40L on platelets (Mirabet *et al.* 2008), while targeting the latter could interfere with the development of immune regulation, as CD25 is also expressed on natural regulatory T cells (Benghiat *et al.* 2005).

Preclinical studies provide solid rationale for development in UC

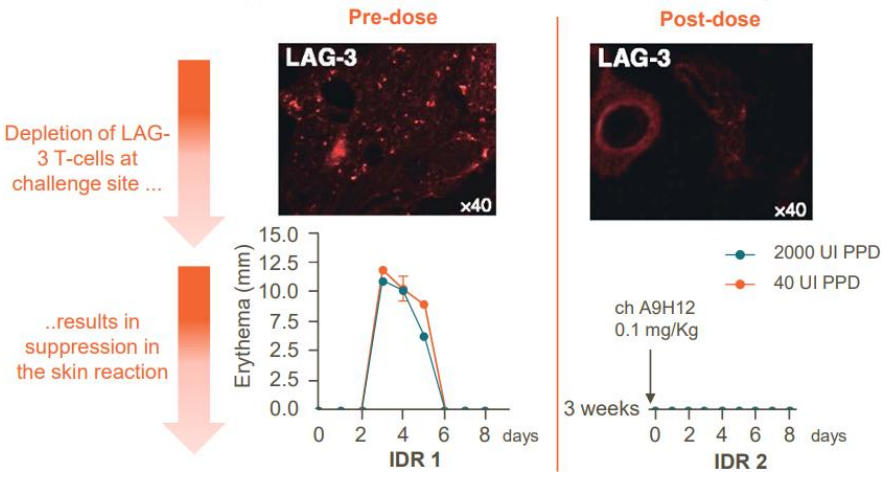
Both Immutep and partner GSK conducted a range of preclinical studies that together provide robust evidence supporting further development of GSK2831781 for autoimmune diseases, particularly inflammatory bowel disease ("IBD"), which includes both Crohn's disease ("CD") and UC. CHART 9 summarises three important studies, one of which was conducted in a non-human primate model and the other two using samples taken from the colon of patients with UC or healthy controls. The study in primates showed that the injection of a LAG-3 depleting mAb efficiently removed activated CD4+ and CD8+T cells via ADCC and as such prevented inflammation of the skin following sensitisation with the BCG vaccine, an effect that persisted long after the mAb was washed out. The two studies in humans demonstrated that there was a strong increase of LAG-3 expressing cells in the inflamed colon of UC patients, but not in the blood, and that this number fell sharply in those patients who responded to biological therapy.

CHART 9: Preclinical studies supporting development of GSK2831781 in autoimmune disease

Authors	Piorier *et al.* 2011	Slevin *et al.* 2018	Slevin *et al.* 2019
Biological model	Non-human primate (baboon). Delayed-type hypersensitivity ("DTH") model following sensitization with BCG vaccine	Human colonic biopsy samples from HC and UC pts	Human colonic biopsy samples from HC (n=9) and UC pts (n=42)
Key findings	One single dose of 1 or 0.1 mg/kg LAG-3 chimeric mAb (A9H12) given IV: ▪ Depleted LAG-3+ activated CD4+ and CD8+T cells in lymph nodes *in vivo* and by ADCC *in vitro* (CHART 10) ▪ Reduced T lymphocyte and macrophage infiltration into the skin ▪ Prevented skin inflammation in tuberculin-inducted DTH model (CHART 10) ▪ Elicited a long-lasting *in vivo* effect	▪ CD3+ LAG-3+ cells were significantly increased in the inflamed colon of UC pts vs. HC and uninflamed UC ▪ Expansion of LAG-3+ cells was only seen in inflamed bowel, but not blood ▪ The no. of LAG-3+ cells correlated with endoscopic and histological inflammation ▪ The no. of total mucosal LAG-3+ cells fell substantially in patients who responded to biological Tx	▪ The frequency of LAG-3+ cells in peripheral blood was negligible, regardless of disease activity ▪ In the lamina propria, the frequency of LAG-3+ T cells was markedly increased in active UC vs. uninflamed and HC ▪ LAG-3+ expression was enriched on effector memory and CD161+ T cells ▪ Mucosal LAG-3+ T cells demonstrated robust production of IFNγ and IL-17A (two pro-inflammatory cytokines) ▪ In pts receiving Tx for UC, the no. of LAG-3+ cells decreased in pts who responded to Tx, but not in non-responders
Conclusions	▪ The depletion of LAG-3+ effector cells has an inhibitory action on Th1-mediated immune responses into the skin after antigen challenge	▪ LAG-3 is selectively upregulated on colonic T cells from patients with active UC ▪ Mucosal expression correlated with inflammatory activity and normalises after successful biological Tx	▪ LAG-3 expression is not altered in circulating blood ▪ Mucosal LAG-3 expression is increased in inflammation & normalises after successful Tx ▪ In human IBD, LAG-3+ cells are mainly effector memory cells that produce INFγ and IL-17A

Abbreviations: ADCC: antibody-dependent cellular cytotoxicity; CDC: complement-dependent cytotoxicity; ECCO: European Crohn's and Colitis Organisation; HC: healthy controls; IBD: inflammatory bowel disease; pts: patients; Tx: therapy; UC: ulcerative colitis
Source: Poirier *et al.* (2011) *Clin Exp Immunol*, Slevin *et al.* (2018) ECCO meeting, Slevin *et al.* (2019) ECCO meeting

CHART 10: Targeted depletion of LAG-3+ T cells with GSK2831781 precursor suppresses immune reaction to tuberculin



Source: GSK presentation from 2015 based on data from Poirier *et al.* (2011) *Clin Exp Immunol*

Delayed-type hypersensitivity ("DTH") test is used to detect immune cell infiltration to the skin

Type IV or delayed-type hypersensitivity ("DTH") typically occurs at 24 – 48 hours after exposure to an antigen. It is a cell-mediated immunoreaction that involves activated T cells, macrophages, eosinophils and cytotoxic CD8+ T cells. It is known to occur in tissue damage during infections with slow-growing intracellular organisms such as *Mycobacterium tuberculosis*. The prototypical reaction is the tuberculin test, a sensitising agent that leads to reactions in the skin characterised by erythema (redness), cellular infiltration and vesicles in subjects previously immunised with the Bacillus Calmette–Guérin ("BCG") live vaccine (used to protect against tuberculosis), which leads to an immune response that includes a significant number of antigen-specific T cells. Tissue damage can be caused both through the secretion of inflammatory molecules by macrophages and eosinophils, but also directly by cytotoxic T cells.

Phase I trial in psoriasis shows safety and encouraging efficacy signals

The preclinical study in non-human primates showing that the depletion of LAG-3+ effector cells inhibited the immune response in the skin prompted GSK to conduct a first-in-human Phase I / Ib trial in psoriasis patients. The design of the trial, which was conducted in Europe (Germany, UK), is shown in (CHART 11). Part A replicated the study done in primates by Poirier *et al.*, while Part A tested safety and tolerability in patients with psoriasis.

CHART 11: GSK has completed a first-in-human Phase I trial for GSK2831781

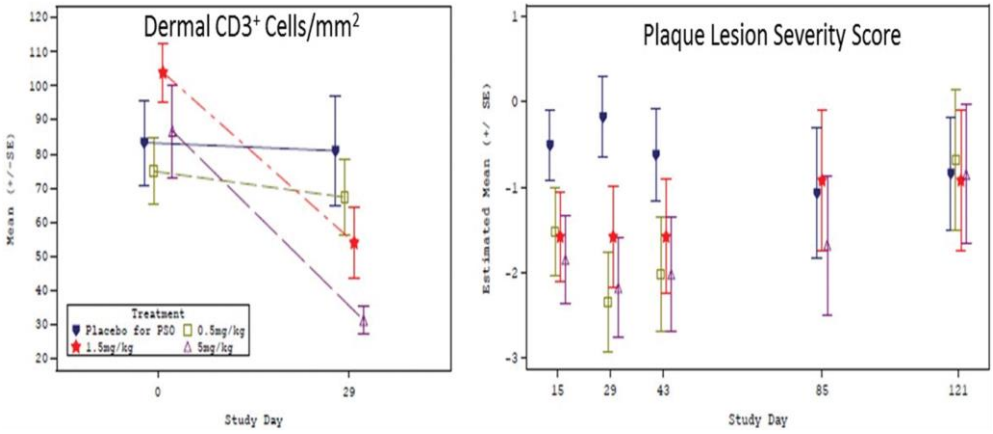
Indication	Phase	N	Design	Objectives	NCT / Data
Plaque psoriasis	I / Ib	67	Randomised, pcb-controlled, double-blind, single-ascending dose in 2 parts: ▪ **Part A | Delayed type hypersensitivity [DTH] cohorts (n=40):** safety, tolerability, PK, PD and immunogenicity administered to healthy subjects previously vaccinated with BCG ▪ **Part B (n=27):** 3 cohorts of 9 patients with mild-moderate plaque psoriasis randomised to GSK2831781 0.5, 1.5 or 5mg/kg or placebo (2:1)	▪ 1^{ary}: safety and tolerability ▪ 2^{ary}: PK, PD, immunogenicity, PASI, PLSS	NCT02195349 Started Jul-2014, 1^{ary} completion date Mar-2018

Abbreviations: BCG: Bacillus Calmette Guérin; FIH: first-in-human; PASI: psoriasis activity severity indices; PD: pharmacodynamics; PK: pharmacokinetics; PLSS: plaque lesion severity scores
Source: Company data, clinicaltrials.gov

Data presented at ECCO 2019 showed that the mAb was well tolerated with no safety concerns. Key findings include the following:

- Dose-dependent depletion of circulating LAG3+ memory T cells was observed for 6 - 8 weeks following a single 5mg/kg dose;
- Lag-3+ and CD3+ cells were reduced in psoriasis skin biopsies at 1.5 and 5mg/kg (CHART 12);
- Downregulation of pro-inflammatory mRNA transcripts including IL-17F, IFNγ and S100A12 as well as upregulation of those associated with epithelial integrity, e.g. CDHR1;
- Improvement of PASI and PLSS at all doses (CHART 12), with a percent change of -30.9%. from baseline PLSS mean for 5mg/kg at day 29.

CHART 12: A single injection of GSK2831781 reduces CD3+ cells in psoriasis skin biopsies (left) and improves PLSS scores (right)



Source: Ellis *et al.* (2019) *Journal of Crohn's and Cholitis*

Differentiated profile addressing large UC market opportunity

We believe that the reasons underlying GSK's decision to select UC as lead indication may include the following: (1) Limited number of approved mAbs for UC – to our knowledge four, as can be seen in CHART 13; (2) high unmet need given limited efficacy of approved treatments; (3) large size of the overall IBD market (UC and Crohn's combined); and potentially (4) more robust preclinical data in UC compared to other autoimmune indications that GSK may have investigated. Most approved mAbs for autoimmune diseases target (soluble) inflammatory markers, particularly TNF-α; pro-inflammatory interleukins ("IL") or their receptors; and integrins that are necessary for lymphocyte trafficking to the site of inflammation. In contrast, GSK2831781 aims to tackle the root cause of the disease by tagging pathogenic cells for destruction before they trigger inflammatory cascades.

CHART 13: Monoclonal antibodies approved for autoimmune conditions

Company	Product	mAb	Target	FDA appr.	UC	CD	PP	PA	AS	RA	MS	JIA	HS	GCA	UV	SLE
AbbVie	Humira	adalimumab	TNF-α	2002	✓	✓	✓	✓	✓	✓		✓	✓		✓	
J&J	Remicade	infliximab	TNF-α	1998	✓	✓		✓	✓	✓						
Takeda	Entyvio	vedolizumab	α4β7 integrin	2014	✓	✓								✓		
J&J	Simponi	golimumab	TNF-α	2009	✓			✓	✓	✓						
UCB	Cimzia	certolizumab	TNF-α	2008		✓	✓	✓	✓	✓						
Biogen	Tysabri	natalizumab	α4 integrin	2004		✓					✓					
J&J	Stelara	ustekinumab	IL-12 / IL-23	2009		✓	✓	✓								
Novartis	Cosentyx	secukinumab	IL-17A	2015			✓	✓	✓							
Bausch	Siliq[1]	brodalumab	IL-17 receptor	2017			✓	✓								
Eli Lilly	Taltz	ixekizumab	IL-17A	2016			✓									
J&J	Tremfya	guselkumab	IL-23	2017			✓									
Roche	Actemra	tocilizumab	IL-6 receptor	2010						✓		✓		✓		
Roche	Rituxan	rituximab	CD20	1997						✓						
Sanofi	Lemtrada	alemtuzumab	CD52	2014							✓					
Roche	Ocrevus	ocrelizumab	CD20	2017							✓					
GSK	Benlysta	belimumab	BlyS	2011												✓
Genentech	Raptiva[2]	efalizumab	CD11a	2003			✓									
Biogen	Zinbryta[3]	daclizumab	CD25	2016							✓					

(1) Kyntheum in Europe; (2) withdrawn in 2018 due to cases of serious brain inflammation; (3) withdrawn in 2009 due to cases of progressive multifocal leukoencephalopathy ("PML")
Abbreviations: AS: ankylosing spondylitis; CD: Crohn's disease; GCS: giant cell arteritis; HS: hidradenitis suppurative; JIA: juvenile idiopathic arthritis; MS: multiple sclerosis; PP: plaque psoriasis; RA: rheumatoid arthritis; SLE: systemic lupus erythematosus; UC: ulcerative colitis; UV: uveitis
Source: Product labels, goetzpartners Research

Selectivity is key

That this approach works can be exemplified with anti-CD20 mAb Ocrevus (ocrelizumab), approved for both relapsing ("RRMS") and primary progressive ("PPMS") multiple sclerosis. Its clinical effects are thought to involve immunomodulation through selective binding to CD20-expressing B cells, which results in ADCC and complement-mediated lysis, leading to their destruction. It is crucial that a mAb only targets pathogenic cells, leaving other cells needed for a normally functioning immune system intact. An example for what can happen when non-pathogenic cells are depleted is Biogen's Zinbryta (daclizumab). The anti-CD25 mAb was originally approved for MS, but then had to be withdrawn from the market two years after its approval due to cases of serious brain inflammation.

Most patients do not respond to biologic UC therapy and responses wane over time

IBD including UC and CD are chronic, progressive and disabling disorders characterised by lifelong treatment. For many decades, therapeutic options for IBD were limited to non-biological therapies, such as aminosalicylates, thiopurines and steroids, which provide symptomatic improvement, but do not alter the course of the disease. Advances in the understanding of the pathological mechanisms involved in IBD led to the development of new therapies, with the most important development being the introduction of TNF-α agents, i.e. infliximab, adalimumab and certolizumab pegol. More recently, the selective lymphocyte trafficking inhibitor vedolizumab was approved. These mAbs have reduced the need for surgery and improved the quality of life of patients and are therefore recommended by guidelines in moderate-to-severe IBD, particularly if non-biological therapy fails. However, these treatments are not effective in most patients, and even patients who initially respond to treatment lose their responses over time (CHART 14). In addition, the prolonged use of anti-TNF agents is associated with an increased risk of infection and malignancies.

CHART 14: Efficacy demonstrated by currently approved biologics for UC

Trial	Treatment	n	Remission Induction (week 8)	Delta	p-value	Sustained remission (week 6 or 8)	Delta	p-value
UC-I	Humira 160/180mg	130	18.5%	9.3%	<0.05	n.a.		
	Placebo	130	9.2%			n.a.		
UC-II	Humira 160/180mg	248	16.5%	7.2%	<0.05	8.5%	4.4%	<0.05
	Placebo	246	9.3%			4.1%		
UC I	Remicade 10mg/kg	121	32%		<0.01	26%		<0.001
	Placebo	121	15%			8%		
UC II	Remicade 10mg/kg	121	28%		<0.001	23%		<0.001
	Placebo	123	6%			2%		
UC I	Entyvio 300mg	225				17%	12%	0.001
	Placebo	149				5%		
UC I	Simponi 200/100mg	253				18%	11%	<0.0001
	Placebo	251				6%		

Source: Product labels

Multibillion market opportunity in UC

GSK2831781 could achieve peak sales in excess of $2.4bn in UC assuming only 5% market penetration and pricing in line with Takeda's IBD-specific biologic Entyvio (vedolizumab)

There were an estimated 11.2 million people worldwide living with IBD in 2015, with UC accounting for 54% – 57% of these (Vos *et al.* 2015). The prevalence of UC is estimated at 2.1 million in Europe (Burisch *et al.* 2013) and 907,000 in the US (Crohn's and Colitis Foundation of America). The annual cost of Takeda's Entyvio (vedolizumab) is approx. $23,000 in the US and €13,100 in Europe (assuming 7 doses of 300mg per year). Based on these figures, and assuming a peak penetration of only 5% of the total patient pool, yields a peak sales estimate of c.$2.4bn for GSK2831781. To put this value into perspective, we note that (1) GlobalData estimates the global UC market at $6.85bn in 2022E, of which we would assume that the US and Europe account for 80% - 90%, and (2) Entyvio – approved in both UC and CD since 2014 – generated sales of c.$1.83bn in FY2018. Under the agreement with GSK, Immutep would be entitled to single-digit tiered royalties on sales, which together with the outstanding milestone payments could significantly lift our valuation as we currently only include limited value related to milestones only.

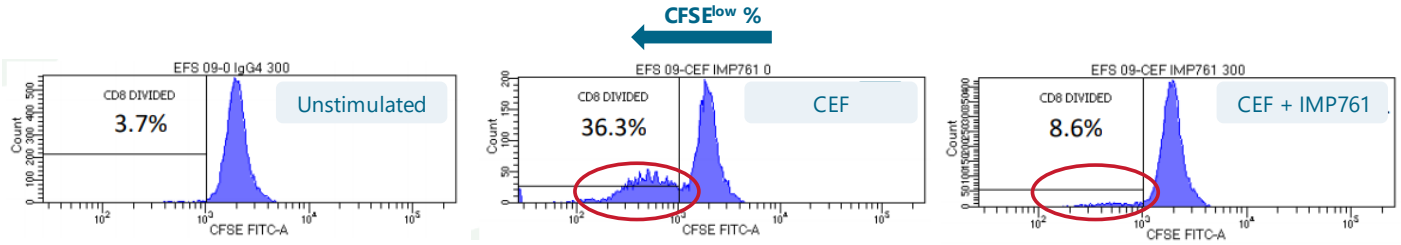
IMP761 MoA validated. First-in-human trial to start in 2020E

At a recent webcast, Immutep reviewed recent preclinical data obtained for IMP761 in a highly relevant primate model (cynomolgus monkeys) and provided an update on the compound's developmental status. Taken together, the preclinical work completed provides strong support for the proposed mechanism of action for the mAb, showing (1) inhibition of T cell proliferation *in vitro*, and (2) profound reduction in T cell infiltration *in vivo*. We understand that Immutep is currently working on the CHO cell line development for GMP manufacturing in order to start the first-in-human trial. The preclinical work is projected to take around 18 months and we therefore expect a Phase I trial to start in H2/2020E.

IMP761 inhibits CD8+ T cell proliferation *in vitro*…

An early experiment Immutep carried out to demonstrate that IMP761 can block T cell activation consisted in a standard T cell proliferation assay where T cells were stimulated with a CEF peptide pool derived from three viruses (cytomegalovirus ["CMV"], Epstein Barr virus ["EBV"], influenza). As shown in CHART 15 overleaf, IMP761 effectively inhibited the proliferation of CD8+ T cells (image on the right).

CHART 15: IMP761 inhibits peptide-induced T cell proliferation *in vitro*

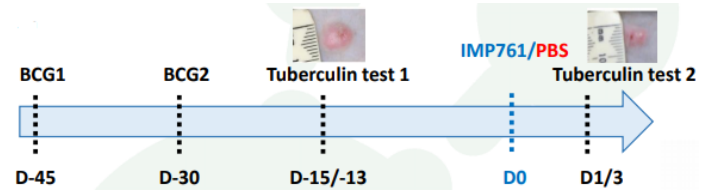


Source: Immutep webcast presentation, 26 March 2019

...and blocks inflammatory T cell infiltration *in vivo* in a primate animal model...

Since IMP761 is a humanised mAb that cross-reacts with other primate species but not rodents, Immutep conducted an experiment in cynomolgus monkeys. The design is shown in CHART 16. Briefly, the animals first received two doses of the BCG live vaccine (45 and 30 days prior to the administration of IMP761). The scientists then conducted the first skin test by sensitising with tuberculin to check for tissue damage. (The tuberculin test is commonly conducted in individuals who have been previously vaccinated with BCG to test if the vaccine worked, in which case T cell infiltration into the skin occurs causing a visible erythema.) After two weeks, the monkeys were injected with either IMP761 or PBS (Day 0). The second tuberculin test was then carried out 1 - 3 days later to investigate if the IMP761 injection had the expected effect, namely, prevention of T cell infiltration to the tissue, in which case the size of the erythema should be reduced.

CHART 16: Protocol and timeline of *in vivo* experiment



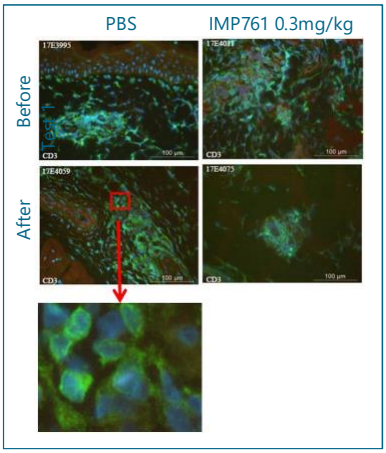
Source: Immutep webcast presentation, 26 March 2019

...as evidenced through a reduction in erythema and fewer T cells in the skin

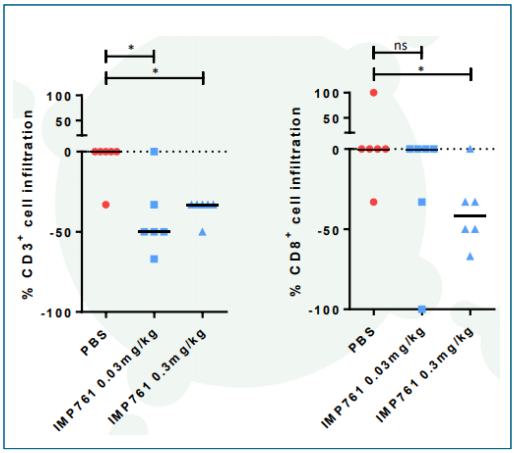
Those animals that received an injection of IMP761 after the first tuberculin test and before the second test demonstrated a significant reduction in T cell infiltration (CHART 17, left). This was reflected both in a smaller lesion on the skin where the test was conducted (right), as well as statistically significant reductions in the number of CD3+, CD4+ and CD8+ T cells (middle and right).

CHART 17: Injecting IMP761 (but not PBS) inhibits inflammatory T cell infiltration at the tuberculin test site

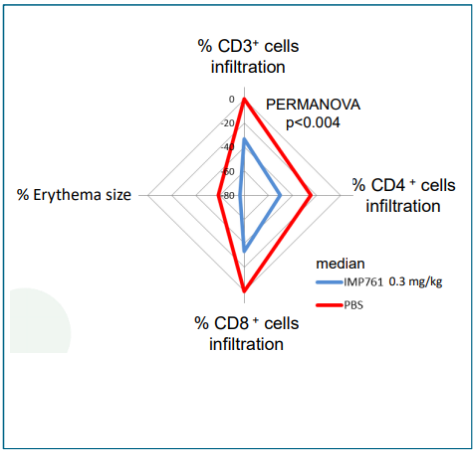
Reduction of T cell infiltration at the tissue site



Reduction of CD3+ (left) and CD8+ (right) T cell infiltration



Reduction of erythema size, as well as CD3+, CD4+ and CD8+ T cell infiltration



Source: Immutep webcast presentation, 26 March 2019

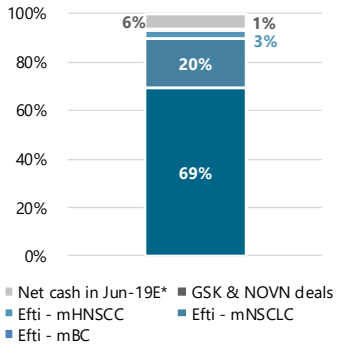
Lead indication yet to be determined

Immutep has not yet communicated which indication it intends to pursue for IMP761, and all options are therefore currently on the table. Based on the mAb's mechanism of action, there is a broad range of autoimmune indications that could be pursued. Ultimately, this decision may be taken by a potential licensing partner.

SoTP valuation largely based on eftilagimod

Lead asset eftilagimod alpha ("efti") accounts for >90% of our SOTP valuation (CHART 19), mainly because it is the most advanced asset with the largest body of data and fully owned by Immutep (excl. Chinese rights). Efti in mBC alone accounts for c.70% of the valuation. Net cash and revenue from the company's assets partnered with Novartis and GSK account for the remaining value. We do not currently include a value for IMP761 due to its early stage of development. We use a WACC of 12% to discount free cash flows.

CHART 18: Immutep SOTP valuation



Source: goetzpartners Research estimates

CHART 19: Immutep sum-of-the-parts valuation

			Peak sales		NPV		Adj. NPV	NPV/sh
Product	**Indications**	**Stage**	**($m)**	**Year**	**(A$m)**	**Prob.**	**(A$m)**	**(A$)**
Eftilagimod alpha	mBC	Phase IIb	820	2028E	459	40%	184	0.054
Eftilagimod alpha	mNSCLC	Phase II-ready	1,826	2035E	542	10%	54	0.016
Eftilagimod alpha	mHNSCC	Phase II-ready	326	2035E	84	10%	8	0.002
Novartis & GSK deals	Multiple	Phase I/II	n.a.	n.a.	8	25%	2	0.001
Net cash in Jun-19E*					16	100%	16	0.005
Fair value					**1,110**		**265**	**0.078**
Current Share Price (A$)								0.027
Upside								190%

Source: goetzpartners Research estimates.
Warning Note: Forecasts are not a reliable indicator of future results or performance. The return may increase or decrease as a result of currency fluctuations.

AIPAC Phase II data for efti in mBC the key catalyst

Efti, Immutep's lead asset, is a lymphocyte activation gene-3 ("LAG-3") Ig fusion protein that kick-starts the immune response by driving the maturation and activation of dendritic cells, the most powerful antigen-presenting cells ("APC") of the human immune system. It is currently being tested in multiple advanced solid tumours. The most advanced programme is the Phase IIb AIPAC trial in HR-positive, HER2-negative mBC which started in December 2015 (CHART 20). Immutep expects to report progression free survival ("PFS") data in Q4/2019E. As of mid-January 2019, 179 patients (80% of the total number of 226) had been recruited and the PFS read-out, will be based on 152 events.

Positive outcome would increase our TP to A$0.110

Efti accounts for > 90% of our TP. Positive PFS data from the AIPAC trial would increase our TP by 44% to A$0.110, based on increasing the probability of success in mBC to 65% (from 40%), and could form the basis of a conditional marketing approval. We forecast approval and launch in Europe in 2020E (one year later in the US) and peak sales of c.$820m in mBC alone. Our model assumes that Immutep signs an attractive licensing deal with a large pharma partner by YE2019E that includes a $50m upfront, up to $1bn in total milestones payments, and 15% - 21% royalties on sales.

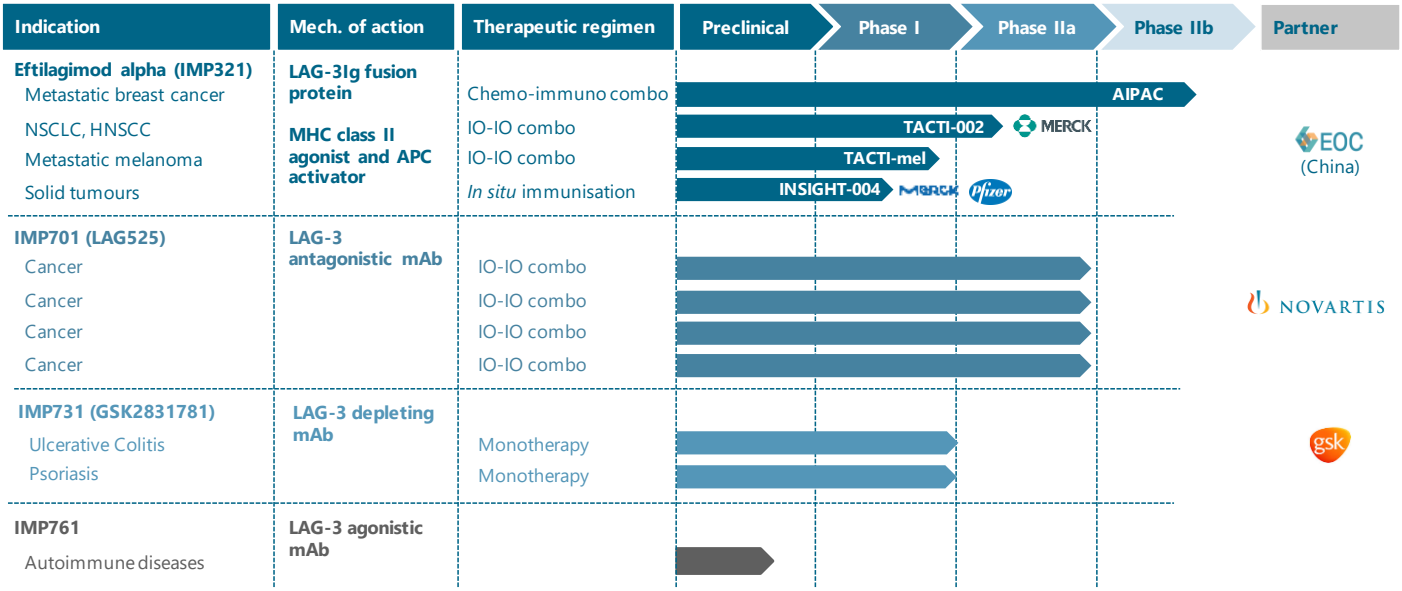
Safety run-in and previous Phase I / II trial showed encouraging efficacy signals

The safety run-in of the AIPAC trial (15 patients) was completed and the data presented at ASCO 2017, showing a partial response rate ("PR") of 47% and disease control rate ("DCR") of 87%. This is consistent with data from a previously conducted 30-patient Phase I / II trial with a similar design and dosing schedule as the AIPAC trial (Brignone *et al.* 2010), where the ORR was 50% and the DCR 90%.

Partnered pipeline moving forward

Immutep has collaborations with Novartis and GSK for its two early-stage assets (CHART 20).

CHART 20: Immutep has the broadest LAG-3 focused pipeline in the industry



Source: Company data, goetzpartners Research

Data from all clinical programs expected in 2019E

We look forward to multiple data points in the next 12 months (CHART 21), particularly (1) PFS data from the Phase IIb AIPAC study and (2) first data from the Phase II TACTI-002 study.

CHART 21: Immutep news flow for eftilagimod alpha

Event	Timing
Final data from ongoing TACTI-mel trial in melanoma	2019E
Start of patient recruitment for TACTI-002 Phase II trial HNSCC	H1/2019E
Start of patient recruitment for INSIGHT-004 Phase I trial in solid tumours	H1/2019E
AIPAC Phase IIb PFS data	Q4/2019E
TACTI-002 Phase II interim data	H2/2019E

Abbreviations: HNSCC: head and neck squamous-cell carcinoma; PFS: progression free survival
Source: Company data, goetzpartners Research estimates

Fully funded to mid-2020E

In December 2018, Immutep raised $5.2m in a private placement led by US specialist healthcare investor Altium Capital. Proceeds will be used to fund ongoing LAG-3 clinical development programmes including the AIPAC, TACTI-mel, TACTI-002 and INSIGHT studies as well as the preclinical development of IMP761. Meaningful clinical data is expected between now and mid-2020E by which time we expect Immutep to have partnered efti with a large pharma company.

Financial models

CHART 22: Immutep profit and loss model

Profit & Loss Statement	2016A	2017A	2018A	2019E	2020E	2021E	2022E	2023E	2024E	2025E	2026E
Jun YE (A$k except EPS)	30-Jun-16	30-Jun-17	30-Jun-18	30-Jun-19	30-Jun-20	30-Jun-21	30-Jun-22	30-Jun-23	30-Jun-24	30-Jun-25	30-Jun-26
Revenue	**2,029**	**4,222**	**7,353**	**8,055**	**48,163**	**19,080**	**26,066**	**37,926**	**57,551**	**225,013**	**245,914**
growth	(3%)	108%	74%	10%	498%	(60%)	37%	46%	52%	291%	9%
License income	175	-	2,630	3,675	43,711	14,552	21,413	33,078	52,467	219,362	236,341
% sales	9%	0%	36%	46%	91%	76%	82%	87%	91%	97%	96%
growth	4%	(100%)		40%	1090%	(67%)	47%	54%	59%	318%	8%
Other income	1,854	4,222	4,723	4,380	4,452	4,527	4,653	4,848	5,084	5,651	9,573
% sales	91%	100%	64%	54%	9%	24%	18%	13%	9%	3%	4%
growth	(4%)	128%	12%	(7%)	2%	2%	3%	4%	5%	11%	69%
R&D and intellectual property	(7,060)	(7,526)	(9,990)	(9,793)	(9,854)	(10,051)	(10,437)	(10,934)	(12,367)	(23,375)	(35,319)
% sales	348%	178%	136%	122%	20%	53%	40%	29%	21%	10%	14%
growth	(21%)	7%	33%	(2%)	1%	2%	4%	5%	13%	89%	51%
Corporate administrative expenses	(6,983)	(4,347)	(7,242)	(6,804)	(6,940)	(7,079)	(7,221)	(7,434)	(9,499)	(11,548)	(13,828)
% sales	344%	103%	98%	84%	14%	37%	28%	20%	17%	5%	6%
growth	22%	(38%)	67%	(6%)	2%	2%	2%	3%	28%	22%	20%
D&A expenses	(1,993)	(1,702)	(1,809)	(1,833)	(1,667)	(1,525)	(1,414)	(1,330)	(1,278)	(1,273)	(1,603)
% sales	98%	40%	25%	23%	3%	8%	5%	4%	2%	1%	1%
growth	49%	(15%)	6%	1%	(9%)	(8%)	(7%)	(6%)	(4%)	(0%)	26%
Other external expenses	(49,182)	(752)	(1,057)	(997)	(1,146)	(1,318)	(1,516)	(4,689)	(2,005)	(2,306)	17,678
% sales	2424%	18%	14%	12%	2%	7%	6%	12%	3%	1%	(7%)
growth	168%	(98%)	41%	(6%)	15%	15%	15%	209%	(57%)	15%	(867%)
Total costs & operating expenses	**(65,217)**	**(14,326)**	**(20,098)**	**(19,427)**	**(19,607)**	**(19,974)**	**(20,588)**	**(24,387)**	**(25,150)**	**(38,502)**	**(33,073)**
EBIT	**(63,188)**	**(10,105)**	**(12,744)**	**(11,372)**	**28,556**	**(894)**	**5,478**	**13,539**	**32,401**	**186,512**	**212,841**
Interest expenses	(8)	-	-	-	-	-	-	-	-	-	-
Profit/Loss before tax	**(63,196)**	**(10,105)**	**(12,744)**	**(11,372)**	**28,556**	**(894)**	**5,478**	**13,539**	**32,401**	**186,512**	**212,841**
growth	96%	(84%)	26%	(11%)	(351%)	(103%)	(713%)	147%	139%	476%	14%
% sales	(3115%)	(239%)	(173%)	(141%)	59%	(5%)	21%	36%	56%	83%	87%
Income tax	1,181	737	(2)	-	-	-	-	(1,354)	(6,480)	(55,954)	(63,852)
Tax rate	(2%)	(7%)	0%	0%	0%	0%	0%	10%	20%	30%	30%
Net income/loss	**(62,015)**	**(9,367)**	**(12,746)**	**(11,372)**	**28,556**	**(894)**	**5,478**	**12,185**	**25,921**	**130,558**	**148,989**
EPS calculation											
Earnings per Share (Basic)	**(0.031)**	**(0.004)**	**(0.005)**	**(0.003)**	**0.008**	**(0.000)**	**0.002**	**0.004**	**0.008**	**0.039**	**0.044**
growth	52%	(87%)	19%	(31%)	(351%)	(103%)	(713%)	122%	113%	404%	14%
Underlying EPS (Basic)	**(0.007)**	**(0.006)**	**(0.006)**	**(0.004)**	**0.008**	**(0.001)**	**0.001**	**0.004**	**0.007**	**0.038**	**0.036**
Earnings per Share (Diluted)	**(0.031)**	**(0.004)**	**(0.005)**	**(0.003)**	**0.008**	**(0.000)**	**0.002**	**0.004**	**0.008**	**0.039**	**0.044**
growth	52%	(87%)	19%	(31%)	(351%)	(103%)	(713%)	122%	113%	404%	14%
Underlying EPS (Diluted)	**(0.007)**	**(0.006)**	**(0.006)**	**(0.004)**	**0.008**	**(0.001)**	**0.001**	**0.004**	**0.007**	**0.038**	**0.036**
Number of Shares (basic)	2,016,566	2,284,361	2,608,328	3,383,598	3,383,598	3,383,598	3,383,598	3,383,598	3,383,598	3,383,598	3,383,598
Number of Shares (diluted)	2,016,566	2,284,361	2,608,328	3,383,598	3,383,598	3,383,598	3,383,598	3,383,598	3,383,598	3,383,598	3,383,598

Source: Company data, goetzpartners Research. Warning Note: Past performance and forecasts are not a reliable indicator of future results or performance. The return may increase or decrease as a result of currency fluctuations.

CHART 23: Immutep balance sheet model

Balance Sheet	2016A	2017A	2018E	2019E	2020E	2021E	2022E	2023E	2024E	2025E	2026E
Jun YE (A$k)	30-Jun-16	30-Jun-17	30-Jun-18	30-Jun-19	30-Jun-20	30-Jun-21	30-Jun-22	30-Jun-23	30-Jun-24	30-Jun-25	30-Jun-26
ASSETS											
CURRENT ASSETS	**21,671**	**15,919**	**28,643**	**91,332**	**114,787**	**108,406**	**108,141**	**111,305**	**130,690**	**251,191**	**372,140**
Cash and cash equivalents	20,880	12,237	23,476	85,661	108,838	102,338	101,951	104,992	124,250	244,622	365,440
GST receivable	74	187	171	174	178	181	185	189	192	196	200
Grant and other receivables	95	2,007	3,261	3,587	3,767	3,842	3,919	3,997	4,077	4,159	4,242
Other current assets	623	1,488	1,736	1,909	2,005	2,045	2,086	2,127	2,170	2,213	2,258
FIXED ASSETS	**20,883**	**19,045**	**18,356**	**16,696**	**15,282**	**14,157**	**13,290**	**12,757**	**12,688**	**16,140**	**19,701**
Tangible assets, net	32	24	26	27	29	37	50	71	108	307	522
Plant & Equipment	15	11	11	14	15	23	36	56	93	292	507
Computer	14	12	15	13	13	14	14	14	15	15	15
Furniture and fittings	3	1	0	0	-	-	-	-	-	-	-
Goodwill	110	110	110	110	110	110	110	110	110	110	110
Intangible assets, net	20,742	18,910	18,219	16,558	15,143	14,011	13,131	12,576	12,470	15,723	19,069
Patents	-	-	-	-	-	-	-	-	-	-	-
Intellectual property	20,742	18,910	18,219	16,558	15,143	14,011	13,131	12,576	12,470	15,723	19,069
TOTAL ASSETS	**42,554**	**34,964**	**46,999**	**108,028**	**130,069**	**122,563**	**121,431**	**124,062**	**143,377**	**267,331**	**391,841**
LIABILITIES											
CURRENT LIABILITIES	**1,472**	**2,632**	**3,853**	**10,825**	**11,008**	**11,095**	**11,182**	**11,272**	**11,364**	**11,457**	**11,552**
Trade payables	561	1,139	1,615	1,777	1,866	1,903	1,941	1,980	2,020	2,060	2,101
Borrowings	-	-	-	-	-	-	-	-	-	-	-
Current tax payable	22	-	-	-	-	-	-	-	-	-	-
Employee benefits	28	43	190	199	207	211	215	220	224	228	233
Other payables	862	1,450	2,048	2,151	2,237	2,282	2,327	2,374	2,421	2,470	2,519
Deferred revenue	-	-	-	6,699	6,699	6,699	6,699	6,699	6,699	6,699	6,699
NON-CURRENT LIABILITIES	**5,765**	**5,799**	**9,623**	**69,234**	**63,683**	**58,303**	**53,121**	**45,222**	**40,529**	**36,137**	**11,761**
Convertible note liability	5,027	5,779	6,646	7,643	8,789	10,107	11,624	13,367	15,372	17,678	-
Warrant liability	-	-	2,945	2,945	2,945	2,945	2,945	-	-	-	-
Employee benefits	43	20	32	33	34	34	35	36	36	37	38
Deferred tax liability	694	-	-	-	-	-	-	-	-	-	-
Deferred revenue, less of current portion	-	-	-	58,613	51,915	45,216	38,517	31,819	25,120	18,421	11,723
TOTAL LIABILITIES	**7,237**	**8,431**	**13,477**	**80,060**	**74,691**	**69,398**	**64,304**	**56,494**	**51,892**	**47,593**	**23,313**
EQUITY											
SHAREHOLDERS EQUITY	**35,318**	**26,532**	**33,522**	**27,968**	**55,378**	**53,166**	**57,127**	**67,569**	**91,485**	**219,737**	**368,528**
Contributed equity	194,531	195,353	213,233	219,051	217,905	216,586	215,070	213,327	211,322	209,016	208,818
Reserves	63,258	63,019	64,874	64,874	64,874	64,874	64,874	64,874	64,874	64,874	64,874
Accumulated losses	(222,472)	(231,839)	(244,585)	(255,957)	(227,401)	(228,295)	(222,817)	(210,632)	(184,711)	(54,153)	94,836
TOTAL LIABILITIES & SHAREHOLDERS' EQUITY	**42,554**	**34,964**	**46,999**	**108,028**	**130,069**	**122,563**	**121,431**	**124,062**	**143,377**	**267,331**	**391,841**
GEARING											
Gross debt	**5,027**	**5,779**	**9,591**	**10,588**	**11,734**	**13,053**	**14,569**	**13,367**	**15,372**	**17,678**	**-**
Total ST debt	-	-	-	-	-	-	-	-	-	-	-
Total LT debt	5,027	5,779	9,591	10,588	11,734	13,053	14,569	13,367	15,372	17,678	-
Cash and cash equivalents plus investments	**20,880**	**12,237**	**23,476**	**85,661**	**108,838**	**102,338**	**101,951**	**104,992**	**124,250**	**244,622**	**365,440**
Net debt/(cash)	**(15,852)**	**(6,458)**	**(13,884)**	**(75,073)**	**(97,103)**	**(89,285)**	**(87,382)**	**(91,625)**	**(108,878)**	**(226,944)**	**(365,440)**

Source: Company data, goetzpartners Research. Warning Note: Past performance and forecasts are not a reliable indicator of future results or performance. The return may increase or decrease as a result of currency fluctuations.

CHART 24: Immutep cash flow model

Cash Flow Statement	2016A	2017A	2018A	2019E	2020E	2021E	2022E	2023E	2024E	2025E	2026E
Jun YE (A$k)	30-Jun-16	30-Jun-17	30-Jun-18	30-Jun-19	30-Jun-20	30-Jun-21	30-Jun-22	30-Jun-23	30-Jun-24	30-Jun-25	30-Jun-26
OPERATING CASH FLOW											
Payments to suppliers and employees	(13,336)	(10,819)	(13,572)	47,489	(24,734)	(25,179)	(25,905)	(29,789)	(30,604)	(43,962)	(38,204)
License income	175	-	2,630	3,675	43,711	14,552	21,413	33,078	52,467	219,362	236,341
License fee received	-	-	-	-	-	-	-	-	-	-	-
Interest received	264	104	127	181	184	188	192	196	200	204	208
Tax received / paid	(2)	22	(2)	-	-	-	-	(1,354)	(6,480)	(55,954)	(63,852)
Miscellaneous income	703	800	1,004	1,059	1,112	1,168	1,226	1,287	1,352	1,419	1,490
Grant income	887	1,385	2,036	3,140	3,156	3,172	3,235	3,365	3,533	4,029	7,875
NET CASH USED IN OPERATING ACTIVITIES	**(11,310)**	**(8,507)**	**(7,777)**	**55,544**	**23,429**	**(6,099)**	**161**	**6,783**	**20,466**	**125,097**	**143,858**
CASH FLOW FROM INVESTING											
Payments for held-to-maturity investments	-	-	-	-	-	-	-	-	-	-	-
Proceeds from held-to-maturity investments	-	-	-	-	-	-	-	-	-	-	-
Payments for P&E and intangibles	(27)	(7)	(12)	(173)	(253)	(401)	(547)	(796)	(1,209)	(4,725)	(5,164)
Proceeds from disposal of P&E	130	-	-	-	-	-	-	-	-	-	-
Acquisitions, net of cash acquired	-	-	-	-	-	-	-	-	-	-	-
Net cash provided by investing activities	**103**	**(7)**	**(12)**	**(173)**	**(253)**	**(401)**	**(547)**	**(796)**	**(1,209)**	**(4,725)**	**(5,164)**
CASH FLOW FROM FINANCING											
Proceeds from issue of shares / options / warrants	13,761	0	19,724	7,250	-	-	-	-	-	-	-
Proceeds from borrowings	13,751	-	-	-	-	-	-	-	-	-	-
Repayment of borrowings	(1,508)	-	-	-	-	-	-	(2,945)	-	-	(17,876)
Transaction costs	(283)	(9)	(1,319)	(435)	-	-	-	-	-	-	-
Net cash provided by financing activities	**25,720**	**(9)**	**18,405**	**6,815**	**-**	**-**	**-**	**(2,945)**	**-**	**-**	**(17,876)**
Net change in cash and cash equivalents	14,513	(8,522)	10,616	62,186	23,177	(6,500)	(387)	3,041	19,258	120,372	120,818
Effect of exchange rate on cash and cash equivalents	(393)	(121)	623	-	-	-	-	-	-	-	-
Cash and cash equivalents, beginning of period	6,760	20,880	12,237	23,476	85,661	108,838	102,338	101,951	104,992	124,250	244,622
Cash and cash equivalents, end of period	**20,880**	**12,237**	**23,476**	**85,661**	**108,838**	**102,338**	**101,951**	**104,992**	**124,250**	**244,622**	**365,440**
Cash generation/(burn)	**(11,207)**	**(8,513)**	**(7,789)**	**55,370**	**23,177**	**(6,500)**	**(387)**	**5,986**	**19,258**	**120,372**	**138,694**

Source: Company data, goetzpartners Research. Warning Note: Past performance and forecasts are not a reliable indicator of future results or performance. The return may increase or decrease as a result of currency fluctuations.

Charts

COMPANY DESCRIPTION

Immutep (known as Prima BioMed until November 2017) is an Australian clinical-stage biotechnology company that develops immunotherapies for cancer and autoimmune diseases. Immutep is the global leader in the understanding of and in developing therapeutics that modulate Lymphocyte Activation Gene-3 ("LAG-3"). LAG-3 was discovered in 1990 at the Institut Gustave Roussy by Dr Frédéric Triebel, Immutep's Chief Scientific Officer and Chief Medical Officer. The company has three assets in clinical and one asset in preclinical development. The lead product candidate is eftilagimod alpha ("efti"), a first-in-class antigen presenting cell ("APC") activator being investigated in combination with chemotherapy or immune therapy for advanced breast cancer and melanoma. Immutep is dual-listed on the Australian Stock Exchange ("IMM") and on the NASDAQ Global Market ("IMMP") in the US (American Depository Receipts), and has operations in Europe, Australia, and the US. The company has licensing deals with Novartis, GSK and EOC (China only), and clinical trial collaboration and supply agreements with Merck & Co. and Merck KGaA / Pfizer, the latter for lead asset efti.

SCENARIOS

Base Case - GP Investment Case

Eftilagimod alpha completes the Phase IIb AIPAC trial in mBC in 2019, Immutep signs a $1bn licensing deal with a large pharma partner in H2/2019E, and efti receives conditional approval in 2020E in Europe. US launch follows one year later. Immutep has sufficient cash to fund operations until Q4/2019E. Revenue from the expected efti licensing deal means that Immutep does not need to raise further funds.

Bluesky Scenario

Immutep signs a more lucrative licensing deal for efti than the $1bn reflected in our forecasts, including a substantially larger upfront payment (we model $50m).

Downside risk

Efti fails to shows a benefit in the Phase IIb AIPAC trial. Conditional approval is not granted based on Phase IIb data. Immutep is unable to sign a licensing deal for efti by Q4/2019E.

Peer Group Analysis

SWOT

Strengths: Leader in the understanding of LAG-3; broadest LAG-3 focused pipeline; validation from large pharma partners (Novartis, GSK, Merck & Co.); funded for >12 months.

Weaknesses: One single asset (eftilagimod alpha) accounts for the lion share of value; efti has not demonstrated convincing efficacy in monotherapy settings; efti is protected mainly by use and formulation patents, as the composition of matter patent has already expired.

Opportunities: LAG-3 could become the third pillar in immune checkpoint therapy and efti is the most advanced LAG-3 focused asset; efti could be the first immuno-oncology drug to be approved for metastatic breast cancer; oncology drugs addressing high unmet needs often enjoy shorter development and approval timelines than therapeutics in other disease areas; significant M&A activity in the immuno-oncology space.

Threats: EMA and FDA raise the hurdles for immunotherapy drugs.

INDUSTRY EXPECTATIONS

Immutep is developing immunotherapies for cancer, with a focus on the immune checkpoint LAG-3. The immune checkpoint inhibitor ("ICI") class has experienced rapid adoption since the launch of BMS's Yervoy (ipilimumab) in 2011, owing to their ability to elicit durable responses in 20 - 50% of patients for up to 10 years. The global ICI market was worth $10.5bn in 2017 and is expected to nearly triple by 2022E, driven largely by expanding use of existing therapies both in approved and new indications. The race is on to develop novel compounds with complementary mechanisms of action for combination therapy able to augment response rate without increasing toxicity, which, if successful, are expected to enjoy rapid uptake.

Important Disclosures: Non-Independent Research

Analyst Certification

I, Brigitte de Lima, PhD, CFA, hereby certify that the views regarding the companies and their securities expressed in this research report are accurate and are truly held. I have not received and will not receive direct or indirect compensation in exchange for expressing specific recommendations or views in this research report.

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- (NOVARTIS AG (NOVN SW))
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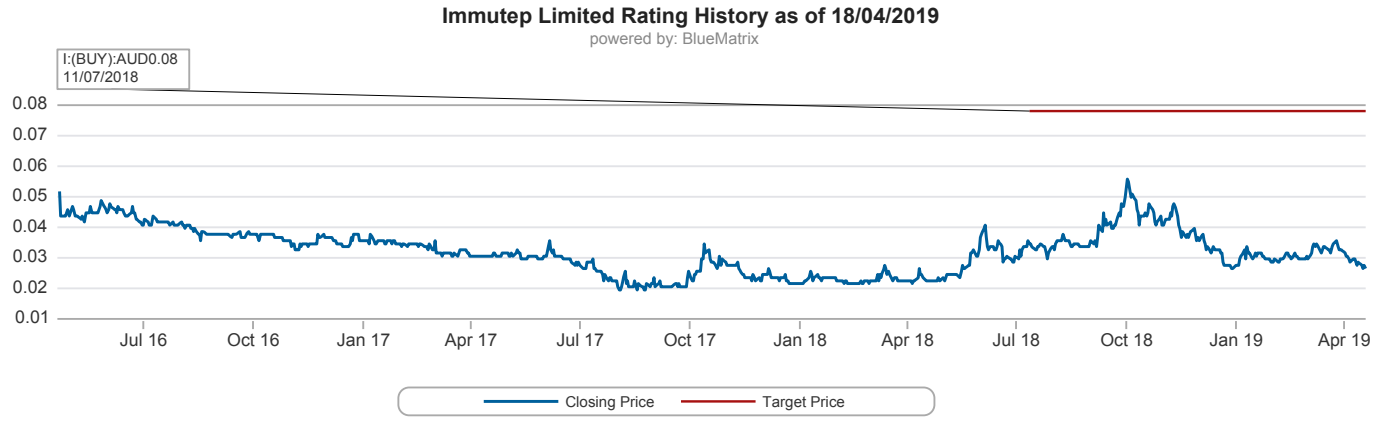

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