WILSONS ADVISORY

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Theme Detailed Analysis

Sector Healthcare

Company

Immutep Limited (IMM)

IMP761 | Can I&I success fund I-O upside?

We maintain our OVERWEIGHT rating with revised risked PT of $1.20/share for Immutep (IMM). Recent updates from INSIGHT-003 and TACTI-003 highlight the potential opportunity of Efti in oncology. In this report we shine a light on another facet of LAG-3-directed drug development. Immutep has been quietly working away on the development of IMP761: a first-in-class LAG-3 agonist for the treatment of autoimmune diseases. IMP761 differs from the checkpoint inhibitor programs previously developed in oncology, wherein those programs promote T cell activity, IMP761 has the potential to suppress the immune response. Initial indications are yet to be announced, here we theorise development in rheumatoid arthritis and inflammatory bowel disease; both with blockbuster potential. A Phase I in healthy volunteers is underway with an KLH challenge serving as an initial proof of mechanism. Un-risked price target upgraded 77% to $11.63/share.

Recommendation	**OVERWEIGHT**
12-mth target price (AUD)	$1.20
Share price @ 14-Jul-25 (AUD)	$0.24
Forecast 12-mth capital return	400.7%
Forecast 12-mth dividend yield	0.0%
12-mth total shareholder return	**400.7%**

Market cap ($m)	348.6
Enterprise value ($m)	166.8
Shares on issue (m)	1,453
Sold short (%)	1.9
ASX All Ords weight (%)	0.0
Median turnover/day ($m)	0.5

Key Points

Introducing IMP761 and Immutep's prospects in autoimmune disease. IMP761 is a first-in-class, LAG-3 agonist antibody, with immunosuppressive properties and the potential to address a range of inflammatory and autoimmune indications. T cell driven autoimmune diseases are an attractive space having high unmet needs, as chronic, progressive indications with large addressable markets. Currently approved immunosuppressive biologics often have black box warnings due to risk of serious infection and malignancy. Recent Phase I LAG-3 depleting antibody is suggestive of a clean safety profile and offers a clear point of differentiation.

Why now? Immutep's IMP761 agent is approaching 'proof of mechanism' status at a time when Pharma is clearly engaged with the idea of 'checkpoint agonists' as a therapeutic strategy. What sets IMP761 apart (as a LAG-3 agonist) is the direct suppression of $CD4^+$ and $CD8^+$ T cells in conjunction with supporting the suppressive action and commitment of regulatory T cells (Tregs). In this sense, LAG-3 is a near perfect target to address a range of autoimmune indications. We see immense potential deal value in that an out-licensing transaction could attract substantial upfronts, milestones and royalty income.

Un-risked program valuation for IMP761 is A$5.9B. We have recognised '761's multi-indication potential by using Monte Carlo simulation. Guided by a basket of 20 approved biologics for autoimmune diseases (peak sales ranging from US$500M to US$28B) we ran 10,000 option pricing simulations using randomised future values (FVs) and levels of economic return (ER – effectively Immutep's overall share of out-licenced IMP761 indications). The median valuation was US$60M; adding A$0.06 to our risked IMM PT (increased 14% to A$1.20/share). Un-risked valuation of IMP761 is A$3.62 per share or 31% of our revised un-risked PT of $11.63 (77% upgrade).

Forecasts. We have increased near term R&D expense assumptions having reviewed program assumptions: a) TACTI-004 investment (1L NSCLC); b) bringing forward Phase II/III (potentially registrational) for 1LHNSCC in CPS < 1; and c) adding resources for Phase II IMP761 development. We estimate $125M cash and equivalents by the end of FY25e.

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12-mth price performance ($)

	1-mth	6-mth	12-mth
Abs return (%)	(5.9)	(25.0)	(32.4)
Rel return (%)	(6.4)	(28.0)	(37.1)

Financial summary (Y/E Jun, AUD)	FY23A	FY24A	FY25E	FY26E	FY27E
Sales ($m)	0.0	0.0	0.0	0.0	0.0
EBITDA norm ($m)	(38.8)	(44.2)	(62.2)	(81.0)	(62.0)
Consensus EBITDA ($m)			(59.6)	(78.1)	(46.5)
EPS norm (cents)	(4.5)	(3.6)	(4.2)	(5.7)	(4.4)

Key changes		6-May	After	Var %
Sales	FY25E		0.0	
($m)	FY26E	0.0	0.0	0%
	FY27E	0.0	0.0	0%
EBITDA	FY25E	(50.9)	(62.2)	-22%
norm	FY26E	(45.0)	(81.0)	-80%
($m)	FY27E	(35.0)	(62.0)	-77%
Price target		1.05	1.20	14%
Rating		O/W	O/W	

Source: Company data, Wilsons Advisory estimate, Refinitiv, IRESS.
All amounts are in Australian Dollar (A$) unless otherwise stated.

Business Description

Immutep (IMM:ASX) is a clinical stage Australian biopharma operating in the immuno-oncology (IO) sector with their portfolio of LAG-3 directed biologics. Immutep have four assets under development, all with strong IP protection; two of which are out-licensed (LAG525 - Novartis, IMP731 - GSK) with attached milestone and royalty revenue optionality, with the remaining two (Efti and IMP761) being developed in-house for a range of oncology (incl. HNSCC, NSCLC, mBC) and autoimmune indications.

Catalysts

a) achievement of clinical trial endpoints; b) partnership opportunities; c) regulatory approvals (including IND approvals); d) corporate activity.

Investment Thesis

We maintain our OVERWEIGHT rating with revised risked PT of $1.20/share for Immutep. In this report we review IMP761: a LAG-3 agonist with immense potential in treating autoimmune diseases. Initial indications are yet to be announced, here we theorise development in rheumatoid arthritis and inflammatory bowel disease; both with blockbuster opportunity. Un-risked price target upgraded 77% to $11.63/share.

Risks

a) adverse clinical trial outcomes; b) negative regulator interactions; c) competitive intensity of immuno-oncology field; d) available capital.

P&L ($m)	FY23A	FY24A	FY25E	FY26E	FY27E
Sales	0.0	0.0	0.0	0.0	0.0
EBITDA norm	(38.8)	(44.2)	(62.2)	(81.0)	(62.0)
EBIT norm	(40.8)	(46.4)	(64.9)	(84.0)	(65.3)
PBT norm	(39.9)	(42.7)	(61.0)	(82.9)	(64.1)
NPAT norm	(39.9)	(42.7)	(61.0)	(82.9)	(64.1)
NPAT reported	(39.9)	(43.5)	(61.0)	(82.9)	(64.1)
EPS norm (cents)	(4.5)	(3.6)	(4.2)	(5.7)	(4.4)
DPS (cents)	0.0	0.0	0.0	0.0	0.0

Growth (%)	FY23A	FY24A	FY25E	FY26E	FY27E
Sales	n/m	n/m	n/m	n/m	n/m
EBITDA norm	18.6	14.0	40.8	30.2	(23.5)
NPAT norm	15.3	7.1	42.8	35.9	(22.7)
EPS norm (cents)	9.8	(20.4)	18.0	35.9	(22.7)
DPS (cents)	n/m	n/m	n/m	n/m	n/m

Margins and returns (%)	FY23A	FY24A	FY25E	FY26E	FY27E

Interims ($m)	1H24A	2H24A	1H25A	2H25E	1H26E
Sales	0.0	0.0	0.0	0.0	0.0
EBITDA norm	(22.2)	(22.0)	(24.2)	(38.0)	(33.0)
EBIT norm	(23.2)	(23.2)	(25.5)	(39.4)	(34.5)
PBT norm	(21.2)	(21.5)	(22.4)	(38.6)	(33.8)
NPAT norm	(21.2)	(21.5)	(22.4)	(38.6)	(33.8)
NPAT reported	(21.2)	(22.3)	(22.4)	(38.6)	(33.8)
EPS norm (cents)	(1.8)	(1.8)	(1.5)	(2.7)	(2.3)
DPS (cents)	0.0	0.0	0.0	0.0	0.0

Stock specific	FY23A	FY24A	FY25E	FY26E	FY27E
R&D expense (m)	(34.4)	(39.6)	(65.3)	(60.0)	(45.0)
Licensing revenue (m)	0.0	0.0	0.0	0.0	0.0

Balance sheet ($m)	FY23A	FY24A	FY25E	FY26E	FY27E
Cash & equivalents	123.4	181.9	125.2	135.6	72.1
Current receivables	8.0	7.4	5.0	5.0	5.0
Current inventory	0.0	0.0	0.0	0.0	0.0
PPE	0.1	0.1	0.1	0.1	0.1
Intangibles	9.5	8.2	8.2	8.2	8.2
Other assets	6.5	4.0	6.7	5.2	3.7
Total assets	147.4	201.6	145.2	154.1	89.1
Current payables	9.0	9.6	10.0	11.0	11.0
Total debt	0.0	0.0	0.0	0.0	0.0
Other liabilities	1.8	2.3	2.1	3.5	2.6
Total liabilities	11.0	12.1	12.4	14.7	13.8
Minorities	0.0	0.0	0.0	0.0	0.0
Shareholders equity	136.5	189.5	132.8	139.4	75.3

Cash flow ($m)	FY23A	FY24A	FY25E	FY26E	FY27E
Operating cash flow	(35.4)	(34.8)	(62.5)	(83.3)	(63.2)
Maintenance capex	(0.1)	(0.0)	(0.0)	(0.0)	(0.0)
Free cash flow	(35.4)	(34.9)	(62.5)	(83.3)	(63.2)
Growth capex	0.0	0.0	0.0	0.0	0.0
Acquisitions/disposals	0.0	0.0	0.0	0.0	0.0
Dividends paid	0.0	0.0	0.0	0.0	0.0
Other cash flow	(1.2)	(7.0)	(63.5)	(6.3)	(0.3)
Cash flow pre-financing	(36.6)	(41.8)	(126.0)	(89.6)	(63.5)
Funded by equity	80.1	100.3	0.0	100.0	0.0
Funded by cash/debt	(123.5)	(158.7)	126.0	(110.4)	63.5

Liquidity	FY23A	FY24A	FY25E	FY26E	FY27E
Cash conversion (%)	93.6	87.2	106.7	104.2	103.9
Net debt ($m)	(123.4)	(181.9)	(125.2)	(135.6)	(72.1)
Net debt / EBITDA (x)	3.2	4.1	2.0	1.7	1.2
ND / ND + Equity (%)	(945.6)	n/m	n/m	n/m	n/m
EBIT / Interest expense (x)	44.4	12.5	16.6	77.3	54.9

Valuation	FY23A	FY24A	FY25E	FY26E	FY27E
EV / Sales (x)	n/m	n/m	n/m	n/m	n/m
EV / EBITDA (x)	n/m	n/m	n/m	n/m	n/m
EV / EBIT (x)	n/m	n/m	n/m	n/m	n/m
P / E (x)	n/m	n/m	n/m	n/m	n/m
P / BV (x)	2.1	1.5			
FCF yield (%)	(12.4)	(12.2)			
Dividend yield (%)	0.0	0.0	0.0	0.0	0.0
Payout ratio (%)	0.0	0.0	0.0	0.0	0.0
Weighted shares (m)	892.5	1,200	1,453	1,453	1,453

Source: Company data, Wilsons Advisory estimate, Refinitiv, IRESS.
All amounts are in Australian Dollar (A$) unless otherwise stated.

Immutep | Thinking expansively on valuation

New PT $1.20/share | Un-risked: $11.63/share

In **Figure 1** below, our familiar, risked/un-risked sum-of-parts valuation table for Immutep is updated. We have made two important changes: a) restructuring the 'Efti – HNSCC' component, to reflect independent (rather than out-licensed) clinical development (and perhaps commercialisation) in the 1L, CPS < 1 population (pre-empted by this piece of prior research); and b) **adding IMP761** (see detailed valuation treatment which follows). Our new sum-of-parts PT is $1.20 per share (+14%). New un-risked PT is up 77% to $11.63/share.

Figure 1: Sum-of-parts valuation summary for IMM on both risked and un-risked bases

Valuation (SOTP)	Risked valuation (A$m)	Comments / methodology	Un-risked valuation (A$m)
Efti mBC	352	Real options valuation for EU and US market	1,268
Efti HNSCC	168	Real options valuation for EU and US market	2,502
Efti NSCLC	1,329	Real options valuation for EU and US market	9,203
IMP761	94	Real options valuation with Monte Carlo simulation	5,859
Enterprise value ($M)	1,943		18,832
Net cash (end-FY25e) ($M)			
Equity value ($M)	1,943		18,832
SOI (fully diluted)	1,619		1,619
Risked SOTP PT (A$/share)	**1.20**	**Unrisked SOTP PT (A$/share)**	**11.63**

Source: Wilsons Advisory.

Factors impacting the stock and thoughts on ameliorating catalysts

Largest risked/un-risked valuation 'gulf' in our coverage. Why so? The nearly 10-fold difference between risked and un-risked valuations seems at odds with Efti's advanced clinical stage (Phase III, II) and data quality. In recent years, the stock has struggled to achieve sustainable re-ratings, notwithstanding having delivered a stream of industry-leading efficacy outcomes across multiple indications. That price behaviour may reflect a lack of hard, monetary validation for their assets, in the form of partnering outcomes. Although MSD has committed A$100M to the TACTI-004 program in the form of KEYTRUDA supply, that form and amount of 'deal consideration' could be seen as less 'validating' and possibly even low (compared to other 'Phase II/III deals' seen across oncology); especially given the quality of supportive data (e.g. TACTI-002 and INSIGHT-003). As ~70% of the Immutep valuation hinges on TACTI-004 (and MSD's 'will they or won't they' M&A option in relation to Efti-NSCLC) the wait for progress milestones can weigh on share price performance. TACTI-004's futility analysis (expected between 4Q25 and 1Q26) is the next seismic catalyst (and potential 'MSD deal' trigger). Once that go/no-go milestone is passed, the trial's risk profile eases markedly, setting up a potential efficacy signal a year later. Stocks tend to rally hard into such events. We expect similar performance ahead of TACTI-004 readouts. The trial is elegantly designed with dual primary endpoints (PFS followed by OS, only one of which needs to 'hit' with statistical significance. It also offers a layered set of subordinate, registrable outcomes for pre-specified subgroups; subjects stratified by PD-L1 expression status (TPS), histology (squamous / non-squamous), geographic region and performance status (ECOG 0/1).

Capital overhang an unintended side effect of TACTI-003's knockout success. Immutep's financial guidance prioritises TACTI-004 and does not provide for a full-blown Phase II/III trial campaign in 1L HNSCC. The Efti-KEYTRUDA combination is eminently developable in the CPS <1 setting, supported by TACTI-003-B. Immutep is likely to emerge from its meeting with FDA later this year with a clear (if unfunded) development option. With nothing close to being competitive in that setting (WILSe Efti peak sales US$450M for CPS <1) it would be hard to stand by and watch that option not be exercised in a timely fashion. That said, Immutep is standing by its guidance, keen to explore the 'partnered development' option(s) it has first. As a reminder, Immutep has full freedom to operate in the HNSCC indication, so the scope of potential partners extends well beyond its TACTI-003 collaborator, MSD. As stunning as the TACTI-003-B results seem, we're not completely convinced that MSD will opt in. MSD has had mixed results in HNSCC. They failed to secure a monotherapy label for KEYTRUDA (with KEYNOTE-048). The 1L label they eventually received (and only in combination with the EXTREME chemotherapy regimen) is already servicing up to 60-80% of patients, as the indication is limited to CPS ≥ 1. In that sense, our US$450M peak sales estimate for CPS <1 may not be enough upside, in the context of KEYTRUDA's US$25B franchise, which stares down biosimilar substitution from 2028, and any number of potential challengers (e.g. the VEGF/PD-1 and EGFR/LGR5 bispecifics and a galaxy of novel modalities coming through oncology R&D pipelines).

An IMP761 deal to the rescue? Why did we decide to devote all this attention to IMP761, a Phase I asset the market knows almost nothing about? Because it carries immense short term (i.e. next 12-18 month) transaction potential. One of the things that impressed us most about Immutep three years ago when we were going through our diligence phase was the close attention and significant capital being investing behind the scenes in manufacturing development for IMP761. Early-stage assets need to generate intriguing data to partner; but to partner on astounding financial metrics, they need this infrastructure (e.g. cell lines, media development, expression systems, drug product characterisation) essentially as close to CMC-ready, launch-enabling as possible. The next section of this report is going to lay out an un-risked valuation of nearly $6B for IMP761. With Immutep close to 'proof-of-mechanism' and Phase II indication selection this year, ***IMP761 could transact (e.g. out-license) for multiples of what a Phase II/III HNSCC Efti trial might cost***. Another reason we like IMP761 is the Immunology and Inflammation (I&I) field it's aimed at. Although I&I is second to 'oncology' as a field that supports high-value deals, it is more homogeneous in many ways. The major players (e.g. Janssen, AbbVie, Sanofi) all want the same thing: a piece of druggable biology that they can own exclusively and exploit across a dozen indications. The chances of running a clean, competitive process (a phrase which here means auction) for IMP761 are therefore greater, too.

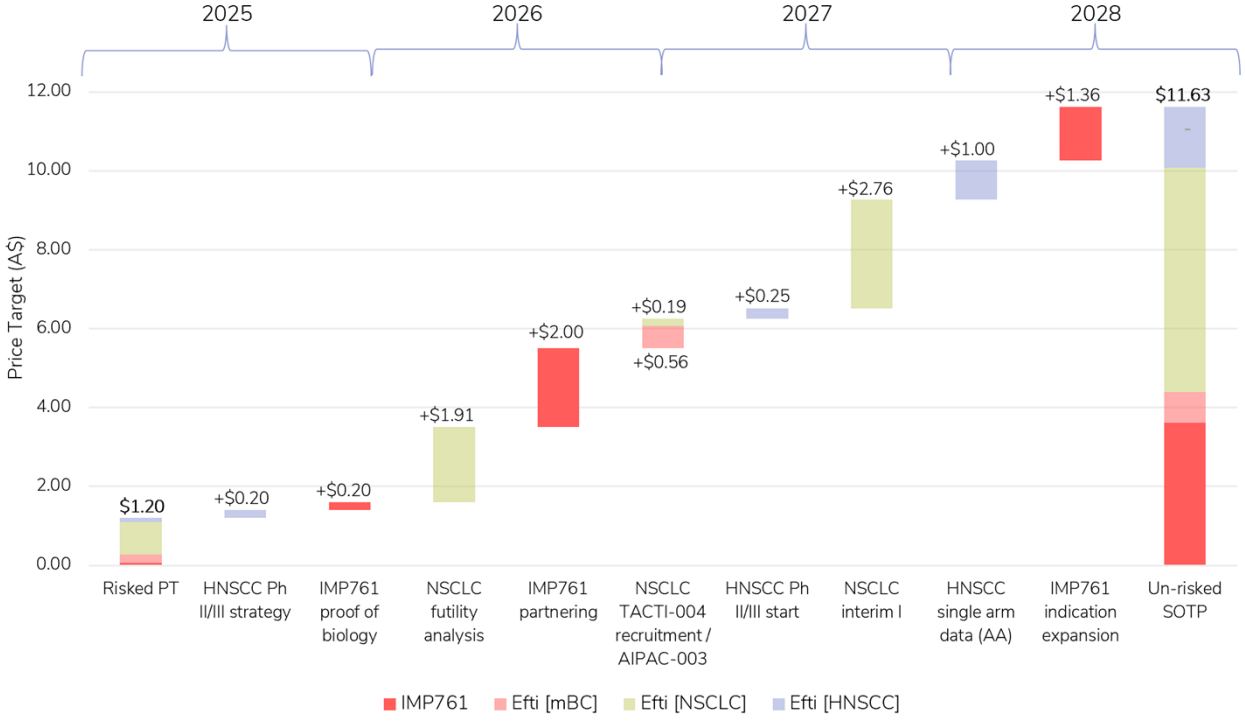
Meditating further on catalysts. What will we know, when … and how can it help valuation? In **Figure 2** we parse out the catalysts we see over the next three years. We have also tried to give an indication (by asset) of both indicative timing and valuation impact for each. Isolating the 'top three' over FY26-27e:

- **TACTI-004 futility analysis | +$1.91/share | 1Q-2026**. This pivotal go/no-go decision point is potentially assessable between 4Q25 and 1Q26 (calendar) based on objective response rates (ORR, a surrogate endpoint) for 150-200 evaluable patients.

- **IMP761 partnering (highly speculative) | +$2.00/share | 2026 (our estimate)**. Immutep's Phase I single and multiple ascending dose work is looking for a dose that maximises LAG-3-mediated T cell inhibition; but minimises the two countervailing forces of infection risk (from immune suppression) and the generation of anti-IMP761 immune responses. The first would be a safety red flag. The second may limit the potential for repeat dosing. After Phase I, the natural next step could be a 'basket' Phase II study testing at one or more doses in an array of (small n) indications. The ultimate partner for IMP761 would be one that can conduct multiple Phase II indication assessments in parallel.

- **TACTI-004 interim analysis | +$2.76/share | 2027**. Assuming full TACTI-004 recruitment by 3Q26 it seems likely that the first efficacy interim can become available between 4Q26 and 1Q27. Our understanding is that this analysis could support an early registration in the case of overwhelming early efficacy. More broadly, and assuming the trial goes full term, the primary endpoint design allows two opportunities for a full approval in progression free survival (PFS) and overall survival (OS) a year later.

Figure 2: Valuation development by catalyst and asset (FY25-28e)



Source: Wilsons Advisory.

IMP761 valuation: real options with Monte Carlo simulation

Super-optionality available in autoimmunity. This area of medicine offers opportunities to build vast product franchises because so many large indications share a common, underlying pathophysiology. **Figure 3** shows a selection of products from the sector's history, illustrating that 'peak sales' is often a large multiple of what a newly approved agent achieves in its first five years. As an example, AbbVie's IL-23 antagonist SKYRIZI is expected to emulate or even surpass the multi-indication success of past star, HUMIRA. Equally, Argenx's FcRn inhibitor VYVGART is expected to drive into a dozen or more autoimmune indications that have a strong autoantibody component, giving a peak sales estimate of US$13.6B. We assess a similar opportunity for IMP761 if LAG-3-agonism can suppress autoreactive T cells across multiple indications.

Figure 3: Actual or forecast peak sales for a basket of approved autoimmune agents. Indication expansion can drive immense upside. Median peak sales to 5-year sales ratio is 5.4x for I&I assets

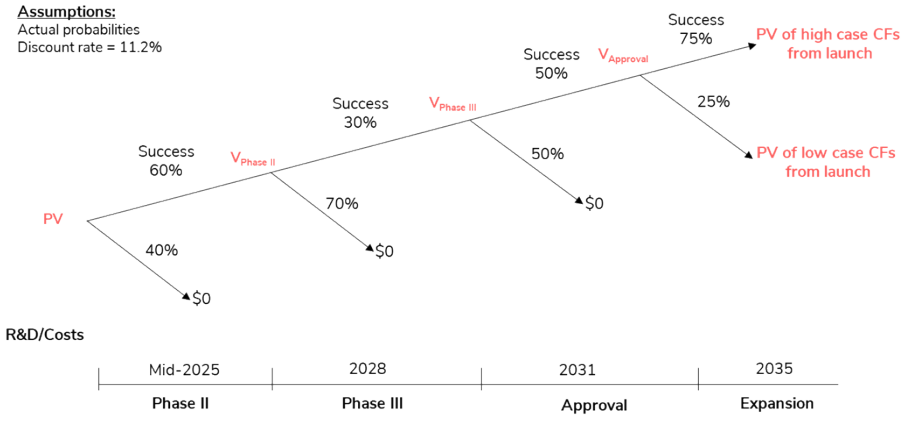
Generic Name	Brand Name	Manufacturer	5th year sales (US$b)	Peak sales (US$b)	Peak/5yr	Approved indications
Risankizumab	Skyrizi	AbbVie	2.5	28.2	11.3x	PP, PA, CD, UC
Dupilumab	Dupixent	Regeneron/Sanofi	3.5	24.8	7.1x	AD, Asthma, CR, EE, PN, CPU
Adalimumab	Humira	AbbVie	4.9	21.2	4.3x	RA, PA, AS, CD, UC, PP, HS, JIA, U
Upadacitinib	Rinvoq	AbbVie	2.0	15.4	7.7x	RA,PS, AS, AD, UC, CD, JIA, GCA
Efgartigimod	Vyvgart	Argenx	2.2	13.6	6.2x	MG, CIDP
Ustekinumab	Stelara	Janssen	1.3	11.3	8.7x	PP, PA, CD, UC
Infliximab	Remicade	Janssen (J&J)	1.6	9.9	6.2x	RA, PA, AS, CD, UC, PP
Etanercept	Enbrel	Amgen	1.5	8.7	5.8x	RA, PA, AS, PP, JIA
Secukinumab	Cosentyx	Novartis	2.0	8.3	4.2x	PP, PA, AS, NAS
Vedolizumab	Entyvio	Takeda	1.5	8.2	5.5x	UC, CD
Bimekizumab	Bimzelx	UCB	3.1	5.9	1.9x	PP,PA, HS
Eculizumab	Soliris	AstraZeneca	0.8	4.1	5.2x	PNH, aHUS, MG
Ixekizumab	Taltz	Eli Lilly	1.2	3.4	2.8x	PP, PA, AS, NAS
Tofacitinib	Xeljanz	Pfizer	0.9	2.5	2.8x	RA, PA, UC, AS
Natalizumab	Tysabri	Biogen	0.8	2.1	2.6x	MS, CD
Certolizumab	Cimzia	UCB	2.0	6.2	3.1x	CD, RA, PA, AS, PP, NAS, JIA
Belimumab	Benlysta	GSK	0.2	1.8	7.8x	SLE, LN
Rozanolixizumab	Rystiggo	UCB	n/a	1.1	n/a	MG
Zilucoplan	Zilbrysq	UCB	n/a	1	n/a	MG
Sarilumab	Kevzara	Sanofi/Regeneron	0.3	0.5	1.7x	RA, PMR, JIA

PP: plaque psoriasis; PA: psoriatic arthritis; CD: Crohn's disease; UC: ulcerative colitis; AD: atopic dermatitis; CR: chronic rhinosinusitis; EE: eosinophilic esophagitis; PN: prurigo nodularis; CPU: chronic spontaneous urticaria; RA: rheumatoid arthritis; AS: ankylosing spondylitis; HS: hidradenitis suppurativa; JIA: juvenile idiopathic arthritis; U: uveitis; GCA: giant cell arteritis; MG: myasthenia gravis; CIDP: chronic inflammatory demyelinating polyneuropathy; NAS: non-radiographic axial spondyloarthritis; PNH: paroxysmal nocturnal hemoglobinuria; aHUS: atypical hemolytic uremic syndrome; MS: multiple sclerosis; SLE: systemic lupus erythematosus; LN: lupus nephritis; PMR: polymyalgia rheumatica

Source: Company data, Visual Alpha, Wilsons Advisory.

Real options technique, revisited. Every individual program valuation we have for Immutep is based on the same framework: a stage-wise series of binomial options for each phase of product development (e.g. Phase I through to market access). The technique frames each stage of project R&D investment as a 'strike price' invested by Immutep for the option to increase asset value. **Figure 4** illustrates a 4-stage binomial for IMP761 including Phase II → Phase III → Approval (in a first indication) followed by indication expansion. Each stage is characterized by inputs: a) anticipated R&D investments; b) expected timeframes for phase completion; c) probabilities of success [p(success)]; d) downside valuations (zero for the first three phases); and e) upside valuations or the 'pay off' for each stage.

Figure 4: Real option framework for IMP761 valuation



Source: Wilsons Advisory.

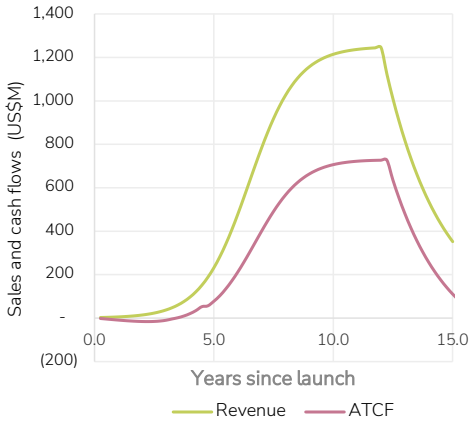
Ultimately, these models should be driven by an estimate of future project NPV; which in turn, rests on detailed market models of expected after tax cash flows from launch. The 'upside case' for IMP761 is commercialization in multiple autoimmune indications (the *PV of high case cash flows from launch* in **Figure 4** above). We frame the downside case as approval in a single (first) indication. We like this technique for valuing pipeline assets because it allows us to describe **risked** [p(success) < 100%] and **un-risked** [p(success) = 100%] valuations through each stage of development. In other words, the potential valuation upside from achieving each milestone catalyst, in turn. The p(success) we have assigned for IMP761 development phases are consistent with those used elsewhere in our Efti valuations (i.e. for NSCLC, HNSCC). Multiplying the probabilities together gives an overall project p(success) of approximately 7%, which is appropriate for a Phase I asset.

Dealing with uncertainty and IMP761's super-optionality … a job for Monte Carlo simulation. IMP761 is a special case because whatever Immutep chooses as a first indication is unlikely to adequately describe the asset's ultimate potential via indication expansion. For that reason, we wanted a valuation technique that could consider a vast range of potential outcomes (i.e. from US$1B to US$25B peak sales, informed by the basket of autoimmune products in **Figure 3** on the previous page. Using a range of peak sales 'predicates' to frame value excuses us from making indication-by-indication assessments of competitive intensity and market share. Another factor that is impossible to predict is the range of commercial pathways that Immutep could choose. Even when we limit ourselves to partnering scenarios (as opposed to outright M&A or independent commercialization) the mix of outcomes and potential deal structures is enormous. Our solution is to set up a Monte Carlo simulation, randomizing two parameters:

- **The upside available (FV)**. In **Figure 5** to the right we illustrate the general approximation used to model revenue and after tax cash flows (ATCFs). The model was used to assess the relationship between peak sales (varied from US$1-25B) and the future value (FV) assessed at time of launch. We assumed an economic life of 15 years; losing all forms of exclusivity (e.g. patent, biologic) at 12.5 years (followed by rapid decline, assuming biosimilar substitution. We assess a linear relationship between peak sales assumption and FV (**Figure 6**). In the Monte Carlo simulation, we randomized FV between US$1B and US$36B.
- **IMP761 economic returns (ER)**. Our base case assumption is that Immutep elects to pursue one or more global out-licensing transactions in relation to IMP761. We model Immutep capturing between 15% and 50% of the total project value (as economic returns) via some mix of upfront payments, royalties and milestone payments. We assume that Immutep invests up to US$75M over the 10-year program life.

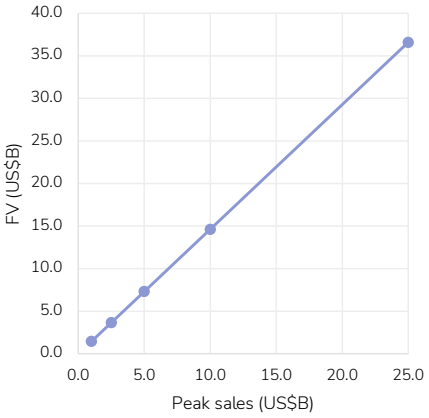
Real option calculation. We ran 10,000 option pricing valuation estimates, each scenario driven by Immutep's estimated share of IMP761 economics (i.e. randomized FV x randomized ER).

Figure 5: Sample commercial profile for revenue and after tax cash flow projections (ATCF)



Source: Wilsons Advisory.

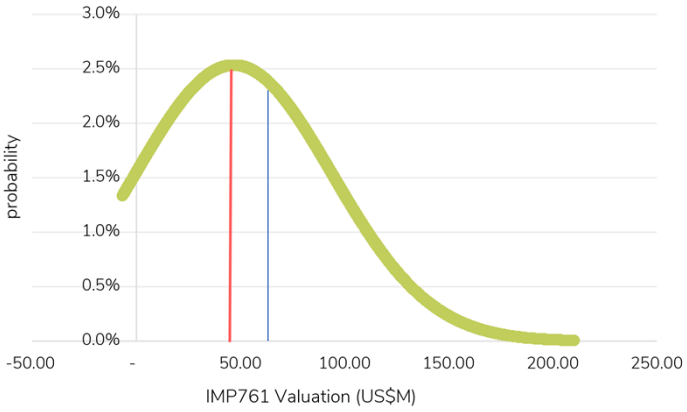
Figure 6: Linear relationship between peak sales and the NPV of after tax cash flows (asset FV)



Source: Wilsons Advisory.

Result: Risked IMP761 valuation of US$50-60M. We ran 10,000 simulations for randomized FY and ER; [US$1B ≤ FV ≤ US$36B] and [15% ≤ ER ≤ 50%]. Simulation results are depicted at right in **Figure 7** modelled as a normal distribution. The mean and median valuations were US$47M and US$60M, respectively. Assessed as a normal distribution we assess a wide 90% confidence interval of -US$30M to US$124M. All else equal, the median valuation of US$60M (A$0.06 per share) corresponds with US$3.75B of future value to Immutep shareholders (A$3.62 per share as an indication of un-risked value).

Figure 7: Monte Carlo simulation results identifying mean and median risked IMP761 valuations of US$47M and US$60M, respectively



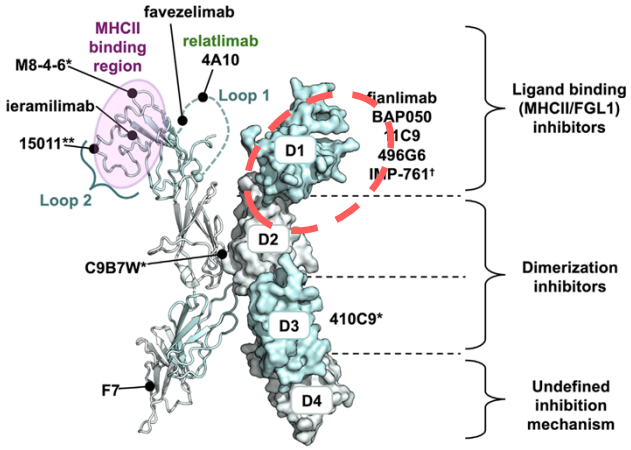
Source: Wilsons Advisory.

IMP761 | Targeting LAG-3 for the treatment of autoimmune disease

A novel LAG-3 agonist

Background on the drug molecule. IMP761 is a humanised, monoclonal antibody developed to agonise (activate) membrane bound LAG-3 activity. To create the molecule, mice were 'immunised' with soluble LAG-3 to elicit an antibody-mediated immune response. Antibody-producing hybridoma cells[1] were then generated and screened for LAG-3 binding and agonist activity. Once an appropriate murine anti-LAG-3 antibody was identified and cloned, its LAG-3 binding regions were taken and grafted onto a human, IgG4 antibody 'scaffold' to create a 'humanised' anti-LAG-3 monoclonal antibody. This molecule was named IMP761 and cloned into a qualified, mammalian cell line for large scale production. Binding studies show that IMP761 binds to the same part of LAG-3 that suppresses immune activation in nature (**Figure 8**)[2]. In late 2022, Immutep confirmed the development of a GMP manufacturing process for IMP761, in collaboration with CDMO Northway Biotech (Boston, MA) at 200L scale. In parallel, Immutep chose Charles River Laboratories to conduct pre-clinical toxicology studies to enable an Investigational New Drug (IND) application (in preparation). Specific immunological characterisation is being performed in The Netherlands at the Centre for Human Drug Research (CHDR).

Figure 8: LAG-3 shown in dimer form, describing points on the molecule have have become targets for drug discovery. IMP761 binds the natural site for LAG-3 activation, which triggers immunosuppressive signalling

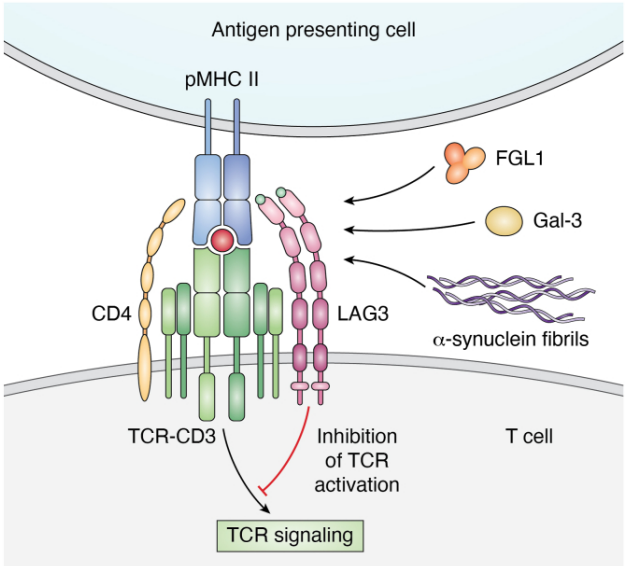


Source: Luca (2025)[3].

IMP761 mechanism of action

How LAG-3 works in T cell activation. The LAG-3 inhibitory receptor was first described by Immutep's CSO Dr Frederic Triebel in 1990[4]. Triebel et. al. initially described LAG-3 as a novel lymphocyte activation gene, that is closely related to CD4[5]. Whilst much has been learned about LAG-3 biology since, the mechanism by which it achieves immune suppression remains incompletely elucidated. Clues come from examining the mechanisms through which LAG-3 regulates immune responses, more generally. LAG-3 is expressed on several cell types including T cells, natural killer and dendritic cells. Briefly, T cells are a vital component of the adaptive immune system for both mounting an appropriate immune response; as well as directly killing cells that have become infected or cancerous. T cell 'activation' requires antigen-specific signalling through an antigen presenting cell (APC). APCs process antigens and present them to T cells for recognition/assessment. The junction at which this APC-to-T cell interaction takes place is called the 'immune synapse' and is illustrated in **Figure 9**. It shows the antigen (red circle) being presented by a large receptor complex called MHC class II (MHCII) on the APC's surface. The T cell side of that interaction involves the T cell receptor (TCR) and other receptors, including LAG-3; whose expression is upregulated with continuous antigen exposure. Importantly, unlike other immune checkpoint molecules, LAG-3 binds directly to the MHCII complex with direct involvement at the immune synapse. LAG-3's binding to the MHCII complex disrupts normal TCR activation and thereby restrains subsequent T cell differentiation and proliferation. Intriguingly, LAG-3 is also highly expressed on regulatory T cells (Tregs), cells that restrain immune-mediated inflammatory responses.

Figure 9: LAG-3 inhibition of TCR signalling

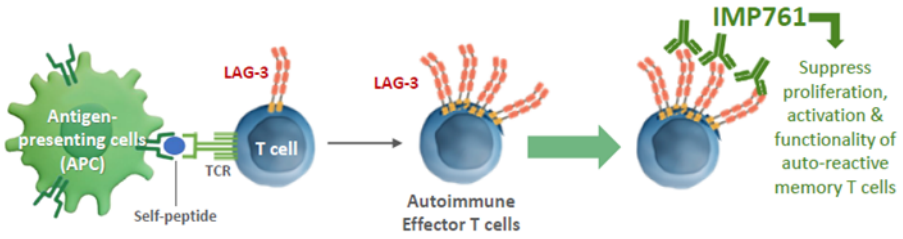


Source: Mariuzza et al. (2024)[6].

[1] Antibody generating spleen cells from the immunized mice, fused with myeloma cells to 'immortalise' them so they can be grown in cell culture.
[2] Agnihotri, P., et al. (2022) Epitope mapping of therapeutic antibodies targeting human inhibitory receptor lymphocyte activation gene 3 protein (LAG-3) J. Immunol. 209: 1586–1594.
[3] Luca, V. C. (2025) LAG time in the era of immunotherapy – new molecular insights into the immunosuppression mechanism of lymphocyte activation gene-3 Immunological Reviews 330:e70002.
[4] Triebel, F., et al. (1990) LAG-3, a novel lymphocyte activation gene closely related to CD4. Journal of Experimental Medicine. 171 (5):1393-1405.
[5] CD4 (cluster of differentiation 4) is a glycoprotein that serves as a co-receptor for the T-cell receptor (TCR). CD4 is found on the surface of immune cells such as helper T cells, monocytes, macrophages, and dendritic cells.
[6] Mariuzza, R. A., et al. (2024) The immune checkpoint receptor LAG-3: structure, function and target for cancer immunotherapy J. Biol. Chem. 300(5): 107241.

How LAG-3 'agonism' works as a selective strategy for immune suppression. LAG-3 is a near-perfect target for autoimmune drug development given its expression is so restrictive and specific to antigen exposure. LAG-3 immunosuppressive activity acts early in de-escalating inappropriate, autoreactive immune responses, therefore IMP761 targets autoimmune pathogenesis. By mimicking LAG-3's natural ligand(s)[7] and independently 'switching on' its inhibitory functions, IMP761 inhibits TCR signalling and suppresses the immune response to self-peptides (see **Figure 10**).

Figure 10: Suppression of auto-reactive memory T cells



Source: Immutep.

A multi-layered, nuanced dismantling of autoimmune disease drivers. As discussed above, LAG-3 agonist disrupts TCR signalling thereby directly suppressing activation and proliferation of $CD4^+$ and $CD8^+$ T cells and subsequently reducing proinflammatory cytokines. Autoreactive memory T cells accumulate at the sites of disease with high and sustained LAG-3 expression on their surface. IMP761 therefore has the potential to directly suppress the immune response through $CD4^+$ and $CD8^+$ T cells; whilst supporting peripheral tolerance. LAG-3 is highly expressed on Tregs – cells that are critical for maintaining peripheral tolerance and immune homeostasis. As with all T helper subsets, Tregs exhibit plasticity as Th17 or Th17-like cells with high levels of IL-6; such as that found in the synovial fluid of RA patients[8]. These cells then contribute to further inflammation rather than limiting the response. As such, there are two possible mechanisms through which LAG-3 agonists may support Treg function. First, LAG-3 signalling plays a central role in Treg metabolism crucial to suppression during inflammatory conditions; second, reducing inflammation supports Treg commitment by reducing cytokines that drive plasticity[9].

What sets a LAG-3 agonist apart is the direct suppression of $CD4^+$ and $CD8^+$ T cells; in conjunction with supporting the suppressive action and commitment of Tregs. GSK/IMM have previously collaborated a LAG-3 targeted program that depletes $LAG\text{-}3^+$ cells through antibody-dependent cell cytotoxicity (GSK2831781). They were able to demonstrate proof of concept in a Phase I trial in psoriasis patients however, they couldn't demonstrate efficacy in UC patients. Generally speaking, all 'depleting antibody' strategies suffer from this lack of specificity, wiping out all LAG-3-positive cells indiscriminately. IMP761 is powerfully differentiated in its ability to support Tregs rather than killing off this subset that is central to maintaining tolerance. Validating the 'checkpoint inhibitory receptor agonist idea', there are now four PD-1 agonist programs in clinical development for autoimmune indications. Whilst both LAG-3 and PD-1 are inhibitory receptors, up-regulated in exhausted T cell phenotypes, we believe LAG-3 to be a better target in localized inflammatory autoimmune conditions. PD-1 is more widely expressed on activated T cells and B cells, whereas LAG-3 expression is more concentrated to chronic activation.

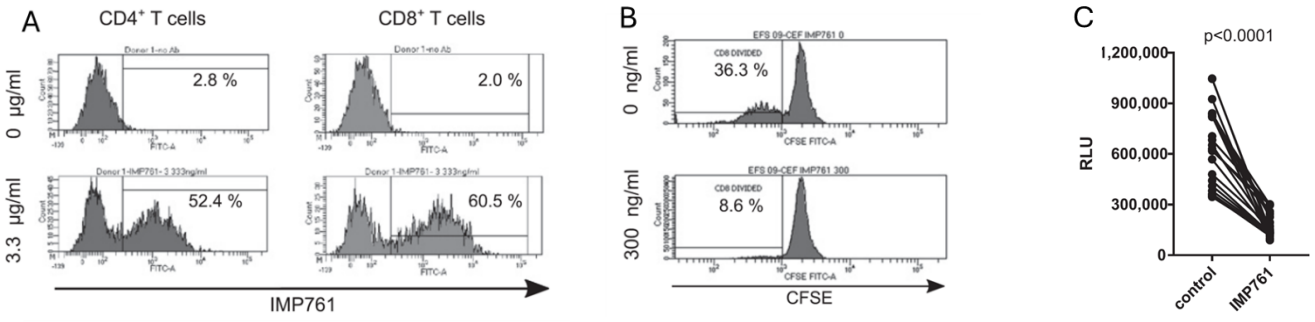
[7] Whilst MHCII is regarded as the 'canonical' ligand for LAG-3, four others have been described: a-syn, Gal-3, LSECtin and FGL-1.
[8] Su, Q., et al. (2024) Exploring the therapeutic potential of regulatory T cell in rheumatoid arthritis: Insights into subsets, markers, and signaling pathways, Biomedicine And Pharmacotherapy, 174:116440.
[9] Kim, D. et al. (2024) Inhibitory co-receptor LAG-3 supports Foxp3+ regulatory T cell function by restraining Myc dependent metabolic programming, Immunity. 57, 2634-2650.

Preclinical evidence for IMP761's immunosuppression

Immutep have quietly been developing IMP761 for over five years with the first publication of their preclinical data in 2020[10]. In that enigmatic paper, Triebel's group published encouraging results from *in vivo* and *in vitro* studies, demonstrating the immunosuppressive properties of IMP761. This study provided further context to the structure and function of IMP761 and provided enough confidence to embark upon Phase I safety and further studies seeking to validate the biology of LAG-3 agonism as a therapeutic strategy in autoimmune disease.

IMP761 bound to both CD4⁺ and CD8⁺ T cells with high affinity and inhibited antigen-induced proliferation of activated T cells. Flow cytometry staining was used to demonstrate IMP761 binding to CD4⁺ and CD8⁺ T cells that had been activated by exposure to an antigen[11] (**Figure 11A**). The immunosuppressive properties of IMP761 were tested in a standard assay that tracks cell division and proliferation (CFSE[12]). **Figure 11B** shows that IMP761 (present at a concentration of 300 mg/mL) inhibited CD8⁺ T cell activation and proliferation in the presence of viral antigens[13]. Further to this, IMP761 inhibited NFAT[14] activation in a dose dependent manner (**Figure 11C**).

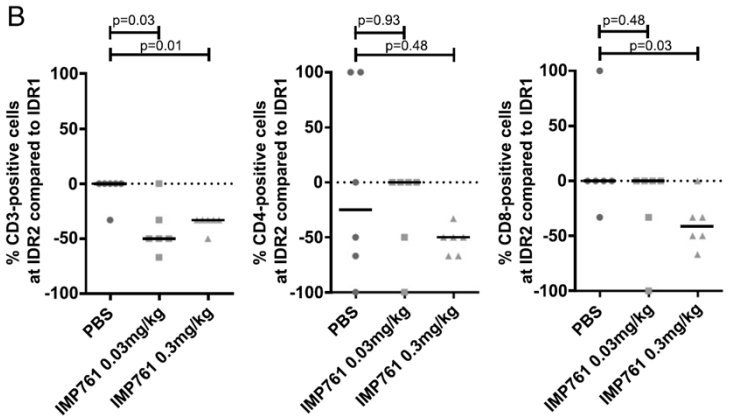
Figure 11: Binding of IMP761 to CD4⁺ and CD8⁺ T cell subsets, reduced CD8 division in the presence of 300ng/ml IMP761



Source: Angin et. al. 2020.

Well tolerated by non-human primates (NHP) with potentially active concentrations achieved. The NHP component of this study demonstrated that IMP761 was well tolerated in 6 animals at a pharmacodynamically active range. Eighteen animals were vaccinated with BCG vaccine and then challenged twice with tuberculin (antigen). This model is considered a surrogate model for psoriasis[15]. One day before the second tuberculin challenge, the animals were treated with 0.03 mg/kg IMP761, 0.3mg/kg IMP761 and a saline control (6 animals in each group). Pharmacodynamically active blood levels of drug were achieved. Although evidence of an anti-IMP761 immune response emerged 2 weeks after exposure, this immunogenicity (unsurprising, exposing macaques to a humanised antibody) did not impact the experiment. Skin biopsies were also performed to evaluate cell infiltration at the intradermal challenge sites before and after the administration of IMP761/saline. The IMP761 injections did not influence circulating T cell subsets following the intradermal injection. This is consistent with the fact that LAG-3 positive cells are extremely rare in the peripheral blood of animals. CD8⁺ T cell infiltration was decreased at the dermal challenge site; whilst CD4⁺ T cells were not affected, possibly due to lower LAG-3 expression (**Figure 12**).

Figure 12: Tuberculin-induced CD3-, CD4- or CD8-positive T cell inflitration at intradermal reaction site



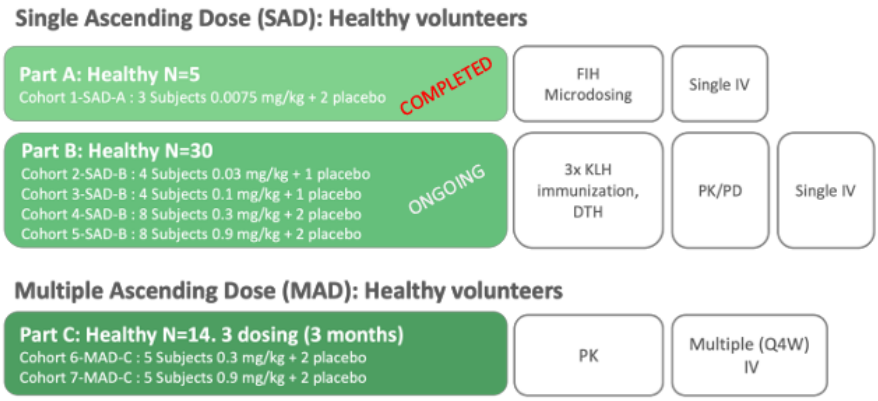
Source: Angin et. al. 2020.

[10] Angin, M., et al. (2020) A LAG-3–Specific Agonist Antibody for the Treatment of T Cell–Induced Autoimmune Diseases. The Journal of Immunology. 204: 810-818.
[11] These human T cells were pre-incubated for two days with staphylococcal enterotoxin B.
[12] CFSE dilution is a technique that uses a dye (carboxyfluorescein diacetate succinimidyl ester). As cells divide, they distribute the dye equally between daughter cells. The intensity of fluorescence is related to the amount of cell division.
[13] Human CD8⁺ T cells incubated for six days with a peptide pool derived from CMV, Epstein-Barr and influenza virus.
[14] NFAT: nuclear factor of activated T cells; a family of transcription factors crucial in regulating T cell development, activation and self-tolerance.
[15] Poirier, N., *et al.* (2016) Selective CD28 antagonist blunts memory immune responses and promotes long-term control of skin inflammation in nonhuman primates. J. Immunol. 196: 274–283.

Phase 1 clinical trial in healthy volunteers

IMP761 is currently being investigated in a placebo controlled double-blind Phase I trial to evaluate safety, pharmacodynamics and pharmacokinetics in up to 49 healthy volunteers (NCT06637865). There are 3 parts to the study as per the schematic below in **Figure 13**.

Figure 13: Phase I trial design evaluating IMP761



Source: Company presentation.

First 'proof of mechanism' observations. To demonstrate the immunosuppressive mechanism of IMP761 in healthy volunteers, Part B of the study includes a Keyhole Limpet Haemocyanin (KLH) skin challenge, which is a widely used clinical model for human immunotoxicology studies[16]. Briefly, the KLH antigen is a very large, copper-containing protein derived from an inedible mollusc. It is also a potently immunogenic, T cell dependent antigen that provokes potent immune responses. Healthy subjects were immunised with KLH and rechallenged on subsequent days (day 2 for cohorts 2-3; days 2, 9 and 23 for cohorts 4-5). The KLH challenge induces infiltrating LAG-3$^+$ T cells at the site, harvested through blister sampling [17]. Immutep recently announced results from cohort 5 (dosing at 0.9 mg/kg), demonstrating an 80% reduction of infiltrating T cells at day 10 with no TRAEs. These are exciting results for an initial immune challenge, and we look forward to gaining a better understand of IMP761 as further results are released during 2025. The full KHL challenge dataset will demonstrate initial proof of mechanism in humans including cutaneous microcirculation with laser speckle contrast imaging and changes to erythema severity/shape with multispectral skin imaging.

[16] Swaminathan, A., *et al.* (2014) *Keyhole limpet haemocyanin – a model antigen for human immunotoxicology studies* Br. J. Clin. Pharmacol. 78(5): 1135 – 1142.
[17] Saghari, M., et al. (2022) Characterization of KLH-driven immune responses in clinical studies: A systematic review. Frontiers in Drug Discovery. 2.

Autoimmune disease market opportunities

Immune checkpoint inhibitors have predominantly been developed for use in the oncology setting, having revolutionized the treatment of certain cancers. Merck's Keytruda being the highest grossing, generating US$29.5B in FY2024. Checkpoint receptors such as PD-1, CTLA-4 and LAG-3 limit the immune response to ongoing activation, therefore therapeutics that block receptor/ligand interactions can restore/enhance immune function to fight cancer cells. This MOA targeting checkpoint inhibitors in cancer has provided real-world evidence for their potential use within the inflammation and immunology setting. Patients being treated with these therapeutics that have preexisting autoimmune conditions may experience flares post-treatment[18]. These immune related AEs to checkpoint antagonist/receptor blocking highlights the potential for receptor agonism in this population.

Autoimmune disease market primer

Autoimmune disease is set to be the next big opportunity for targeting checkpoint inhibitory receptors, with estimates as high as 50M patients in the US alone[19]. Autoimmune encompasses a broad spectrum of immune-mediated diseases, with over 100 recognised conditions, affecting almost every tissue[20]. They are characterised by the immune system's inability to distinguish 'self' from foreign antigens (e.g. viruses or other pathogens), causing cells to become 'autoreactive' and attack healthy cells. Autoreactive cells can lead to chronic inflammation and tissue damage. Targeting checkpoint inhibitory receptors to suppress this autoreactivity is a new, disease modifying mechanism of action to a complicated and often underserved treatment paradigm.

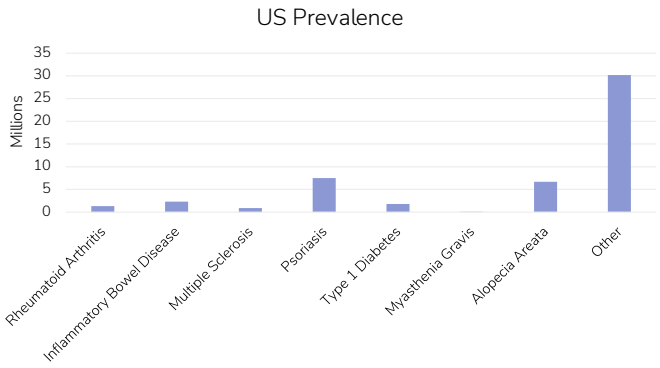
As T cells are central to mounting adaptive immune responses, limiting their activation will be immunosuppressive. Checkpoint molecules (including LAG-3) being investigated in the autoimmune therapeutic space can further constrain the immune response by limiting the production of cytokines or the T cell-mediated production of antibodies. Therefore, an approach exploiting a checkpoint receptor agonist has the potential to be widely used across the autoimmune space. T cell driven autoimmune conditions that localise to a specific region or tissue will be the lowest hanging fruit. Rheumatoid arthritis (RA) and inflammatory bowel disease (IBD) are good examples of initial indications with large addressable markets and a strong rationale to target LAG-3. Other potential indications Immutep could go after include multiple sclerosis (MS), psoriasis, type 1 diabetes, myasthenia gravis, alopecia areata and Hashimoto's disease.

Autoimmune disease encompasses many indications with market size and projections varied. Additionally, diagnosis of autoimmune diseases has been rising, in part due to improved diagnosis and physician awareness, with the changes to our environment also likely to be contributing[21]. The blockbuster indications like RA and IBD are still only a small portion of the potential total in a market like autoimmune disease (**Figure 14**).

Abbvie's Humira highlights the opportunity in the autoimmune space. Humira was first approved in 2002, indicated for use in a range of diseases including RA, Crohn's disease, ulcerative colitis (UC) and psoriasis. Humira's peak revenue reached US$21B and has generated >US$200B in cumulative sales to date.

With so much opportunity in the Immunology and Inflammation (I&I) space including autoimmune disease, Pharma has been making large investments to build out their internal pipelines and product portfolios

Figure 14: US addressable markets for leading autoimmune indications



Source: as referenced[22,23,24,25,26,27,28], Wilsons Advisory.

[18] Sparks, J. (2024) Pre-existing Autoimmune Diseases and Immune Checkpoint Inhibitors for Cancer Treatment: Considerations About Initiation, Flares, Immune-Related Adverse Events, and Cancer Progression. Rheumatic Disease Clinics of North America. 50 (2): 147-159

[19] Murray, M. (2024) Guest Blog: A Major Health Crisis: The Alarming Rise of Autoimmune Disease. National Health Council Guest Blog. https://nationalhealthcouncil.org/blog/a-major-health-crisis-the-alarming-rise-of-autoimmune-disease/#:~:text=Data%20indicates%20that%20autoimmune%20diseases,of%203%2D12%25%20annually.

[20] Cleveland Clinic. Autoimmune Disease (https://my.clevelandclinic.org/health/diseases/21624-autoimmune-diseases) Accessed 05/2025.

[21] Miller, F. (2022) The increasing prevalence of autoimmunity and autoimmune diseases: an urgent call to action for improved understanding, diagnosis, treatment, and prevention. Current Opinion in Immunology. 50: 102266

[22] Xu, Y., et al. (2021) Prevalence Trend and Disparities in Rheumatoid Arthritis among US Adults, 2005–2018. Journal of Clinical Medicine 15:2389

[23] Weisman, M., et al. (2023) Inflammatory Bowel Disease Prevalence: Surveillance data from the U.S. National Health and Nutrition Examination Survey. Preventive Medicine Reports. 33:102173.

[24] De Nadai, A., et al. (2024) Multiple Sclerosis Subgroups: Data-Driven Clusters Based on Patient Reported Outcomes and a Large Clinical Sample. Multiple Sclerosis Journal. 30(13):1642-1652.

[25] Armstrong, A., et al. (2021) Psoriasis Prevalence in Adults in the United States. JAMA Dermatology. 157(8):1-7.

[26] CDC. National Diabetes Statistics Report 2024 (https://www.cdc.gov/diabetes/php/data-research/index.html)

[27] Ye, Y., et al. (2024) Epidemiology of myasthenia gravis in the United States. Frontiers in Neurology. 15:1339167.

[28] National Alopecia Areata foundation. New Research: Prevalence of Alopecia Areata across Races and Ethnicities. 05/2023 (https://www.naaf.org/news/new-research-prevalence-of-alopecia-areata-across-races-and-ethnicities/)

(**Figure 15**). There have been several major acquisitions and in-licensing deals over the last few years. Most notable was Merck's acquisition of Prometheus in April 2023 for $10.8B. The acquisition followed the announcement of Phase II results Prometheus' lead drug PRA023 (anti-TL1A) in UC and CD. Importantly Phase II results from PRA023 demonstrates the potential value of a first-in-class and best-in-class asset within these indications. Additionally, there have been several large acquisitions and licensing deals or preclinical assets further highlighting the potential value behind these assets. The autoimmune market is projected to exceed $116B by 2032[29].

Figure 15: Major deal activity in immunology and inflammation since 2023

Acquirer	Acquisition Target	Deal Type	Transaction Value (upfront + milestones)	Date	Lead Asset Stage	Indication
Merck & Co	Prometheus Biosciences	M&A	$10.8b	16-Apr-23	Ph2	Inflammatory Bowel Disease
Eli Lilly And Co	DICE Therapeutics	M&A	$2.4b	20-Jun-23	Ph2	Chronic Autoimmune Diseases
Roche	Telavant	M&A	$7.1b	23-Oct-23	Ph2	Inflammatory Bowel Disease
AstraZeneca	Gracell	M&A	$1.2b	16-Dec-23	Ph2 IIT	Haematoloic Malignancies Autoimmune
Sanofi	Inhibrx	M&A	$1.7b	23-Jan-24	Ph2	Alpha-1 Antitrypsin Deficiency
AbbVie	Landos Biopharma	M&A	$212m	25-Mar-24	Ph2	Ulcerative Colitis
Vertex Pharmaceuticals	Alpine Immune Sciences	M&A	$4.9b	10-Apr-24	Ph3	IgA Nephropathy
Johnson & Johnson	Proteologix	M&A	$850m + milestones	16-May-24	Ph1	Atopic Dermatitis
Biogen	Human Immunology Biosciences	M&A	$1.15b	22-May-24	Ph2	IgA Nephropathy
Asahi Kasei	Calliditas Therapeutics	M&A	$1.6b	28-May-24	Approved	IgA Nephropathy
AbbVie	FutureGen	Licensing	$1.7b	13-Jun-24	Preclinical	Inflammatory Bowel Disease
AbbVie	Celsius Therapeutics	M&A	$250m	27-Jun-24	Ph1	Inflammatory Bowel Disease
Eli Lilly And Co	Morphic Therapeutics	M&A	$3.2b	08-Jul-24	Ph2	Inflammatory Bowel Disease
Johnson & Johnson	Yellow Jersey Therapeutics	M&A	$1.25b	11-Jul-24	Ph2	Atopic Dermatitis
Novartis	Monte Rosa	Licensing	$2.25b	28-Oct-24	Ph1	Autoimmune
AbbVie	Nimble Therapeutics	M&A	$200m	13-Dec-24	Preclinical	Psoriasis
Gilead	Leo Pharma	Licensing	$1.7b	11-Jan-25	Preclinical	Inflammatory Diseases
Sanofi	Earendil Labs	Licensing	$1.8b	17-Apr-25	Preclinical Preclinical	IBD autoimmune
Sanofi	Dren Bio	M&A	$1.9b	20-Mar-25	Preclinical	B-cell mediated autoimmune

Source: Company data, Wilsons Advisory.

[29] Strategy & Stats. Global Autoimmune Disease Therapeutics Market to Reach USD 116.81 Billion by 2032, Growing at a 5.52% CAGR. 2024. (https://www.globenewswire.com/news-release/2024/12/13/2996844/0/en/Global-Autoimmune-Disease-Therapeutics-Market-to-Reach-USD-116-81-Billion-by-2032-Growing-at-a-5-52-CAGR-SNS-Insider.html)

Strategic positioning for IMP761

As briefly noted in the Valuation section, the autoimmune disease market offers opportunities to build vast product franchises because so many large indications share a common, underlying pathophysiology. RA and IBD are both blockbuster opportunities, with annual drug cost/patient sitting between ~US$20,000-$50,000 for approved biologics, and around 30% of RA (~400,000 US patients: ~US$8-20B), 32% of CD (~240,000 US patients: ~US$4.8-12B) and 15% of UC (~90,000 US patients: ~US$1.8-4.5B) patients on biologics, generating substantial revenue[30,31]. There are several approved therapeutics and additional programs in development with novel mechanisms of action IBD and RA are highly competitive indications. While both offer a good opportunity for IMP761, IMM could make the strategic decision to pursue a less competitive space as an initial indication. This could be along the lines of primary sclerosing cholangitis a chronic progressive liver disease wherein inflammation and scaring damage the bile ducts, leading to liver failure. What would make sclerosing cholangitis an attractive indication is the localised inflammation of bile ducts suggesting LAG-3 expression as a T cell driven autoimmune condition with high unmet need and no approved disease modifying therapy.

FcRn inhibitors Vyvgart (ArgenX) and Rystiggo (UCB) are good examples of the potential for indication expansion possible when addressing autoimmune indications. FcRn inhibitors promote IgG degradation to address a broad range of autoimmune indications, with an estimated 2M people living with IgG autoantibodies. Vyvgart initially focused on approval in generalized myasthenia gravis and chronic inflammatory demyelinating polyneuropathy. Indications with appropriate pathophysiology, clear unmet needs and strong clinical endpoints serve as good initial indications to demonstrate efficacy and commercial opportunity. Vyvgart has now entered registrational trials for an additional 6 indications. IMP761 could follow a similar pathway with a strong initial indication (and high pricing) followed by a series of registrational studies in larger, T cell driven autoimmune indications.

M&A and licensing activity in I&I is high with preclinical assets able to fetch deals in excess of $1B. Abbvie, JNJ (Janssen), Pfizer, Eli Lilly, Novartis, Amgen, Roche, Sanofi, BMS, Merck, GSK and AstraZeneca all have a focus on I&I with more than 200 clinical trials across more than 100 programs currently in development (program tracker available on request). These programs are either broad immunosuppressive agents or targeting a specific cell subset.

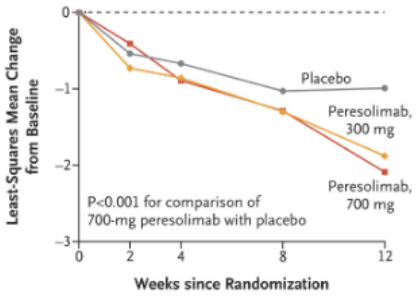
[30] Detert, J., et al. (2015) Biologic monotherapy in the treatment of rheumatoid arthritis, Biologics. 14(9):35-43.
[31] Xu, F., et al. (2022) Trends and demographic patterns in biologic and corticosteroid prescriptions for inflammatory bowel disease: findings from electronic medical records, 2011–2020, Journal of Investigational Medicine, 70(8):1771-1776.

Previously investigated checkpoint inhibitory receptors in autoimmune conditions

The idea of developing immunosuppressive drugs targeting checkpoint inhibitory receptors is further validated by recent Pharma development activity. It represents a fundamentally different approach compared to traditional immunosuppressive therapies, which often indiscriminately dampen the immune system, leaving patients vulnerable to infections and other complications. PD-1 was a natural target, given the incredible success of its various inhibitors in oncology. Pharma has pursued two basic strategies, developing 'depleting antibody' and 'agonist' programs, summarised below in **Figure 17**.

There are now 5 checkpoint agonist or depleters that have entered clinical trials. These programs have had mixed result in clinical trials, with GSK2831781 showing some efficacy in psoriasis patients during a Phase I study, yet no improvement over placebo in a Phase II UC study. Eli Lilly investigated peresolimab (a PD-1 agonist) in a Phase 2 study for the treatment of RA. Peresolimab met the primary endpoint demonstrating efficacy at week 12 in the disease activity score 28 joints – C reactive protein (DAS28-CRP) compared to placebo (**Figure 16**)[32]. Eli Lilly has since discontinued development of peresolimab following an analysis of the overall risk/benefit profile despite being very positive on Phase II results[33]. Discontinuation of this asset could have been due to concerns of systemic immunosuppression with few patients exhibiting respiratory and skin infections. JNJ recently presented results from JNJ4703 trials demonstrating safety and efficacy in treated patients. Finally, Gilead just commenced a Phase I with its PD-1 agonist (GS-0151).

Figure 16: Phase II trial of peresolimab in RA patients, primary endpoint changes from baseline DAS28-CRP



Source: Tuttle, J. et al. 2023.

Figure 17: clinical trials that have investigated checkpoint inhibitory receptors

Company	GlaxoSmithKline plc/Immutep		Eli Lilly		AnaptysBio			Johnson and Johnson		Gilead
Program	GSK2831781		Peresolimab		Rosnilimab			JNJ 4703		GS-0151
MOA	LAG-3 depleter		PD-1 agonist		PD-1 depleter and agonist			PD-1 agonist		PD-1 agonist
Phase	I	II	I	II	II	II	II	I	II	I
Indication	Psoriasis	Ulcerative Colitis	Healthy Volunteers	Rheumatoid Arthritis	Alopecia Areata	Rheumatoid Arthritis	Ulcerative Colitis	Rheumatoid Arthritis	Rheumatoid Arthritis, Ulcerative Colitis and Sjogren Disease	Rheumatoid Arthritis
Size	40 HV + 27 Psoriasis	104 UC	57 HV	98 RA	45 Alopecia	424 RA	132 UC	44 RA	17 RA, 5 UC and 15 SjD	75 RA
Results	Reduction from baseline compared with placebo PLSS -2.0 and PASI -3.3	Limited response - discontinued	-	DAS28-CRP between group difference change from baseline at week 12 -1.09 dose: 700mg	-	69% achieved LDA over 6 months	Recruiting	DAS28-CRP improvement from baseline compared to placebo.	Reduced PD1 high Tfh cells	Recruiting
Clinical trial	NCT02195349	NCT03893565	NCT05959109	NCT05516758	NCT05205070	NCT06041269	NCT06127043	NCT04985812	2022-001528-14 (EudraCT number)	NCT06902519

Source: as referenced, Wilsons Advisory.

[32] Tuttle, J., et al. (2023) A Phase 2 Trial of Peresolimab for Adults with Rheumatoid Arthritis, The New England Journal of Medicine, 388:1853-1862.

[33] Taylor, N., (2024, Oct 30). Eli Lilly rolls the dice, axing psoriasis prospect and pivoting to follow-up 1 year after $2.4B buyout. Fierce Biotech. https://www.fiercebiotech.com/biotech/eli-lilly-rolls-dice-axing-psoriasis-prospect-and-pivoting-follow-1-year-after-24b-buyout.

IMP761 development examples

There are numerous autoimmune conditions wherein treatment with a LAG-3 agonist may be beneficial, although it is still not clear which indication will be the first investigated. Here, we explore rheumatoid arthritis (RA) and Inflammatory bowel syndrome (IBS) as potential initial indications. RA and IBD both have substantial unmet needs and scientific rationale for targeting LAG-3. A first-in-class MOA would likely produce a successful commercial launch.

Rheumatoid Arthritis (RA)

RA is likely to be one of the first indications investigated given the large addressable market, unmet need and validated LAG-3 expression. RA is a common systemic autoimmune condition affecting >200 people/100,000[34] translating to ~17m people worldwide or ~1.3m adults with RA in the US[35]. Joint inflammation is a defining feature of RA, resulting in arthritic joint presentations. Briefly, autoreactive T cells in the affected joint activate macrophages and fibroblasts via proinflammatory cytokines secreted by Th1 or Th17 cells. Macrophages further contribute to the proinflammatory milieu with TNFα, IL-1β and IL-6 also present. Activated T cells support B cell activation and the production autoantibodies. Although RA generally does not pose an immediate life-threatening risk, chronic RA leads to the destruction of joints and may reduce life expectancy by 3-10 years[36]. Untreated RA can lead to cardiovascular disease, lung disease and lymphoma. Although there are numerous diseases modifying drugs approved for the treatment of RA, ~20% of patients are considered uncontrolled or difficult to treat wherein after several rounds of treatment they are unable to meet treatment goals [37]. Approval of new mechanisms of action will likely be required to see an improvement in these patients. IMP761 has the potential to be a first-in-class therapeutic in RA.

The treatment of RA has improved over the years however there is still an unmet need with many patients progressing through lines of therapy. The primary treatment of RA is disease modifying antirheumatic drugs (DMARDs), this is a class of drugs indicated for use in arthritis as well as several other inflammatory indications. DMARDs are immunosuppressive or immunomodulatory with unique MOAs that can interfere with inflammatory pathways[38]. DMARDs are now categorized as conventional synthetic, biologic and targeted synthetic. Conventional synthetic DMARDs (csDMARDs) are small molecules with broad immunosuppression, used as first-line treatment in RA, in particular Methotrexate (MTX: inhibits dihydrofolate[39]). MTX is recommended over biologic DMARDs and small molecule drugs by the American college of Rheumatology. Within 2 years 66% of patients discontinue MTX due to efficacy or toxicity [40]. Patient will often move onto other csDMARDs with high failure rates prior to biologic use. TNFα inhibitors are often the first biologics used, this includes AbbVie's Humira. Targeted synthetic DMARDs are used if the patient fails biologics or when injectables are undesirable, this is often a JAK inhibitor such as Rinvoq. Many biologic and targeted synthetic DMARDs have black box warnings on the label (**Figure 18**). As such IMP761 has the potential to differentiate with a clean safety profile. IMP761 is expected to have a good safety profile due to targeting only activated cells with LAG-3 expression. A clean safety profile may also mean that IMP761 could be used in combination with other DMARDs. The RA market is estimated to be >$26b; IMP761 would likely sit behind a generic MTX and other csDMARDs with potential to be used as the first biologic assuming good efficacy and safety. Humira continues to be the most utilised TNFα inhibitor; in combination with MTX patients achieve 63% ACR20 at 24 weeks [41].

Figure 18: Black box warnings on FDA approved biologics for the treatment of RA

Class	Name	Black box warning
TNFα inhibitors	Humira Cimzia Simponi Enbrel Remicade	Serious Infection and Malignancy
IL-6 inhibitors	Actemra Kevzara	Serious Infection
Costimulation blocker	Orencia	-
B-cell depletion	Rituxan	Fatal Infusion Reactions, Tumour Lysis Syndome, Severe Mucocutaneous Reactions and Progressive Multifocal Leukoencephalopathy
IL-1 antagonist	Kineret	-
Jak inhibitors	Xeljanz	Serious Infection and Malignancy
	Olumiant	Serious Infection, Mortality, Malignancy, Major Adverse Cardiovascular Events and Thrombosis
	Rinvoq	

Source: Company data, Wilsons Advisory.

There is clear rationale to target LAG-3 in RA patients. Inflammatory arthritis is one of the most common immune related adverse events in oncology patients treated with immune checkpoint inhibitors [42]. LAG-3 is highly expressed on memory CD4$^+$ T cells in the synovial fluid (compared with PBMCs) and have the capacity express cytokines in RA patients[43]. This demonstrates that RA is an autoimmune condition wherein LAG-3$^+$ memory T cells accumulate at the site of inflammation with limited expression in the periphery. Therefore, IMP761 treatment would likely benefit these patients by limiting the T cell response at the site of the disease while having minimal effect on the wider immune system.

[34] Black, R., et al (2023) Global, regional, and national burden of rheumatoid arthritis, 1990–2020, and projections to 2050: a systematic analysis of the Global Burden of Disease Study 2021, The Lancet Rheumatology. 5:e594-610.
[35] Xu, Y., et al. (2021) Prevalence Trend and Disparities in Rheumatoid Arthritis among US Adults, 2005–2018, Journal of Clinical Medicine. 10(15):3289.
[36] Dedmon, L., (2020) The genetics of rheumatoid arthritis, Rheumatology, 59(10):2661-2670.
[37] Takanashi, S., et al. (2004) Unmet Needs and Current Challenges of Rheumatoid Arthritis: Difficult-to-Treat Rheumatoid Arthritis and Late-Onset Rheumatoid Arthritis, Journal of Clinical Medicine, 13:7594.
[38] Onecia, B., et al. (2023) Disease-Modifying Antirheumatic Drugs (DMARD), StatPearls Publishing; Available from: https://www.ncbi.nlm.nih.gov/books/NBK507863/
[39] Cronstein, B., et al. (2020) Methotrexate and its mechanisms of action in inflammatory arthritis, Nature Reviews Rheumatology, 16:145-154.
[40] Van Der Kooij, S., (2007) Limited efficacy of conventional DMARDs after initial methotrexate failure in patients with recent onset rheumatoid arthritis treated according to the disease activity score, Annals of the Rheumatology Diseases, 66(10):1356-1362.
[41] Keystone, E., et al. (2004) Radiographic, clinical, and functional outcomes of treatment with adalimumab (a human anti–tumor necrosis factor monoclonal antibody) in patients with active rheumatoid arthritis receiving concomitant methotrexate therapy: A randomized, placebo-controlled, 52-week trial, Arthritis and Rheumatology, 50(5):1400-1411.
[42] Small, A., et al. (2023) Immune checkpoints in rheumatoid arthritis: progress and promise, Frontiers Immunology, 14.
[43] Pedersen, J., et al. (2023) Lymphocyte activation gene 3 is increased and affects cytokine production in rheumatoid arthritis, Arthritis Research and Therapy, 97.

Inflammatory bowel disease (IBD)

IBD is a group of disorders that cause inflammation of the digestive tract: ulcerative colitis (UC) and Crohn's disease (CD).

- UC is the inflammation of and ulcers lining your colon. Symptoms typically include diarrhea, bleeding, pain, weight loss and fatigue however more severe symptoms may manifest, including severe bleeding, perforated colon, dehydration, anaemia and osteoporosis. UC affects ~5 million patients worldwide and ~600,000-900,000 in the US. Patients often have a genetic predisposition and environmental factors that may affect the gut epithelial barrier[44]. Although there are several approved therapeutics an unmet need remains with 10-20% of patients progressing to require a proctocolectomy. Additionally, patients with uncontrolled or untreated UC have increased risk of developing colorectal cancer[45].

- Unlike UC that only affect the colon and the lining, CD may occur throughout the digestive tract with inflammation affecting all layers of the bowel wall. Patients commonly exhibiting diarrhea, fatigue, pain and reduced appetite with inflammation of the liver, skin, eyes and joints leading to increased risk of colorectal cancer, skin disorders, osteoporosis, kidney stones, gallbladder and liver disease. CD is estimated to affect ~6m people worldwide and ~750,000 people in the US[46].

Figure 19: FDA approved biologics for IBD

Class	Name	Indication
TNFα inhibitors	Humira	CD, UC
	Cimzia	CD
	Simponi	UC
	Remicade	CD, UC
IL-12/23 inhibitor	Stelara	CD, UC
IL-23 inhibitors	Skyrizi	CD, UC
	Omvoh	CD, UC
Anti-integrin	Entyvio	CD, UC
	Tysabri	CD (refactory)

Source: Company data, Wilsons Advisory.

Treatment of UC and CD varies based on the diagnosis however moderate-severe late-stage patients are moved onto biologic treatment following inadequate responses to conventional therapy (**Figure 19**). 32% of CD patients and 15% UC patients go on to be prescribed biologics with 20% of UC patients and 80% of CD patients requiring surgical intervention throughout their lifetime [47]. Demonstrating a clear unmet need in IBD and the need for additions mechanisms of action to improve outcomes for patients.

There is a clear rational for IMP761 development in IBD with several studies investigating LAG-3 expression. Single cell RNA sequencing revealed the expression and LAG-3 in cytotoxic T cells in the intestinal mucosa of CD patients[48]. Beyond this study there is little evidence of LAG-3 in CD, however, there has been several investigating LAG-3 in UC. LAG-3 is expressed by T cells in the inflamed region of the colon compared to the uninflamed or circulating T cells of UC patients [49]. GSK's investigation of GSK2831781 in a Phase II trial for UC patients. GSK licenced GSK2831781 from Immutep in 2010, as a depleting anti-LAG-3 antibody for development in autoimmune indications. Wherein GSK2831781 binds to LAG-3$^+$ cells and depleting them through antibody-dependent cellular cytotoxicity. GSK discontinued the Phase II trial due to lack of efficacy with little difference between placebo and those treated with GSK2831781 [50]. Unlike depleters, IMP761 will not deplete cells critical to immune homeostasis that express high levels of LAG-3 specifically, Tregs and Tr1 cells.

As above, moderate to severe patients that do not respond to conventional therapy move on to biologics, however these tend to have a poor safety profile associated with the risk of serious infection and malignancy. Patients typically begin on an anti-TNFα such as Humira and move to anti-IL-12/IL-23 if they don't respond. If the patient is refractory they may move onto an anti-integrin. As with RA, IMP761 may be able to differentiate on both safety and efficacy. Rinvoq has shown some of the best efficacy of approved therapeutics in UC with 26% of patients achieving clinical remission. Although the indication is competitive there is room for additional mechanisms of action and improved efficacy.

[44] Le Berre, C., et al. (2023) Ulcerative Colitis, The Lancet, 40(10401):12-18.
[45] National Institute Of Diabetes And Digestive and Kidney Diseases. Definition & Facts of Ulcerative Colitis (https://www.niddk.nih.gov/health-information/digestive-diseases/ulcerative-colitis/definition-facts#:~:text=Does%20ulcerative%20colitis%20have%20another,of%20Jewish%20descent4) Accessed 05/2025.
[46] Cleveland Clinic. Crohn's Disease (https://my.clevelandclinic.org/health/diseases/9357-crohns-disease) Accessed 05/2025.
[47] Lightner, A., et al. (2017) The surgical management of inflammatory bowel disease, Current Problems In Surgery. 54(4):172-250.
[48] Uniken Venema, W., et al. (2019) Single-Cell RNA Sequencing of Blood and Ileal T Cells From Patients With Crohn's Disease Reveals Tissue-Specific Characteristics and Drug Targets, Gastroenterology, 156(3):812-815.
[49] Slevin, S., et al. (2020) Lymphocyte Activation Gene (LAG)-3 Is Associated With Mucosal Inflammation and Disease Activity in Ulcerative Colitis, Journal Of Crohn's and Colitis. 14(10):1446-1461.
[50] D'Haens, G., et al. (2023) A randomised, double-blind, placebo-controlled study of the LAG-3-depleting monoclonal antibody GSK2831781 in patients with active ulcerative colitis, Alimentary Pharmacology And Therapeutics. 58(3):283-296.

Challenges and Risks

While the therapeutic potential of LAG-3 agonists in autoimmune diseases is promising, there are several challenges and limitations that must be addressed before they can become widely adopted in clinical practice.

- Heterogeneity Of Autoimmune Diseases: Autoimmune diseases are highly heterogeneous, with distinct pathophysiological mechanisms that vary between different conditions. The role of LAG-3 in immune regulation may not be uniform across all autoimmune diseases, and the effectiveness of LAG-3 agonists may differ depending on the specific disease. Therefore, it will be crucial to identify which autoimmune conditions and patient populations are most likely to benefit from LAG-3 agonist.

- Mechanism Of Action: Although we understand the inhibitory receptor function of LAG-3 we are yet to fully elucidate the MOA. We see some contradictory studies wherein LAG-3 expression on Tregs supports their suppressive function[51] or LAG-3 activation led to reduced IL-10-producing Tregs [52]. Intriguingly, recent studies have indicated the cytokine milieu may affect sLAG-3 and may be a critical for indication selection.[53]

- Balancing The Immune Response: suppressing the immune system can lead to an increased risk of infections and malignancies as seen with other biologics. IMM are trying to achieve a clean safety profile with limited circulating LAG-3$^+$ cells in select autoimmune diseases.

- Long-term Safety: Although early clinical trials have suggested that LAG-3 agonists are well-tolerated, the long-term safety of these drugs remains uncertain.

- Regulatory And Manufacturing Challenges: As with all biologic therapies, the development, approval, and manufacturing of LAG-3 agonists face significant regulatory hurdles. These drugs must undergo rigorous testing to ensure their safety, efficacy, and consistency before they can be approved for widespread use. Additionally, the complexity of biologic drug production and the associated costs can pose challenges in terms of accessibility and affordability, particularly in resource-limited settings. The cost-effectiveness of LAG-3 agonists will need to be carefully evaluated, especially considering that many autoimmune diseases are chronic and require long-term therapy.

Conclusion

The potential for LAG-3 agonists to serve as a new class of drug for the treatment of autoimmune diseases is an exciting development. Limiting the autoreactive immune response through LAG-3 agonists offers a targeted and potentially safer alternative to conventional immunosuppressive therapies. Unlike current treatments that broadly suppress immune function with increase the risk of infections and malignancies, LAG-3 agonists have the potential to suppress activation and proliferation of LAG-3$^+$ T cells.

The benefits of LAG-3 agonists could be particularly pronounced in autoimmune diseases driven by T cell dysregulation, such as rheumatoid arthritis and inflammatory bowel disease, where existing treatments often fail to achieve sustained remission or require long-term use with significant side effects. Moreover, LAG-3 agonists may also offer opportunities for combination therapy, enhancing the effectiveness of existing biologics.

Despite promising preclinical and early clinical data, several challenges remain, including understanding the full range of autoimmune diseases that could benefit from LAG-3 agonism, ensuring long-term safety, and addressing regulatory and manufacturing complexities. As trials progress and more data become available, LAG-3 agonists could represent a transformative approach to the management of autoimmune diseases, offering patients improved outcomes and a better quality of life.

[51] Gertel, S., et al. (2022) Lymphocyte activation gene-3 (LAG-3) regulatory T cells: An evolving biomarker for treatment response in autoimmune diseases, Autoimmunity Reviews, 21(6):103085.
[52] Chen, K., et al. (2023) FGL1-LAG-3 axis impairs IL-10-Producing regulatory T cells associated with Systemic lupus erythematosus disease activity, Heliyon, 9(10):20806.
[53] Tian, J., et al. (2024) Expression of lymphocyte activation gene-3 on CD4+T cells is regulated by cytokine interleukin-18 in myasthenia gravis, Journal of Neuroimmunology, 388:578308

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