

06

Investor Update

APRIL 2021

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MARC VOIGT

Message from the CEO

The last few months have certainly been a very busy period, not only for Immutep but for our whole sector. I would like to take the opportunity in our newsletter to add a bit more colour to the significance of Bristol Myers Squibb's (BMS) first validation of a LAG-3 product, and to elaborate on our second collaboration with MSD and our partnership with LabCorp to help develop immuno-oncology diagnostics.

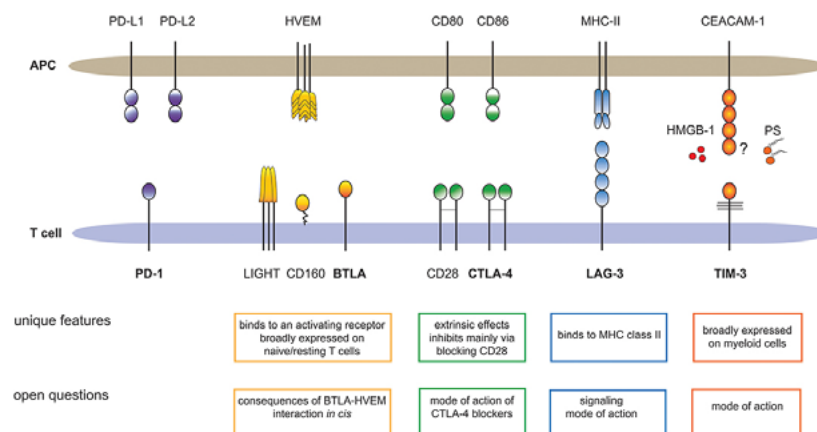
From an industry perspective, March 2021 delivered the news we had all been anticipating for years: the validation of LAG-3 as the next emerging immunotherapy target.

This validation came in the form of **BMS's announcement** that its Phase II/III trial evaluating the combination of relatlimab, an anti-LAG-3 antibody, and Opdivo, an approved anti-PD-1 blockbuster drug, in melano-ma patients had met its primary endpoint of progression-free survival (PFS). In other words, they reported the combination immunotherapy was helping patients.

BMS intends to release the details of the results at an industry conference soon. However, the top line results are enough to confirm the interaction between LAG-3 and MHC II (the ligand that LAG-3 naturally binds to) can be manipulated to help the body's immune system fight cancer (See Diagram 1).

This is a significant milestone for cancer immunotherapy. We can now expect LAG-3-related products to be featured prominently with more interest from partners and investors. LAG-3 is advancing quickly to become the next emerging target for immunotherapy behind PD-1 and CTLA-4.

Diagram 1. Current immunotherapy targets including PD-1 and CTLA-4, which have approved therapeutic products available for patients, plus LAG-3 which uniquely binds to the MHCII complex of an antigen presenting cell.¹



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¹ https://www.frontiersin.org/files/Articles/402607/fimmu-09-01909-HTML/image_m/fimmu-09-01909-g001.jpg

Message from the CEO

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BMS's news is especially significant for the LAG-3 industry. It represents further validation of LAG-3, an immune checkpoint that has been attracting increasing interest over the years, as can be seen by the exponentially increasing rate of publications shown at Diagram 2.

Immutep's CSO and CMO, Prof Frédéric Triebel, discovered LAG-3 in 1990 and has spent the last 30 years working in the field. In the interview below entitled "Validation of LAG-3", Prof Triebel reflects on his journey with LAG-3.

Immutep remains the only LAG-3 pure-play with more programs around LAG-3 than anyone else in the field, including big pharma.

Within the exciting and growing LAG-3 space, Immutep continues to be the worldwide leader in developing LAG-3 therapeutics. We are contributing four product candidates which are being evaluated in multiple clinical trials. No other company has such depth of pipeline or knowledge. This is one of the key reasons LabCorp partnered with us to develop LAG-3 diagnostic tests.

In addition, we have the only MHC II agonist product candidate in development, known as efiti. Efiti is an immune booster that has the versatility to be paired with PD-1 (or PD-L1) antagonists in combination therapies. Notably, efiti has a very different and unique mode of action from anti-LAG-3 products such as relatlimab or leramilimab (LAG525) and this has several advantages that broaden our opportunity (See Two Modes of Action for LAG-3 article). It also puts us in a category with no current competition (see Overview of the LAG-3 Landscape at Diagram 3), while being part of practically every field of LAG-3 related therapies i.e. with an agonist soluble LAG-3 protein and an antagonist antibody for oncology and an agonist and a depleting antibody for autoimmune diseases.

We were also happy to inform you in mid-March 2021 that Immutep had deepened our relationship with MSD through a [second collaboration](#) with them. MSD (known as Merck & Co. in the USA) is an American multinational pharmaceutical company and one of the largest pharmaceutical companies in the world.

As you can imagine, this new collaboration involved much work from our team and I would like to thank them for their steadfast commitment. We have certainly become a very effective team, having successfully secured multiple partnerships already (see our pipeline as summarised in the Operational Snapshot section). Of course, there is always more to do on that front, and I can assure you that we always look to the future for further opportunities to build the value of Immutep!

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Message from the CEO

[Continued from p. 3]

Two Modes of Action for LAG-3

The first mechanism of action relates to **antagonist** LAG-3 drugs, such as relatlimab (BMS) or leramlimab (LAG525 from our licensee Novartis). These drugs are antibodies that bind to the naturally occurring LAG-3 proteins on T cells, blocking anything else from binding. When these antagonist drugs, also known as anti-LAG-3 drugs, are bound, the LAG-3 receptor on activated T cells can't bind MHC II molecules expressed by antigen presenting cells (APC) such as dendritic cells. LAG-3 is an inhibitory receptor on T cells; when LAG-3 is engaged by MHC II, it down-regulates T cell activation. Therefore, blocking the MHC II-LAG-3 interaction is analogous to releasing the brakes on the activated T cells.

Antagonists need to be given to patients at higher doses to ensure the drug can bind to all LAG-3 molecules all the time to completely block the MHC II-LAG-3 interaction. From a medical perspective, the recent BMS announcement validates the physiological importance of this MHC II-LAG-3 interaction as the simple blockade of this interaction at the tumor site or in the draining lymph node leads to clinical benefit.

Efti, which is a soluble recombinant LAG-3 protein, is not an anti-LAG-3 therapy. Its mode of action is as an MHC II agonist. It is an APC activator.

Agonists such as efti can be given at lower doses than antagonists, because they need to only bind to a few percent of MHC II receptors to trigger APC activation which, is the first step of pushing the accelerator on the immune system. Indeed, activated APCs like dendritic cells physiologically instruct the T cells what to do in terms of antigen specificity (they present antigenic peptides to T cells), direction of the immune response (anergy versus proliferation) or functionality (they give the CD8 T cells the "license to kill" signal, so that the latter cells become more cytotoxic).

See our *Mode of Action* video for further explanation of LAG-3 immunotherapy.
<https://www.youtube.com/watch?v=6EISCGYAGQw>



VALIDATION OF LAG-3



Prof Frederic Triebel is Immutep's CMO/CSO and the discoverer of the LAG-3 gene. Talking to him from his home in Normandy, France, where he is in lock down, he reflects on the discovery of LAG-3 and the significance of its recent validation after a lifetime in the pursuit of LAG-3 knowledge for the benefit of patients.

Quick Glossary

Agonists are molecules that bind to a receptor and activate the receptor to produce a biological response.

Alleles are one of two, or more, forms of a given gene variant.

Antagonists are a type of molecule that blocks a receptor-ligand interaction and the associated biological response.

Antigen presenting cells (APCs) process and present antigens for recognition by T cells. They give instructions and a license to kill to CD8 T cells.

Antigens are molecules on the outside of a pathogen cell. Cancer cells have tumour antigens.

MHC class II are a class of major histocompatibility complex (MHC) molecules.

T cells destroy foreign cells (including tumour cells) in the body when given instructions from an APC.

When did you first know that LAG-3 was an important protein in the body's immune system?

Back in 1988, I was working at Institut Gustave Roussy, south of Paris and cloned mRNA specifically expressed in human T cells after their activation, therefore named Lymphocyte Activation Gene-1, -2, -3. LAG-3 was first published in May 1990 and interestingly was found to be related phylogenetically to CD4, a known ligand for MHC class II (MHC II) molecules.

This was exciting because we already knew MHC II was intricately involved in many aspects of the body's immune response. MHC II genes and molecules are related to a multitude of different diseases. For instance, certain MHC II alleles are strongly associated with the risk of inheriting Type I diabetes.

What was the core question that drove your research for more than 30 years?

Because we understood the power of CD8 T cells to destroy tumours in animal models and the role of the

[Continued on p. 6]

VALIDATION OF LAG-3

[Continued from p. 5]

question: how could we get T cells to destroy cancer cells in patients with advanced disease?
I knew the answer lay in part in the interaction between MHC II and LAG-3, that is, bridging professional APCs (dendritic cells) to activated T cells.

What is special about BMS's news?

It shows us we are all “barking up the right tree” looking into the interaction between MHC II and LAG-3. It demonstrates the interaction is important in the immune system and it can be used to destroy cancer cells.

In oncology, this interaction can be leveraged in two ways, via two modes of action.

What are the two different modes of action?

In this MHC II/LAG-3 interaction, one can use a soluble LAG-3 protein to “push the accelerator” (an agonist action) on the MHC II molecules in order to activate APCs (and subsequently CD8 killer T cells) or one can use a blocking anti-LAG-3 antibody to stop MHC II engaging LAG-3 receptors on T cells (an antagonist action), thereby leading directly to more activated T cells.

Both modes of action can be leveraged to restore the immune system's ability to find and fight cancer cells but probably in different patient populations. Patients with so-called “cold or tepid” tumors (the majority of patients, with few activated T cells at the tumor site) need APC activation via efiti to induce more T cells to recognise effectively the tumour cells while “hot” tumor patients, unfortunately the minority, with a strong natural tumour bed infiltration by activated T cells could benefit from the antagonist effect, releasing the brakes on the T cells using an anti-LAG-3 antibody. (For more detail see Two Modes of Action for LAG-3).

Were there any moments along the LAG-3 journey where you had doubts?

I have always believed in LAG-3. However, there were times in the industry where it was difficult to convince others of LAG-3's potential. For a long time, chemotherapy was the number one treatment for many cancers, including breast cancer. The higher the dose, the more effective the chemo. The trouble was, it is very toxic and not very effective in many other indications.

Still, it was difficult to shift the paradigm. Then cancer vaccines came along. They were very hard to develop and there were many failures. They were safe, but they didn't work. This made the oncology world less open to new ideas. And then from 2010 onwards, the change came gradually when oncologists used anti-CTLA-4 and anti-PD-1 antibodies in randomised clinical trials. These antibodies are not anti-cancer agents per se. CTLA-4 or PD-1 are not expressed by tumour cells. They just unleash the power of the natural pre-existing T cell responses specific for tumor antigens. The anti-cancer agents are the patient CD8 T cells themselves that could get rid of massive metastatic tumour lesions in a few weeks. In short, we moved from a small field of cancer immunotherapy experimentations with early phase clinical trials to a new full-fledged medical science, named immuno-oncology.

Have there been sacrifices, personally or professionally along the way?

Absolutely! I wanted to focus completely on LAG-3, so I started Immutep with John Hawken in September 2001. It was just after the attack on the Twin Towers in New York. It was a hard time to start a new biotech, very difficult to raise cash in Europe due to the war in Iraq.

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VALIDATION OF LAG-3

[Continued from p. 6]

I left my secure clinical job at the Institut Gustave Roussy, south of Paris, in 2002 and then quit my teaching job as a Professor at the Paris-Sud university in 2004 and have also been the first investor, risking the financial wellbeing of my family at that time. *I burned my vessels on the beach like Agamemnon!*

Now that LAG-3 has been validated, what will happen next in the LAG-3 space?

We have been waiting for BMS's results for the last three years, ever since they completed their phase II trial. We have never known the results of the earlier phase II trial, but obviously they were strong enough for BMS to decide to proceed with the phase III.

I expect there will be more news coming from BMS which has 38 trials running in different indications. We will also have a flurry of results from others in the space. It will go from being a secret with unknown materiality, to an open race to the finish line! Of course, Immutep and its many collaborators will significantly contribute to the LAG-3 related developments as well!

Where do you see LAG-3 10 years from now?

There is lots of work to be done to fully appreciate the significance of the MHC II/LAG-3 interaction not only in immuno-oncology but also in autoimmunity and infection. We have a good chance of success in autoimmune diseases with IMP761 (a LAG-3 agonist antibody), and in oncology and infectious diseases with efiti.

More broadly, I believe immunotherapies will be expanded beyond cancer and clearly defined autoimmune diseases, into new fields. We can use the new tools we have in immuno-oncology in other therapeutic fields where we don't quite understand the implication of T cell aggressiveness, like arteriosclerosis or Parkinson disease (LAG-3 clearly plays a role there) or Alzheimer's disease. It is truly exciting!!

LAG-3 LANDSCAPE

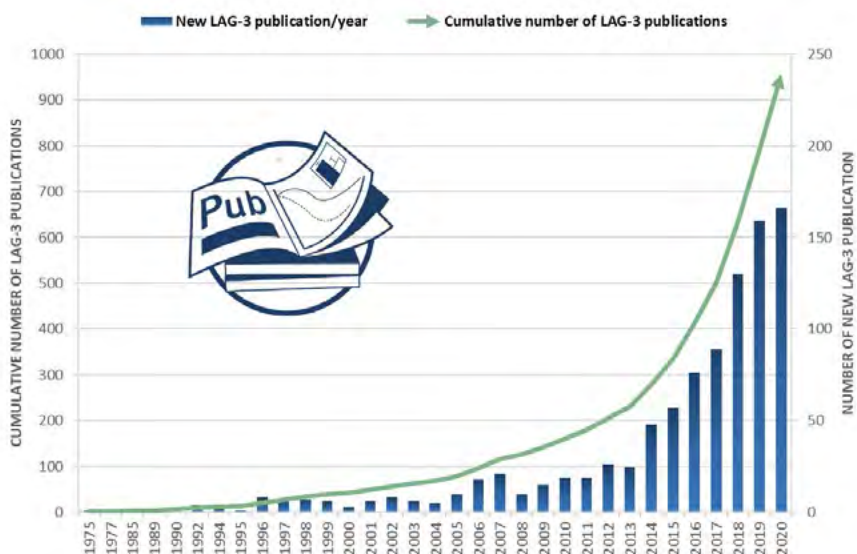


Diagram 2.
Acceleration in
the LAG-3 space.
Source: PubMed

[Continued on p. 8]

VALIDATION OF LAG-3

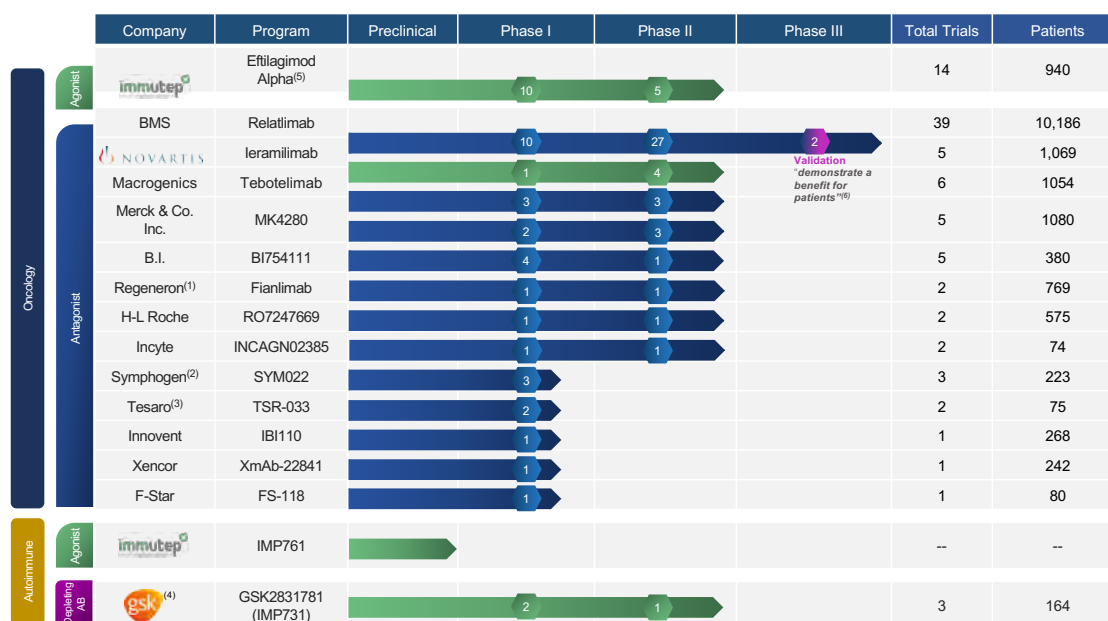
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Interest and investment in LAG-3 therapeutics has continued to accelerate over the last year. There are now more than 91 clinical trials evaluating 15 LAG-3 product candidates for more than 17,000 patients (See diagram 3).

Immutep and our collaboration partners have contributed to 22 trials for more than 2,000 patients. Apart from Immutep's candidates, all other LAG-3 candidates shown at Diagram 3 are all anti-LAG-3 antagonist antibodies with similar mechanisms of action being evaluated in cancer trials. Immutep's IMP761 and IMP731 (licensed to GSK) are being developed for autoimmune diseases.

Many pharmaceutical companies have entered the space and are rapidly advancing promising product candidates, including BMS, Roche, Boehringer Ingelheim, plus our existing partners Novartis, MSD and GSK and more than 30 others. Still, Immutep has an excellent and leading position in the field! However, Immutep continues to have the only MHC II agonist in late-stage clinical development, as shown below (Diagram 3).

Diagram 3. The LAG-3 therapeutic landscape.



Sources: GlobalData, Company websites, clinicaltrials.gov, and sec.gov, as of April 2021. The green bars above represent programs conducted by Immutep &/or its partners. Total Trials includes all active, completed &/or inactive trials. Patient totals are based on estimated total enrolled &/or to be enrolled. The above is not a complete list of currently existing LAG-3 products.

¹ As of January 7, 2019 Regeneron is in full control of program and continuing development (https://www.sec.gov/Archives/edgar/data/872589/000110465919000977/a19-1325_18k.htm)

² On April 3, 2020 Les Laboratoires Servier acquired Symphogen

³ Tesaro was acquired by and is now part of GSK (www.gsk.com/en-gb/media/press-releases/gsk-completes-acquisition-of-tesaro-an-oncology-focused-biopharmaceutical-company/)

⁴ Includes two completed Phase I studies and one discontinued Phase 2 study

⁵ Including IITs, two planned trials (MBC trial by EOC and HNSCC trial)

⁶ RELATIVITY-047 (<https://investors.bms.com/iframes/press-releases/press-release-details/2021/Bristol-Myers-Squibb-Announces-RELATIVITY-047-a-Trial-Evaluating-Anti-LAG-3-Antibody-Relatlimab-and-Opdivo-nivolumab-in-Patients-with-Previously-Untreated-Metastatic-or-Unresectable-Melanoma-Meets-Primary-End-point-of-Progression-Free-Survival/default.aspx>)



PARTNERSHIP PROGRESS CONTINUES

Second Collaboration with MSD

In mid-March 2021, we were delighted to deepen our relationship with MSD (Merck & Co) through a second clinical trial collaboration and supply agreement.

Called TACTI-003, the new trial evaluates efiti in combination with MSD's KEYTRUDA® (pembrolizumab) as a first line therapy in unresectable recurrent or metastatic Head and Neck Squamous Cell Carcinoma (HNSCC) patients.

This is the same combination therapy already being tested in Immutep's ongoing Phase II TACTI-002 study (also designated KEYNOTE-798) and is delivering very promising results in second line HNSCC.

Planning for the new trial is progressing well and we expect to enroll the first patient in the study in mid-2021.

New Agreement with LabCorp

In late October, we started a new collaboration with the Laboratory Corporation of America Holdings, known as LabCorp. Working with LabCorp, we will support the development of immuno-oncology products or services. This work enables Immutep to enter the immuno-oncology diagnostics field.

Interestingly LabCorp and Bristol Myers Squibb have an abstract at AACR 2021, discussing the expression of LAG-3 in different cancer types. It shows how closely the industry works together.

Our deep LAG-3 pipeline and highly experienced team reflects a significant amount of know-how in immuno-oncology, specifically related to LAG-3.



FINANCING

Demonstrating strong interest and confidence in Immutep's clinical program from existing and new high-quality investors, we completed a A\$29.6 million placement in November 2020. Australian Ethical, Perennial Value Management, Regal Funds Management, Firetrail Investments and US-based, Ridgeback Capital were all leading participants in the capital raise.

In addition, in December 2020, the Company received \$10.66 million from the exercise of warrants over American Depository Shares.

The proceeds will drive the ongoing development of our promising immuno-oncology and autoimmune programs. Immutep now has a strong cash runway, into calendar year 2023 and beyond several significant data read-outs.

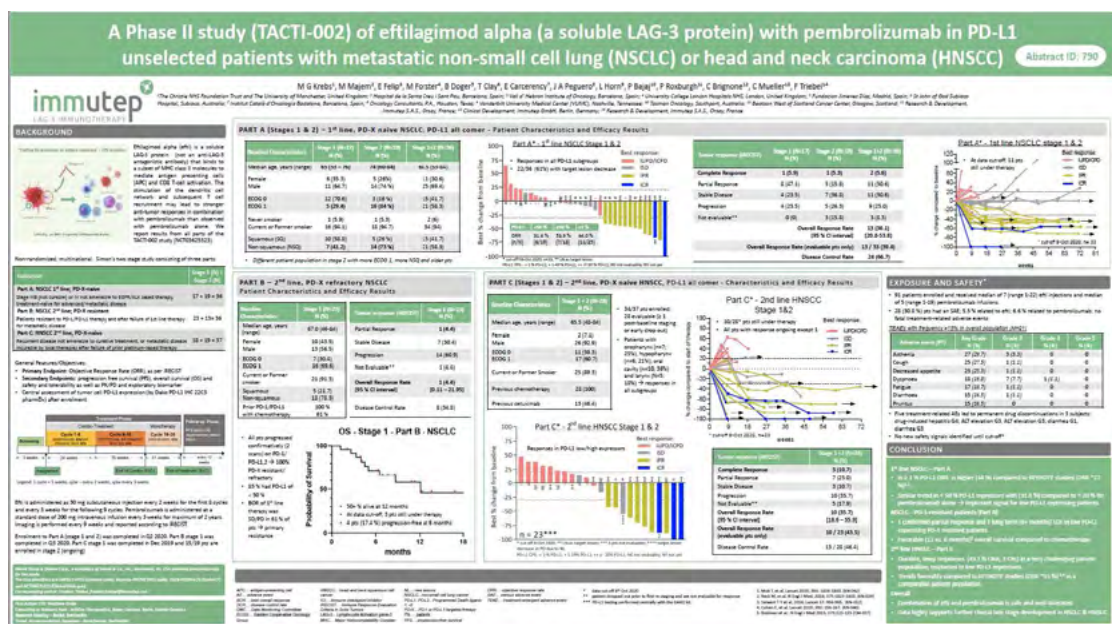


INDUSTRY CONFERENCES

Immutep was pleased to announce new data from our TACTI-002 phase II clinical trial (also designated KEYNOTE-798) at the **Society for Immunotherapy of Cancer (SITC) 35th Anniversary 2020 Annual Meeting** in November. More mature data from the trial was accepted as a late breaker poster presentation and poster walk for highly scored abstracts.

The data showed Overall Response Rates (ORR) in 1st line Non-Small Cell Lung Cancer (NSCLC) and 2nd line HNSCC had continued to be very favourable. Five patients demonstrated a Complete Response (a complete disappearance of all tumour lesions): two patients were in the 1st line NSCLC group and three patients were in the 2nd line HNSCC group. The results also demonstrated encouraging efficacy in low PD-L1 expressing patients who do not typically respond to anti-PD-1 therapy.

SITC is the world's leading member society of medical professionals dedicated to advancing cancer immunotherapy and biological therapy.



A month later, in December 2020, Immutep presented data from our phase IIb APIAC trial in metastatic breast cancer at the **San Antonio Breast Cancer Symposium (SABC)**. The data was selected for a spotlight presentation and showed a statistically significant survival benefit for key patient groups.

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INDUSTRY CONFERENCES AND POSTER PRESENTATIONS

[Continued from p. 11]

Specifically, a statistically significant Overall Survival (OS) benefit was seen in the efi group in pre-defined patient groups. Patients under the age of 65 years showed a +7.1 months survival benefit (median of 21.9 vs. 14.8 months, nearly 50% longer) from efi with chemotherapy, compared to chemotherapy plus placebo (p=0.012). A +9.4 months survival benefit (median of 22.4 vs. 12.9 months, 74% longer) was seen from efi with chemotherapy for patients with a low starting monocyte count (p=0.02).

A statistically significant increase in CD8 T cells (key immune cells) was observed in patients treated with efi plus chemotherapy and this increase correlated with prolonged OS, indicating pharmacodynamic activity and proof of concept of efi's mode of action.

In addition, a promising and improving overall trend in OS in the total population was observed, based on approximately 60% of events. A median survival benefit of +2.7 months from the efi plus chemotherapy group was reported.

The results were the first time an antigen presenting cell (APC) activator has shown an OS benefit in a randomised setting in metastatic breast cancer patients known to be insensitive to immune checkpoint inhibitor therapy. The collection of OS is data ongoing and final data is expected to be reported mid-2021.

SABC is a leading international conference focused on clinical, translational and basic research in breast cancer. It is attended by a broad international audience of academic and private researchers and physicians from over 90 countries.

Following the encouraging results presented at SITC and SABC, we have prioritised the commencement of **scaling up the manufacturing efi**.

Primary efficacy results from LAPAC: A double-blinded, placebo controlled, randomized multinational phase IIb trial comparing weekly paclitaxel plus eftiligidom alpha (soluble LAG-3 protein) vs. weekly paclitaxel plus placebo in HR-positive metastatic breast cancer patients

BACKGROUND
 Eftiligidom alpha (efi) is a soluble LAG-3 protein. It is a soluble LAG-3 protein that binds to a subset of MHC class II molecules to modulate antigen presentation. The combination of paclitaxel and efi is expected to enhance anti-tumor immune response in combination with paclitaxel. Phase IIb study in HR-positive metastatic breast cancer patients. The study compares the combination of weekly paclitaxel plus efi (200 mg) or weekly paclitaxel plus placebo.

TRIAL DESIGN
 Multicenter, double-blind, double-blind, 2:1 randomized phase IIb study in breast cancer patients. Primary endpoint: Overall Survival (OS). Secondary endpoints: Progression-Free Survival (PFS), Time to Next Treatment (TTNT), Health-Related Quality of Life (HRQL).

PATIENT DISPOSITION AND EXPOSURE
 In total, 317 patients were randomized to efi (200 mg) and to placebo (200 mg). All patients were treated for at least 1 treatment and were included in the full analysis and safety population.

Group	Randomized	Completed	Discontinued
efi (200 mg)	209 (66.2%)	192 (91.9%)	17 (8.1%)
placebo (200 mg)	108 (33.8%)	97 (90.0%)	11 (10.0%)



EFFICACY - OVERALL POPULATION
 OS: 21.9 months (95% CI 19.2, 24.6) vs 14.8 months (95% CI 12.9, 16.7), p=0.012. HR: 0.55 (95% CI 0.41, 0.74).
 PFS: 10.2 months (95% CI 8.8, 11.6) vs 8.8 months (95% CI 7.8, 9.8), p=0.001. HR: 0.78 (95% CI 0.68, 0.89).
 TTNT: 12.1 months (95% CI 10.8, 13.4) vs 10.8 months (95% CI 9.8, 11.8), p=0.001. HR: 1.12 (95% CI 1.04, 1.21).

EFFICACY - SUBGROUPS
 OS: 21.9 months (95% CI 19.2, 24.6) vs 14.8 months (95% CI 12.9, 16.7), p=0.012. HR: 0.55 (95% CI 0.41, 0.74).
 PFS: 10.2 months (95% CI 8.8, 11.6) vs 8.8 months (95% CI 7.8, 9.8), p=0.001. HR: 0.78 (95% CI 0.68, 0.89).
 TTNT: 12.1 months (95% CI 10.8, 13.4) vs 10.8 months (95% CI 9.8, 11.8), p=0.001. HR: 1.12 (95% CI 1.04, 1.21).

CONCLUSION
 The combination of weekly paclitaxel plus efi (200 mg) significantly improved OS, PFS, and TTNT compared to weekly paclitaxel plus placebo in HR-positive metastatic breast cancer patients.



OPERATIONAL SNAPSHOT

Oncology	Eftilagimod Alpha (efti or IMP321)	AIPAC Metastatic Breast Cancer (Chemo – IO)	Phase IIb		Global Rights 
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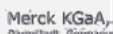

Overall Survival (OS) data from the AIPAC clinical trial were presented at SABCS 2020 (see Industry Conferences for a summary of the results). The collection of OS data is ongoing and final data is expected to be reported mid-2021.

Oncology	Eftilagimod Alpha (efti or IMP321)	TACTI-002 (KEYNOTE-798) Non-Small-Cell Lung Carcinoma (IO – IO) Head and Neck Squamous Cell Carcinoma (IO – IO)	Phase II		Global Rights 
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More mature and encouraging results from ImmuteP's TACTI-002 trial were reported at SITC in November 2020. Since SITC, ImmuteP has completed the recruitment of patients with 2nd line HNSCC and started the recruitment of an additional cohort of 1st line NSCLC patients in January 2021. Further interim data from TACTI-002 is expected in H1 of 2021.

Oncology	Eftilagimod Alpha (efti or IMP321)	TACTI-003 Head and Neck Squamous Cell Carcinoma (IO – IO)	Phase II		Global Rights 
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ImmuteP is progressing well with plans to start the study in mid-2021.

Oncology	Eftilagimod Alpha (efti or IMP321)	INSIGHT-004 Solid Tumors (IO – IO)	Phase I	 	Global Rights 
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In September 2020, ImmuteP reported improving data from the INSIGHT-004 trial. 41.7% of patients showed a Partial Response to the combination therapy of efti and avelumab, building on the previously reported result of 33% showing a Partial Response.

Encouraging early anti-tumour activity signals were observed in a variety of cancer indications not typically responsive to immunotherapy. Importantly, the combination of efti and avelumab continues to be safe and well tolerated. Final data is expected in 2021.

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OPERATIONAL SNAPSHOT

[Continued from p. 13]

Oncology	Eftilagimod Alpha (efti or IMP321)	TACTI-mel Melanoma (IO – IO)	Phase I			Global Rights 
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The results of Immutep’s TACTI-mel trial were featured prominently in Issue 38 of the Melanoma Research Review which is a summary of the latest research in melanoma for health professionals. Health-care professionals can access the Review for free online at:

www.researchreview.com.au/au/Clinical-Area/Internal-Medicine/Medical-Oncology/Melanoma.aspx


Oncology	Eftilagimod Alpha (efti or IMP321)	Solid Tumors (Cancer Vaccine) YNP01 and YCP02	Phase I			Global Rights 
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CYTLIMIC has been conducting two studies (YNP01 and YCP02) with CYT001 which is CYTLIMIC’s lead cancer vaccine. CYT001 comprises peptides designed using artificial intelligence from the HSP30 and GCP-3 proteins, plus two adjuvants, efti and Hiltonol (Poly-ICLC). Under the terms of clinical collaboration, service and supply agreements with Immutep, CYTLIMIC has full responsibility for the continued development of the cancer vaccine and Immutep is eligible to receive development-based milestone payments.

Oncology	Eftilagimod Alpha (efti or IMP321)	Metastatic Breast Cancer (Chemo – IO)	Phase I			Chinese Rights 
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In December 2020, EOC Pharma announced its plans to conduct a new phase II trial of efti in metastatic breast cancer. This follows Immutep’s announcement of encouraging first OS data from its Phase IIb study, AIPAC (see above).

EOC Pharma is the exclusive licensee of efti for the Chinese market.

Infectious Disease	Eftilagimod Alpha (efti or IMP321)	COVID-19	Phase II			Global Rights 
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The University Hospital Pilsen in the Czech Republic has commenced an investigator-initiated randomised phase II clinical trial evaluating efti in up to 110 hospitalised patients with COVID-19. Recruitment of patients commenced in October 2020.

The trial aims to boost a patient’s immune response to prevent the patient from developing severe COVID-19 symptoms that require intensive care and can lead to respiratory failure and death. As an antigen presenting cell (APC) activator, efti could help to control the viral load in hospitalised patients by boosting T cells.

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OPERATIONAL SNAPSHOT

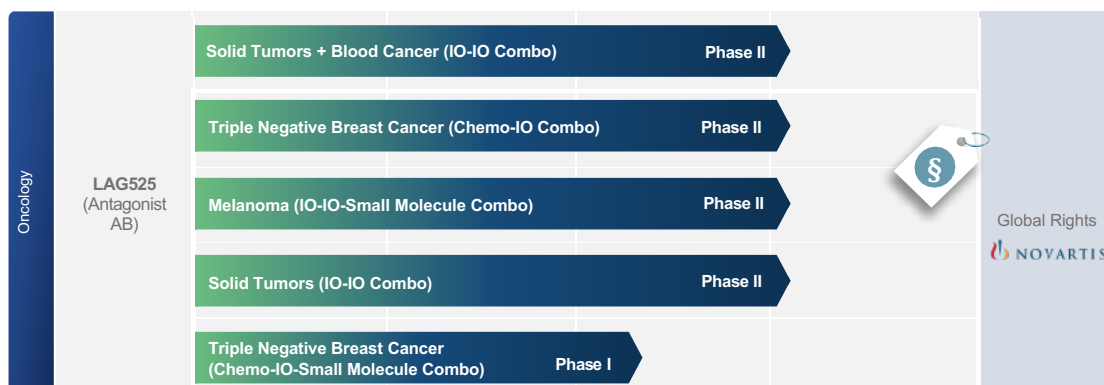
[Continued from p. 14]

Initial results from the safety run in of the trial were reported in January 2020 and showed all six patients received the three planned 10 mg efi injections and were discharged from hospital. No adverse events were reported. An independent review of this safety run-in data prompted the recommendation to proceed with enrolment for the randomised portion of the EAT COVID study.

Initial interim efficacy results are expected to be reported before the end of the year.



Immutep is continuing cell line and other preclinical development activities for IMP761 in preparation for clinical trials.



LAG525, now called leramilimab, a humanised form of IMP701 is being evaluated by Novartis in several Phase I and/or Phase II clinical trials in combination with Novartis' PD-1 inhibitor spartalizumab for the treatment of certain cancers.

Novartis has full responsibility for the continued development of the LAG-3 antibody program and Immutep is eligible to receive development-based milestone payments and sales-based royalties.

[Continued on p. 16]

OPERATIONAL SNAPSHOT

[Continued from p. 15]



In January 2021, GSK stopped its phase II clinical trial evaluating GSK2831781 (derived from Immutep’s IMP731 antibody) in ulcerative colitis based on the assessment of clinical data as part of a planned interim analysis conducted in consultation with the trial’s Data Review Committee. GSK is conducting further reporting, assessment and analyses of the efficacy and safety data and evaluating the biology to determine next steps for the GSK2831781 development program.

Importantly, Immutep’s collaboration with GSK remains in place and GSK2831781 continues to be under an exclusive license with GSK. GSK continues to have full responsibility for the development of this LAG-3 antibody program and Immutep is eligible to receive development-based milestone payments and sales-based royalties.

The discontinuation of the GSK trial has no impact on Immutep’s three other product candidates all of which have different mechanisms of action, including efti. Immutep’s cash runway is also unimpacted.



OUTLOOK

Immutep is advancing strongly towards another step change in 2021. We continue to be in a very strong financial and operational position, following the encouraging clinical results announced for efti in 2020.

We have increasing confidence in efti and accordingly, over the last six months we have announced or commenced three new efti trials, or trial extensions, with up to 386 patients in different cancer indications.

Our business development activity has also advanced with a second collaboration with MSD and a new LAG-3 agreement with LabCorp.

Engagement with regulatory bodies remains a key priority, along with strengthening our IP position and upscaling the manufacturing of efti to prepare for the future. This has already resulted in efti receiving Fast Track designation in 1st line HNSCC from the US FDA, given its encouraging data and potential to meet an unmet medical need.



COMPANY CALENDER

What's next

JUNE 4TH 2021 - JUNE 8TH 2021

ASCO 2021 Annual Meeting

Immutep will participate in this year's virtual ASCO 2021 Annual Meeting.

<https://meetings.asco.org/am/virtual-welcome>

JUNE 10TH 2021 - JUNE 18TH 2021

BIO International Convention - Bio Digital 2021

Immutep will participate in the new virtual event format.

<https://www.bio.org/events/bio-digital>

JULY 13TH 2021 - JULY 15TH 2021

Non-Small Cell Lung Cancer (NSCLC) Drug Development Summit

Immutep will participate in the digital Non-Small Cell Lung Cancer (NSCLC) Drug Development Summit. Frédéric Triebel, CSO & CMO of Immutep speaks on the subject: A Soluble LAG-3 Protein (Eftilagimod Alpha) with an Anti-PD-1 Antibody (Pembrolizumab): Results of a Phase II Study in NSCLC

<https://nslc-drug-development.com/>

SEPTEMBER 17TH 2021 - SEPTEMBER 21ST 2021

ESMO 2021 Congress

Immutep will participate in the virtual ESMO 2021 congress.

<https://www.esmo.org/meetings/esmo-congress-2021>



NOVEMBER 10TH 2021 - NOVEMBER 14TH 2021

SITC - 36th Annual Meeting & Pre-Conference Programs (SITC 2021)

The company will have presentations at the conference and representatives will be attending.

Venue: Walter E. Washington Convention Center in Washington, D.C.



IMMUTEP

Fast Facts

Listings

Australian Securities Exchange (ASX), NASDAQ

Stock Codes

ASX: IMM, NASDAQ: IMMP

Issued Capital – Ordinary Shares

672.4 million (as of 12 April 2021)

Market Capitalisation

A\$299.2 million (US\$227.5 million)
(as of 12 April 2021)

Cash & Term Deposits

~A\$54.9 million (~US\$42.3 million) (as of 31
December 2020)

Board of Directors

Russell J Howard, PhD

Non-executive Chairman

Mr Marc Voigt

Executive Director and Chief Executive Officer

Mr Pete A Meyers

Non-executive Director

Grant Chamberlain

Non-executive Director

Senior Management

Prof Dr Frédéric Triebel

Chief Medical Officer and Chief Scientific Officer

Deanne Miller

Chief Operating Officer, General Counsel and
Company Secretary

www.immutep.com

FOLLOW IMMUTEP'S PROGRESS

Immutep is dedicated to maintaining consistent and clear communications with our investors. In addition to our newsletter, we encourage our shareholders to continue following Immutep's progress in a number of ways:

 www.immutep.com

Our website is a treasure trove for those in search of details about our company, our management team, and archived information. We encourage everyone to check it out regularly.

 www.clinicaltrials.gov

Immutep registers all of our clinical trials, and the details of enrolling doctors, on the ClinicalTrials.gov website, a service of the United States National Institutes of Health. This register is the largest such repository of clinical trial information around the world.

Our ClinicalTrials.gov ID for our trials are as follows:

- TACTI-mel trial is NCT02676869
- AIPAC trial is NCT02614833
- TACTI-002 trial is NCT03625323
- TACTI-003 trial is NCT04811027

 **Twitter**

<https://twitter.com/Immutep>

 **Facebook**

<https://www.facebook.com/Immutep/>

 **LinkedIn**

<https://www.linkedin.com/company/857541/>

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This investor update was authorised for release by the CEO of Immutep Limited.