BRYAN, GARNIER & CO

13th January 2020

OSE IMMUNO

CORPORATE Coverage Initiated

CORPORATE	**EUR7 (+82%)**
Share price	EUR3.85
Bloomberg / Reuters	OSE FP/OSE.PA

Healthcare | **Biotech**

Winning by DEALing with early-stage programs

OSE Immunotherapeutics business model is based on the identification of promising immunotherapy targets for oncologic and autoimmune indications and their early-stage development, with subsequent out-licensing to a partner.

Importantly, OSE has a proven track record of sizable deals for preclinical assets, listing Boehringer Ingelheim (BI), Servier and Janssen Biotech among its partners. Within the company's oncology franchise, we emphasize a novel bispecific antibody platform, BiCKI, based on anti-PD-1 backbone. We currently expect the first BiCKI-derived asset, BiCKI IL-7, to be ready for clinics in 2021, highlighting for a potential partner. Considering the nature of recent deals in the bispecifics space and company's track record of early-stage partnership agreements, we believe that OSE has all cards in hands to secure another landmark deal, providing a significant catalyst for the stock. We also expect BI 765063, a novel checkpoint inhibitor that targets CD47/SIRPα and is out-licensed to BI, to generate next significant milestone payment in 2021 and reach the market in 2025, generating royalty stream for OSE.

Among autoimmune franchise, OSE-127 is being co-developed with Servier, and the full exercise of licensing option could potentially come in 2022. Additionally, in 2H20 – 1H21, OSE could secure another partnership agreement for FR104, a phase-II-ready immuno-suppressive therapy, in kidney transplantation.

In 1Q20, OSE is also expecting an interim readout from a phase III Atalante 1 study of Tedopi. While we are staying on the sidelines in our expectations, we believe it could clarify the future of the asset. Importantly, regardless Atalante 1 interim readout, we see multiple upsides for the stock in the near future. We value OSE at FV of EUR7.0 per share, which represents a significant upside to current share price.

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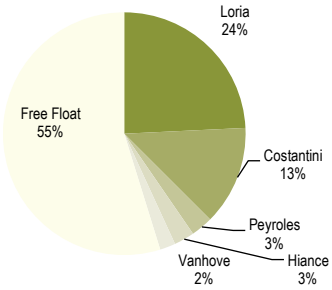
OSE IMMUNO

CORPORATE

Fair Value	**EUR7 (+82%)**
Share price	**EUR3.85**
Market Cap.	**EUR58m**
EPS 3Y CAGR	**68.3%**

Shareholders



Fiscal year end 31/12	2017	2018	2019e	2020e	2021e	2022e
Financial Summary						
EPS (EUR)	-0.68	0.37	-0.16	-0.82	0.04	4.48
Restated EPS (EUR)	-0.68	0.37	-0.16	-0.82	0.04	4.48
% change	-115.2%	-	-142.9%	-416.9%	-	10181.6
Net dividend (EUR)	0.00	0.00	0.00	0.00	0.00	0.00
Average yearly Price	-	3.81	3.74	-	-	-
Avg. Number of shares, diluted (m)	14.8	14.8	14.8	14.8	14.8	14.8
Historical Entreprise value (EURm)	-	-4.53	10.06	-	-	-
Valuation (x)						
EV/Sales	NM	-0.2x	2.62x	11.94x	2.89x	-0.09x
EV/EBITDA	NM	-0.9x	NM	NM	NM	-0.12x
EV/EBIT	NM	-0.9x	NM	NM	NM	-0.12x
P/E	NM	10.3x	NM	NM	88.33x	0.86x
Net dividend yield (%)	NM	0.0%	NM	NM	NM	NM
Profit & Loss Account (EURm)						
Revenues	0.0	24.5	16.4	4.9	20.0	94.1
Change (%)	-100%	266%	-33%	-70%	308%	371%
R&D	-10.0	-15.1	-19.0	-19.0	-19.0	-15.0
Adjusted EBITDA	-13.8	5.0	-6.2	-17.8	-2.7	75.4
EBIT	-13.8	4.8	-6.2	-17.8	-2.7	75.4
Change (%)	-1.2	-	-2.3	-1.9	-0.8	-
Financial results	0.1	-0.1	0.1	0.1	0.1	0.1
Pre-Tax profits	-13.7	4.7	-6.1	-17.7	-2.6	75.5
Exceptionals	0.0	0.0	0.0	0.0	0.0	0.0
Tax	3.6	0.8	3.8	5.5	3.2	-9.1
Profits from associates	0.0	0.0	0.0	0.0	0.0	0.0
Minority interests	0.0	0.0	0.0	0.0	0.0	0.0
Net profit	-10.1	5.5	-2.4	-12.2	0.6	66.4
Restated net profit	-10.1	5.5	-2.4	-12.2	0.6	66.4
Change (%)	-115%	-	-143%	-417%	-	10182%
Cash Flow Statement (EURm)						
Operating cash flows	-10	5	-2	-12	1	66
Change in working capital	0	-5	10	-10	0	0
Capex, net	0.12	0.18	0.12	0.12	0.12	0.12
Free Cash flow	-10	1	8	-22	1	67
Financial investments, net	0.0	-0.6	0.0	0.0	0.0	0.0
Dividends	0	0	0	0	0	0
Capital increase	0	0	5	5	0	0
Other	0	-1	0	0	0	0
Net debt (+)/cash (-)	-57	-9	-15	1	0	-67
Balance Sheet (EURm)						
Tangible fixed assets	0.9	0.9	0.9	0.9	0.9	0.9
Intangibles assets	52.0	52.6	52.5	52.4	52.3	52.1
Cash & equivalents	65.2	12.4	25.0	8.0	8.7	75.2
current assets	10.6	10.6	10.6	10.6	10.6	10.6
Other assets	0.4	0.4	2.2	0.4	0.4	0.4
Total assets	129.1	76.9	91.2	72.2	72.8	139.2
L & ST Debt	9.3	4.5	10.8	9.3	9.3	9.3
Provisions	0.2	0.2	0.2	0.2	0.2	0.2
Others liabilities	10.5	10.5	20.5	10.5	10.5	10.5
Minority interests	0.0	0.0	0.0	0.0	0.0	0.0
Shareholders' funds	109.2	61.8	59.4	52.2	52.9	119.3
Total Liabilities	20.0	15.2	31.5	20.0	20.0	20.0
Ratios						
Gross margin	-	100.0%	100.0%	100.0%	100.0%	100.0%
EBITDA margin	-	20.3%	-37.9%	-362.4%	-13.5%	80.1%
Operating margin	-	19.8%	-37.9%	-362.4%	-13.5%	80.1%
Tax rate	-	-	-	-	-	-
Net margin	-	22.4%	-14.3%	-248.1%	3.2%	70.6%
Dividend payout	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%

Source: Company Data; Bryan, Garnier & Co ests.

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EXECUTIVE SUMMARY

OSE Immunotherapeutics is building a diverse clinical pipeline in oncologic and autoimmune indications based on its in-depth understanding of immune regulation. The unique expertise of the company stems from its historical collaborations with thought leaders in the field, including INSERM, University Hospital of Nantes and Léon Bérard Cancer Center. OSE's business model is based on the identification of promising immunotherapy targets and their early-stage development, with subsequent out-licensing to a partner. Importantly, OSE already has a proven track record of sizable deals for preclinical assets, listing Boehringer Ingelheim (BI), Servier and Janssen Biotech among its partners. Considering the cost of the late-stage clinical development and the resourcefulness of the big pharma, we believe that OSE's approach could significantly reduce development risks for the company as well as improve the odds for the drug candidates to reach the market.

We currently expect BI 765063, a novel checkpoint inhibitor (CPI) out-licensed to BI, to generate next significant milestone payment in 2021 and reach the market in 2025, generating royalty stream for OSE. BI 765063 targets signal regulatory protein-alpha (SIRPα) receptor that delivers "don't eat me" signal to macrophages though CD47 receptor. CD47/SIRPα signaling recently attracted a lot of interest in immuno-oncology field, albeit most of the competitors are targeting CD47 part of the equation. We believe that SIRPα-centric approach could provide certain advantages, including the elimination of hematologic side-effects and off-target inhibition of SIRPγ.

Within company's oncology franchise we also emphasize a novel bispecific antibody platform, BiCKI. BiCKI products are based on company's proprietary anti-PD-1 backbone and will include the second target of choice. The first BiCKI-derived asset, BiCKI IL-7, can (i) antagonize PD-1 and (ii) simultaneously stimulate interleukin-7 (IL-7) signaling. The later is involved in T cell proliferation and could help to overcome T cell exhaustion in solid tumors. We currently expect BiCKI IL-7 to be ready for clinics in 2021, highlighting it for a potential partner. In our view, BiCKI platform has a potential to fuel OSE's pipeline with promising oncology assets in the future, starting with BiCKI IL-7.

OSE is also developing therapies in the autoimmune space, with one of the assets, OSE-127, being co-developed with Servier. The phase II studies in UC and Sjogren syndrome are planned to start in 2022 and, depending on the outcome, Servier could fully exercise its licensing option, generating another milestone payment for OSE. Additionally, we believe that in 2H20-1H21 OSE could secure another partnership agreement for FR104, a phase-II-ready immuno-suppressive therapy, in kidney transplantation.

In 1Q20, OSE is expecting an interim readout from a phase III Atalante 1 study of its neo-epitope cancer vaccine, Tedopi. Atalante 1 is evaluating Tedopi in NSCLC patients who previously received anti-PD/L-1 therapy. At this time, we are staying on the sidelines regarding the expectations for this readout, as we believe that Tedopi could be more suitable for combination therapy as well as might be more efficient in a subset of CPI-refractory patients.

Taken together, we believe that OSE, with a deep pipeline and proven track record of early-stage partnerships, has all cards in hands to secure more licensing deals in the near future and to continue expansion of clinical pipeline.

OSE immunotherapeutics construit un pipeline clinique extrêmement diverse au sein des domaines de l'oncologie et des maladies auto-immunes grâce à sa compréhension intime des phénomènes de régulation de l'immunité. L'expertise unique de la société repose sur des collaborations historiques avec des experts reconnus du domaine tels que d'INSERM, l'Hôpital Univ. de Nantes ou le Centre Léon Bérard de Lyon. Le business model d'OSE est fondé sur l'identification de cibles d'immunothérapie prometteuses et sur leur développement précoce, avec licence ensuite à un partenaire. Il est important de noter que OSE a un track-record éprouvé d'accords signés pour des actifs en PC, incluant BI, Servier ou Janssen parmi ses partenaires. L'approche d'OSE nous paraît réduire significativement les risques de développement et améliorer les chances des candidats-médicaments d'atteindre le marché, compte tenu des coûts relatifs à un développement en phases II et III.

Nous anticipons aujourd'hui que BI765063, un nouvel inhibiteur de point de contrôle licencié à BI, génère un paiement d'étape significatif en 2021 et qu'il rejoigne le marché en 2025, générant un flow de royalties pour OSE. BI765063 cible les récepteurs de la protéine SIRP¤ (signal regulatory protein-alpha) laquelle délivre un message « ne me mange pas » aux macrophages via le récepteur CD47. Cette voie de signalisation CD47/ SIRP¤ a attiré récemment beaucoup d'intérêt dans le domaine, même si la plupart ciblaient surtout la partie CD47. Nous pensons justement qu'un ciblage plus particulier de SIRP¤ peut apporter des avantages, incluant l'élimination d'effets secondaires hématologiques et effets d'inhibition off-target de SIRP¤.

Au sein de la franchise oncologie d'OSE, nous portons l'attention également sur une nouvelle plateforme d'anticorps bispécifiques nommée BiCKI. Les candidats qui en sont issus sont construits à partir d'une ossature d'anti-PD1 propriétaire et inclura une seconde cible au choix. Les premiers actifs qui en dérivent, BiCKI IL-7 pourraient antagoniser simultanément PD-1 et l'IL7. Ce dernier est impliqué dans la prolifération à cellules-T et pourrait aider à contourner l'épuisement en cellules-T retrouvé dans beaucoup de tumeurs solides. Nous anticipons aujourd'hui que BiCKI IL-7 puisse entrer en clinique en 2021, attirant alors un possible partenaire. Cette plateforme a le potentiel pour enrichir le pipeline d'OSE d'actifs en oncologie prometteurs dans le futur.

OSE développe également des molécules dans le domaine des maladies autoimmunes, l'un des actifs ici étant co-développé avec Servier. Les phases II dans la colite ulcéreuse et le syndrome de Sjogren doivent démarrer en 2022 et, fonction des résultats, Servier pourrait exercer son option de licence, générant un nouveau paiement d'étape pour OSE. En outre, nous pensons qu'en 2H20-1H21, OSE pourrait sécuriser un autre accord de partenariat pour FR104, une thérapie immunosuppressive prête à aller en phase II dans la transplantation rénale.

Au Q1-2020, OSE s'attend à recevoir les données intérimaires de la phase III de son vaccin anticancereux à base de neo-epitopes Tedopi. L'étude ATALANTE-1 évalue Tedopi dans le CPNPC chez des patients précédemment traités par PD-(L)1. A ce stade, nous restons prudents quant à ces résultats car nous pensons Tedopi mieux placé pour des combinaisons ainsi que pour une population de patients réfractaires aux anti-PD-(L)1.

Au global, nous pensons qu'OSE, grâce à un pipeline très diversifié et à un track record éprouvé de partenariats, a toutes les cartes en mains pour en sécuriser de nouveaux et poursuivre l'expansion de son pipeline clinique.

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Company's Pipeline

PROGRAM	Indication	Pre-Clinical POC	Phase 1	Phase 2	Phase 3
IMMUNO-ONCOLOGY					
Tedopi® Neoepitopes	NSCLC				EU-US-Israel Ongoing
Tedopi®	Advanced pancreatic cancer			Combo with PD1 Opdivo® Ongoing	GERCOR, Bristol-Myers Squibb
BI 765063 (OSE-172) SIRPα	Various cancers		Ongoing	Boehringer Ingelheim	
OSE-703 IL-7R	Various cancers	2019	Memorial Sloan Kettering Cancer Center		
BiCKI® Bispecific anti-PD-1 & Innovative Targets	Various cancers	2019			
AUTO-IMMUNE DISEASES					
FR104 CD28	Auto-immune diseases & Transplantation			Phase 2 planning ongoing	
OSE-127 IL-7R	Ulcerative Colitis Sjögren syndrome		Positive Phase 1 Results Q4 2019	2020	SERVIER

First-in-class product

Upcoming catalysts

Program	Catalyst	Expected period
Tedopi	Interim readout from phase III Atalante 1 study	1Q20
FR104	Partnership agreement	2H20-1H21
BI 765063	Phase I/II results and milestone payment	2021
OSE-127	Phase II results and exercise of licensing option	2022

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Contents

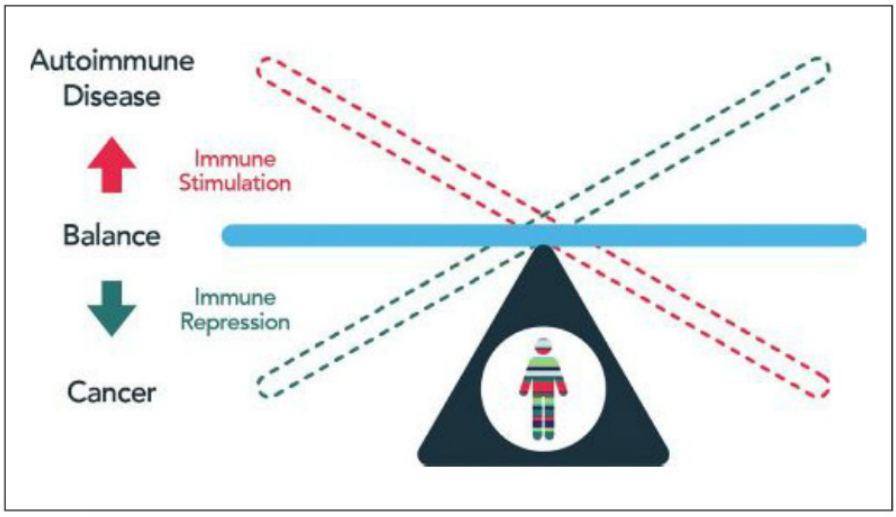
OSE - integrated Immunotherapy player

Leveraging immune system to tackle onco and autoimmune diseases

CENTRAL ROLE OF IMMUNE SYSTEM

OSE Immunotherapeutics is leveraging its expertise in the immune system regulation to develop novel therapies for oncologic and autoimmune indications. Owing to its ability to identify and eliminate 'foreign' or diseased cells, immune system plays a central role in development of both oncologic and autoimmune diseases. If the immune system is repressed this can lead to cancer growth as immune cells fail to recognize and attack tumor. On the other hand, over stimulation of immune system can result in faulty recognition of own healthy cells and subsequent destruction of healthy tissues, the underlying cause of all autoimmune diseases (Fig. 1).

Fig. 1: Role of immune system in cancer and autoimmune diseases



Source: https://www.ddw-online.com

Thus, in cancer immunotherapy, the aim is to push the immune system into action by using molecular signals that:

1) can directly strengthen or activate immune response against cancer cells;
2) can overcome the tumor's immuno-suppressive tactics allowing immune cells to recognize cancer.

In autoimmune diseases, the main goals are:

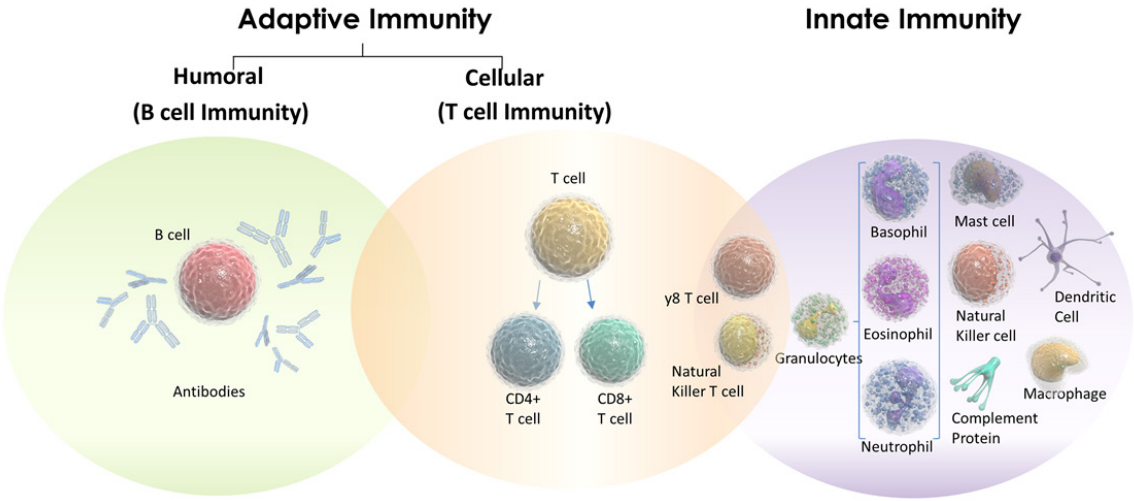
1) to find the ways to suppress an overactive immune system;
2) to avoid excessive immunosuppression so that patient's ability to resist infections and cancer is not compromised.

Therefore, an in-depth understanding of the controlling nods of immune regulation could spur the development of immunotherapies for both oncologic and autoimmune indications, and potentially through the same regulatory pathways.

MULTIPLE ANGLES TO FINE-TUNE IMMUNE RESPONSE

OSE established its expertise in targeting both innate and adaptive immune responses through in-depth understanding of the regulatory frame works of different immune cell types. The immune system is exceptionally complex and there are numerous types of immune cells that either circulate throughout the body or reside in a particular tissue. Each cell type plays a unique role in global defence system, communicates with other cell types, and performs certain functions (Fig. 2).

Fig. 2: Types of immune cells



Source: www.oxfordimmunotec.com

In general, the immune system could be divided into innate immunity and adaptive immunity:

- Innate immune system is at the first front of defence and comes into the play immediately upon pathogen invasion. It is non-specific, meaning it has general surveillance function, which is performed by magnitude of cells, including natural killer cells (NK), macrophages, and dendritic cells.
- Adaptive, or acquired, immunity targets specific threats to the body. It is more complex than innate immunity as the threat signals must be processed and recognized by the body. Moreover, adaptive immune system "remembers" the threat signals, which makes the future responses more agile and robust. Lymphocytes, such as T and B cells, constitute the core of adaptive immune response.

Importantly, the interactions between these cell types as well as between immune cells and other tissues affects the efficiency of the immune response. The intercellular communication is achieved though regulatory molecules, such as interleukins, that support function, survival, and

proliferation of immune cells. Additionally, many types of immune cells possess regulatory mechanisms in order to differentiate 'self' and 'threat' signal in order to spare own healthy cells.

OSE is tackling complex immune system regulation from multiple angles, including activation and suppression of both innate and adaptive immune responses through targeting macrophages, dendritic cells and T cells. Such unique expertise of the company stems from its historical collaborations with thought leaders in the field, including INSERM, University Hospital of Nantes and Léon Bérard Cancer Center. Historically, OSE Immunotherapeutics was created in 2016 through a merger of OSE Pharma and Effimune. Both parental companies had unique expertise and asset portfolio in the field of immunotherapies. Notably, Effimune, which spun out from the Nantes Institute of Transplantation, Urology and Nephrology, had close and longstanding collaborations with research institutes in Nantes, including INSERM. Leveraging these alliances, OSE Immunotherapeutics created a diverse pipeline of clinical drug candidates:

a) Immuno-oncology franchise
    - BI 765063, a checkpoint inhibitor that target innate immune response;
    - Tedopi, a cancer vaccine that targets 10 neoepitopes;
b) Autoimmune franchise
    - OSE-127, an anti-IL-7R antibody to down-regulate overactive immune response;
    - FR-104, an anti-CD28 antibody to inhibit T cell proliferation.

OSE has also revealed a new platform technology (BiCKI) that could spur the development of several next-generation bispecific checkpoint inhibitors based on anti-PD-1 backbone, a well-established clinical target. Thus, with the armamentarium of proprietary immuno-modulating drug candidates and a novel drug-development platform, supported by deep expertise and fundamental research, we believe that OSE is well positioned in immuno-oncology and autoimmune spaces.

Licensing deals to support the value of early-stage assets

OSE's business model is based on the identification of promising immunotherapy targets and their early-stage development, with subsequent out-licensing to a partner. Considering the cost of the late-stage clinical development and the resourcefulness of the big-pharma partners, we believe that OSE's approach could significantly reduce development risks for the company as well as could improve the odds of bringing the drug candidates to the market. Moreover, nondilutive finding in form of upfront and milestone payments could support the development of follow-on assets in the company's pipeline.

Remarkably, OSE succeeded to strike several sizable deals for early-stage assets with well-established pharmaceutical companies. In 2016, the company signed an agreement with Servier for OSE-127 at the preclinical stage a two-step licensing option of €30M and up to €232M in additional milestones, as well as double-digit royalties. Servier decided to exercise its licensing option in early 2019. In 2018, OSE sealed another remarkable pre-clinical deal with Boehringer Ingelheim (BI) for BI 765063 (formerly OSE-172). BI in-licensed OSE-172 with an upfront payment

of €15M and over €1.1B in total potential development, regulatory and sales milestone, and royalty payments. Additionally, FR104 was out-licenced to Janssen (a subsidiary of Johnson & Johnson) in 2016. Albeit Janssen terminated the licensing agreement in November, 2018 due to reprioritization of its pipeline, the initial terms of agreement included €10M upfront payment and up to €155M in additional milestone payments. Thus, we believe that OSE's track record justifies its business model and paves the way for other early-stage assets in the pipeline to follow the same path.

Notably, OSE Immunotherapeutics inherited experienced management team, including CEO Alexis Peyroles, who previously served as CFO of OSE Pharma, chairman Dominique Costantini, a former CEO and co-founder of OSE Pharma, and vice chairman and director of strategy Maryonne Hiance, who co-founded and served as CEO of Effimune, as well as CSO Dr. Nicolas Poirier, who was with Effimune previously. In our view, such commitment of the management team as well as its previous achievements underline the potential to successfully execute company's business strategy with the same rigor. **Thus, we believe that OSE is holding all the cards to secure additional licensing agreements and further develop its pipeline in oncology and autoimmune diseases.**

Part 2: Diverse oncology franchise as a value-driver

OSE is capitalising on development of the novel checkpoint inhibitors that could drive the value of the company in the future. Immune checkpoints serve as a block posts of the immune system. They represent a plethora of inhibitory pathways hardwired into the immune system that are crucial for maintaining self-tolerance and modulating the duration and amplitude of physiological immune responses. These pathways prevent the immune system from attacking own healthy cells, however tumors learned to exploit these regulatory roadblocks to avoid immune attacks. Cancer cells can 'camouflage' themselves with molecules that serve as 'self' recognition and inhibit activation of immune system. Importantly, since many of the immune checkpoints are initiated by ligand–receptor interactions, they can be blocked by antibodies or modulated by recombinant forms of ligands or receptors. Thus, checkpoint inhibitors (CPIs) attracted a lot of attention in the field of immuno-oncology. According to bcc Research, the global checkpoint inhibitors market could reach $29B by 2023.

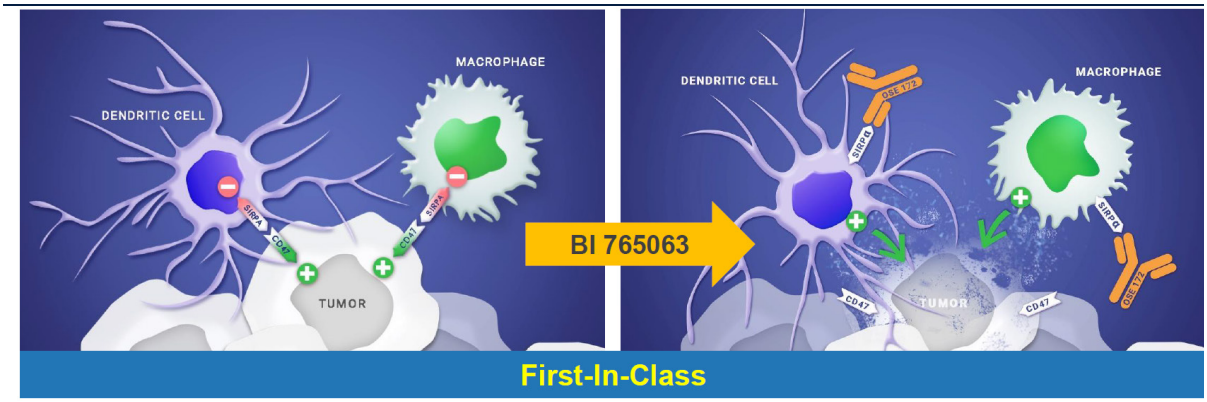
Interestingly, with blockbuster drugs, such as Keytruda form Merck & Co and Opdivo from Bristol-Myers Squibb, T cell - directed CPIs took a central stage. Both Keytruda and Opdivo target PD-1 checkpoint receptor on T cells. Currently, there are over 2300 ongoing clinical studies that involve anti-PD/L-1 therapies, pointing out to the tight competition among CPIs that aim to unleash adaptive immunity. At the same time, there are fewer assets among CPIs that target innate immune response, although innate immune cells can 'hunt' cancer cells as well. For example, a subset of pro-inflammatory M1 macrophages has a strong potential to attack and destroy cancer.

Additionally, not all tumors respond to anti-PD-1 treatments due to several reasons, including immuno-suppressive tumor microenvironment (TME) and T cell exhaustion. Thus, there is a lot of room for the next-generation CPIs that could target well-proven PD-1 pathway along with providing additional immunostimulatory functions.

BI 765063 could help to unleash innate immunity against cancer

One of the promising assets in the company's oncology pipeline, BI 765063 (formerly OSE-172), is a CPI designed to remove the immune breaks on macrophages. BI 765063 targets signal regulatory protein-alpha (SIRPα) receptor that delivers "don't eat me" signal to macrophages. "Hunting" immune cells, such as macrophages, are able to detect "eat me" signals found on foreign or damaged cells, while sparing healthy cells that display "don't eat me" signals. Cancer cells, on the other hand, have developed mechanisms to trick the immune system through camouflaging with specific proteins, such as CD47, that macrophages recognize as a "don't eat me" (Fig. 3). CD47 is over-expressed on numerous solid and hematologic tumours and clinically correlates with poor prognoses in cancer patients. This CD47-mediated immune evasion occurs through binding to signal regulatory protein-alpha (SIRPα) on macrophages. As a result, CD47-SIRPα interaction delivers an anti-phagocytic "don't eat me" signal and cancer cells escape macrophages' attack.

Fig. 3: Targeting CD47-SIRPα axis to fight tumour cells

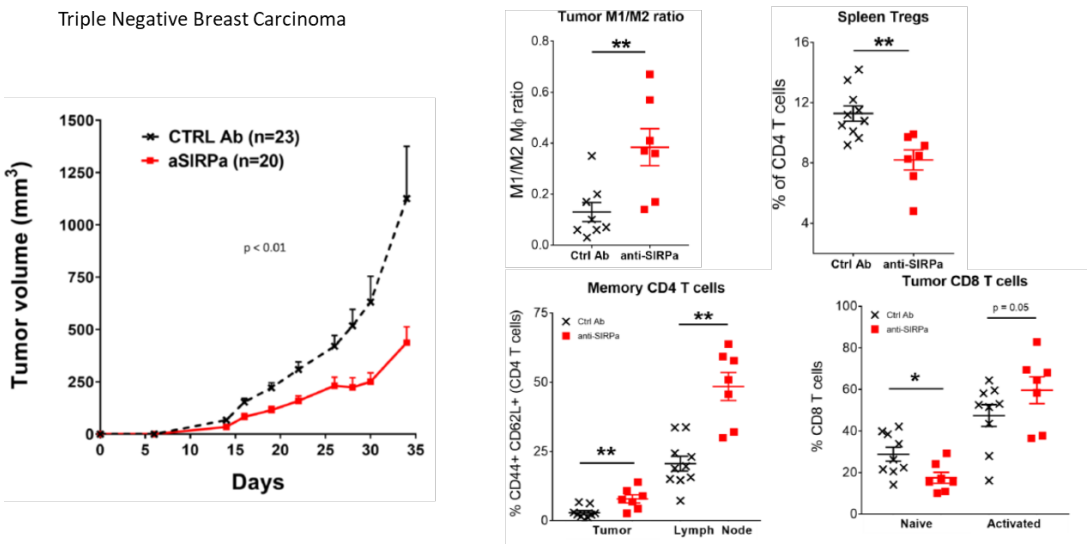


Source: company's presentation, 2019

Furthermore, CD47/SIRPα axis was shown to play an important role in the functioning of dendritic cells that infiltrate tumors, process and present tumor-derived antigens to naïve T cells. CD47/SIRPα signalling also regulates maintenance of the myeloid-derived suppressor cells (MDSCs), which is a population of immature precursors known to suppress immune response. Thus, blocking CD47/SIRPα could help macrophages to recognise tumor cells, as well as promote dendritic cell response and the differentiation of MDSCs into pro-inflammatory tumour-suppressive cells. We also note that the over-expression of CD47 by the range of different tumors suggests the potential of anti-CD47/SIRPα therapies in multiple oncologic indications, making this signalling pathway an attractive therapeutic target.

In preclinical tumor models of triple negative breast cancer (TNBC), colon carcinoma, hepatocellular carcinoma and mesothelioma, BI 765063 was able to significantly increase survival.

Fig. 4: anti-SIRPα exhibits anti-tumor activity

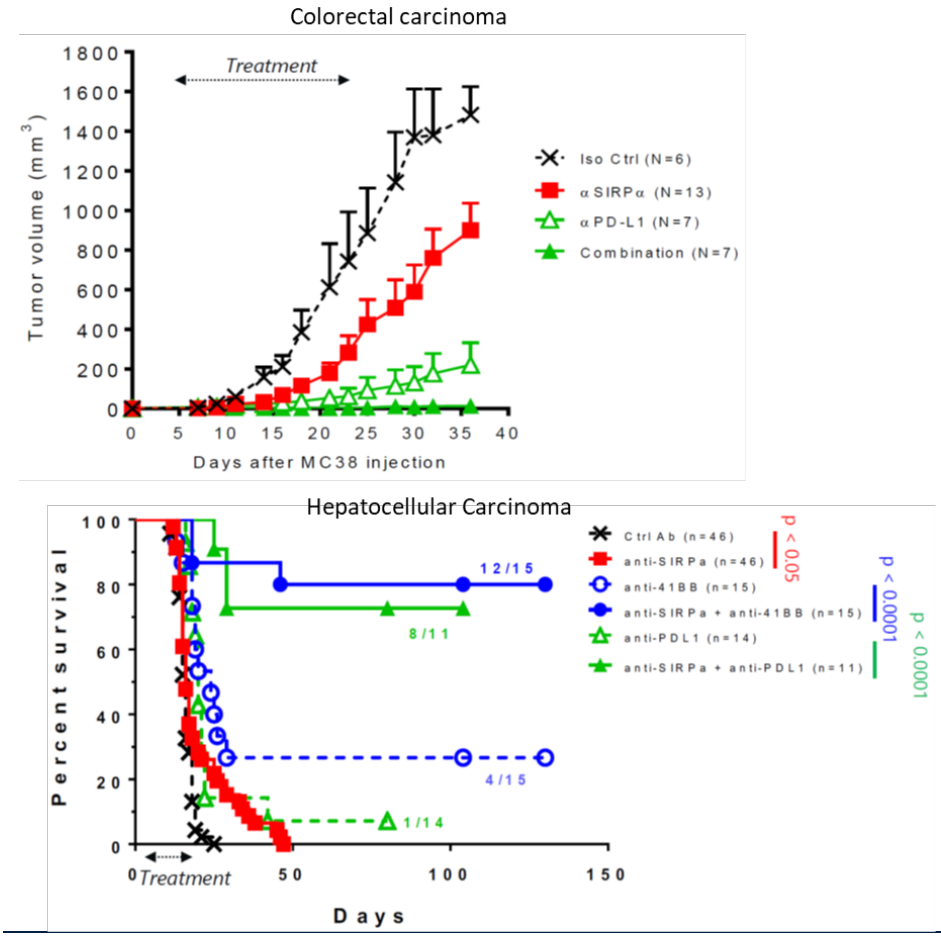


Source: Company's presentation, AACR 2018

Moreover, preclinical studies also showed BI 765063's potential to prevent lung and liver metastasis and to shift TME towards pro-inflammatory tumor-suppressive state by increasing the number of T effs and M1 macrophages and decreasing MDSCs and M2 macrophages (Fig. 4). On one hand, high levels of tumor-infiltrated T effector cells (T effs) and pro-inflammatory M1 macrophages correlate with good prognosis in many solid cancers. On the other hand, high levels of T regulatory cells (T regs) and immunosuppressive M2 macrophage infiltration correlate with a worse clinical outcome.

Additionally, BI 765063 showed synergistic effects when combined with checkpoint inhibitors or costimulatory agents in murine models of hepatocellular carcinoma and colon cancer (Fig. 5). The preclinical studies suggest that synergistic mode of action could potentially stem from BI 765063's ability to modify innate and adaptive tumor microenvironment, to increase tumor-associated antigen presentation by dendritic cells and to induce durable anti-tumor memory lymphocyte responses. In our view, those results are especially encouraging as BI 765063 could be a promising drug candidate for combination therapy with approved CPIs. Thus, we see a solid scientific rationale for the development of BI 765063 in oncologic indications.

Fig. 5: BI 765063 is a promising combination asset



Source: company's presentation, AACR 2018

BI 765063 STANDS OUT FROM THE COMPETITION

Naturally, many companies got interested in development of CD47/SIRPα-targeting therapies against solid and hematologic cancers (Fig. 6). Forty Seven and Trillium Therapeutics have the most advanced clinical assets that target CD47 receptor. Surface Oncology is also focusing on anti-CD47 antibodies, with therapies at the early clinical stage of development. Novimmune developed a different approach through anti-CD47/anti-CD19 bi-specific antibody, which was in-licensed by June TG in 2018. Whereas Alexo Therapeutics is going after disruption of the CD47-SIRPα pathway with mutated SIRPα monomers, which are currently in an early-stage clinical study as an adjuvant therapy.

Notably, such urge of interest was supported by the increased investment activity, with two initial public offerings (IPOs) in 2018. Forty Seven and Surface Oncology both raised over $100M through their IPOs. Albeit CD47 space faced a setback as well, reinforced by Celgene's discontinuation of its anti-CD47 program, CC-90002. Nevertheless, recently Forty Seven revived investors interest with encouraging data from its phase Ib study, presented at ASH 2019. The company showed that its anti-CD47, magrolimab, in combination with azacytidine achieved high response rates in untreated patients with hematologic malignancies (high-risk MDS and AML). We also note that magrolimab as monotherapy achieved clinical responses in some patients with heavily pre-treated solid tumors and the company is currently evaluating it as a part of combination therapy against ovarian and colorectal cancers.

Fig. 6: Clinical-stage programs that target CD47/SIRPα axis

Company	Program	MOA	Indication	Development Stage
Forty Seven	Magrolimab (Hu5F9-G4)	anti-CD47	Hematologic (AML/MDS, NHL) and solid (CRC, ovarian, bladder) cancers	Phase 2 (combo) and Phase 1
	FSI-189	anti-SIRPα	N/A	Preclinical
Trillium Therapeutics	TTI-621	SIRPα-Fc fusion, Ig1	B- and T-cell lymphomas, solid cancers	Phase 1b/2 (combo)
	TTI-622	SIRPα-Fc fusion, Ig4	Lymphoma, myeloma	Phase 1a
OSE/BI	BI 765063	anti-SIRPα	Solid cancers	Phase 1
ALX Oncology	ALX148	High-affinity SIRPα variant	Lymphoma and solid cancers	Phase 1
Surface Oncology	SRF231	anti-CD47	Hematologic and solid cancers	Phase 1/1b

Source: Brayan Garnier research

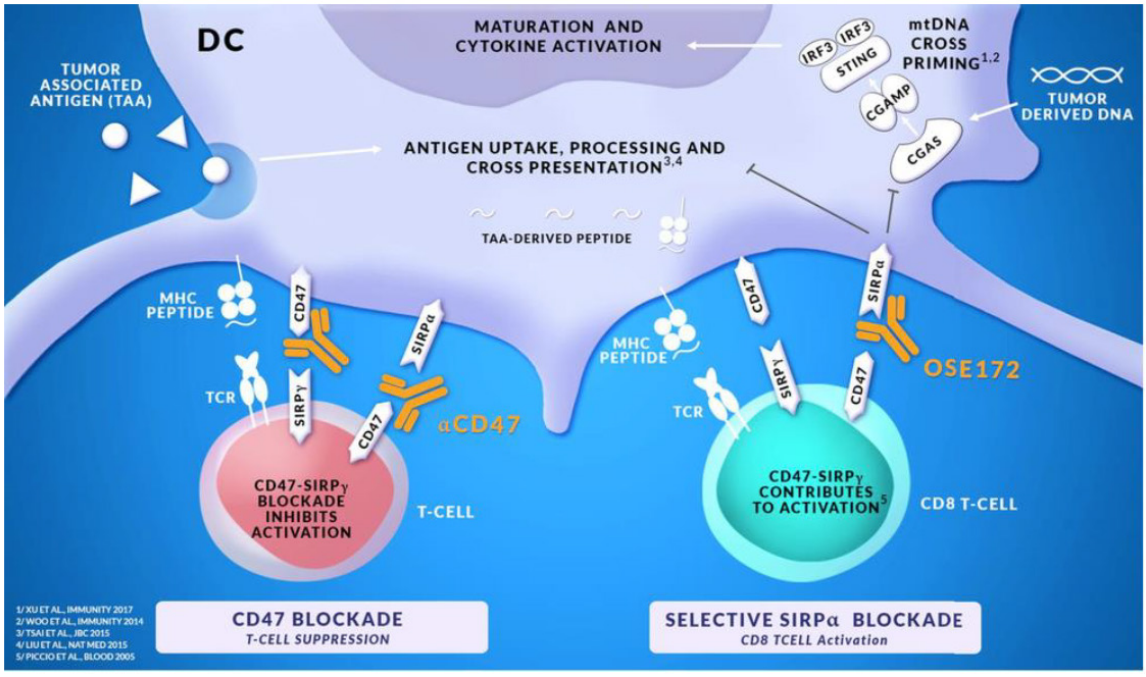
Remarkably, the majority of the companies is focused on targeting CD47, while SIRPα-centric approach is could have a strong differentiated position on the market (Fig. 7). We also note that the intellectual property around ati-CD47 antibodies has been a concern, as evidenced by a legal action between Forty Seven and Synthon. Moreover, Forty Seven has also introduced SIRPα-

targeting therapy, in our eyes, further supporting a SIRPα-centric approach. We believe that targeting CD47/SIRPα signalling form the SIRPα angle could provide certain advantages:

1) Due to ample biological role of CD47, CD47-targeting antibodies could cause sever lymphopenia, thrombocytopenia and anemia. Thus, the systemic administration of anti-CD47 antibodies could be limited, narrowing the therapeutic window. Meanwhile, inhibition of SIRPα could potentially side step these problems.

2) While CD47/SIRPα is a fairly well studied pathway, CD47 could also interact with SIRPγ receptor. SIRPγ is also activated through the interaction with CD47, albeit unlike SIRPα, this protein is expressed on T-cells and might negatively affect their migration to the tumor site. Thus, selective inhibition of SIRPα could bypass a potential off-target inhibition of SIRPγ.

Fig. 7: SIRPα- vs CD47-directed approach



Source: Company's presentation, 2019

Overall, based on the scientific rationale supported by the wide interest from the industry, we believe that CD47/SIRPα-targeting therapies represent an area of significant growth potential. Moreover, SIRPα-centric approach has unique competitive advantages in this space.

ONGOING PHASE I STUDY OF BI 765063 COULD SHOW EARLY SIGNS OF EFFICACY IN 2021

Importantly, the potential of this program was further validated by the licensing agreement with Boehringer Ingelheim, which resulted in €30M in upfront and short-term milestone payments. OSE is also eligible for up to €1.1B in development and commercial milestones, as well as potential royalties on net sales. In our view, such significant commitment from established large-pharma player further validates program's potential, as well as OSE's ability to secure licensing agreement at the early stage of development.

BI is also responsible for all clinical development costs and a phase I study of BI 765063 in multiple solid tumors is currently ongoing. The study will include two steps (dose-escalation and dose-expansion) to evaluate safety and preliminary efficacy of BI 765063 as a single agent and in combination with BI 754091 (anti-PD-1) in 116 patients with advanced metastatic tumours. We also note that while at the initial dose-escalation stage the study is recruiting both V1/V1 homozygous and V1/V2 heterozygous patients, the later cohort-expansion stage is planned to include only homozygous individuals. There are several genetic variants of SIRPα, with V1 and V2 alleles being the most common. Whereas genetic variation of SIRPα does not seem to affect its function, the efficacy of anti- SIRPα antibody may depend on its specificity towards particular allele (V1 or V2). In our view, the study design suggests BI 765063 specificity for V1 allele, thus its efficacy in V1/V2 heterozygous patients could be impaired. According to company, the frequency of V1 homozygous in general population is about 40%. Thus, we believe that BI 765063 has a potential to become a new therapeutic option for a significant number of patients with solid tumors.

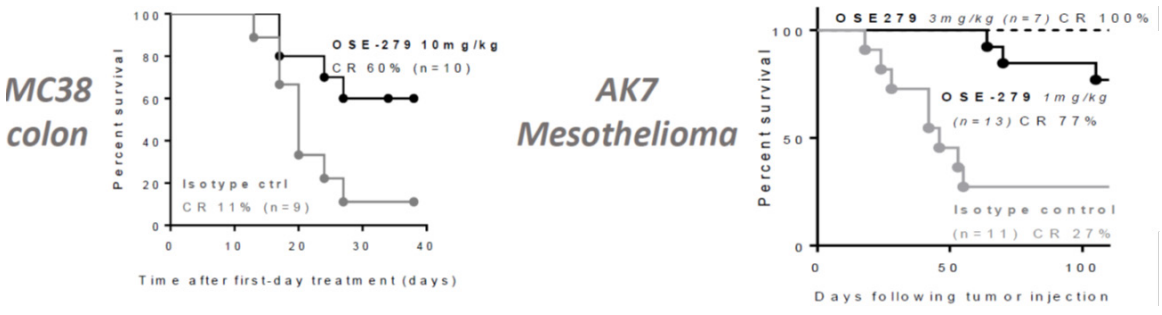
We currently expect the safety results from the study in 2020 and the efficacy data, including response rate to the therapy, in 2021. According to management, the second step of the phase I study or the initiation of a phase 2 study could also lead to another milestone payment from BI. **We currently expect the next significant milestone payment from BI in 2021, providing a positive catalyst for the stock. We project BI 765063 reach the market in 2026, generating royalty revenues of €120M by 2030.**

BiCKI platform could fuel clinical pipeline in the near term

In 2019, OSE disclosed its novel bispecific checkpoint inhibitor platform BiCKI, which could potentially augment the efficacy of CPIs. Bispecifics (BsAbs) are antibodies that can simultaneously engage two targets - hence, bi-specificity. Such simultaneous dual targeting could come in handy in several scenarios: 1) to bring immune and tumor cell close to each other, allowing immune cell to attack and destroy tumor; or 2) to simultaneously engage several receptors on the immune cells in order to augment the immuno-stimulatory properties. OSE's BiCKI platform is based on the key backbone component, a proprietary anti-PD-1 antibody (OSE-279), and will simultaneously engage another target of choice.

OSE-279 was designed to fully antagonize PD-1 binding to PD-L1 or PD-L2. Programmed death 1 (PD-1) receptor is a proven target in cancer treatment, with blockbuster drugs such as Keytruda from Merck & Co and Opdivo from Bristol-Myers Squibb. PD-1 is expressed on activated T cell lymphocytes and upon binding to its partners, PD-L1 and PD-L2, it prevents T cell from attacking own healthy cells. Tumors learned to hijack this mechanism to avoid recognition by immune system. Through overexpressing PD-L1, cancer cells can inhibit T cell activation and prevent an immune attack against them. Checkpoint inhibitors, such as anti-PD-1 (Keytruda and Opdivo) or anti-PD-L1 (Tecentriq and Imfinzi), achieved impressive clinical results changing the treatment paradigm in many oncologic indications. Preclinical studies of OSE's proprietary anti-PD-1 antibody also showed that OSE-279 was able to fully inhibit PD-1 signaling and to suppress tumor growth, potentially providing an effective backbone for BiCKI platform (Fig. 8).

Fig. 8: OSE-279 as a potent PD-L1/2 inhibitor



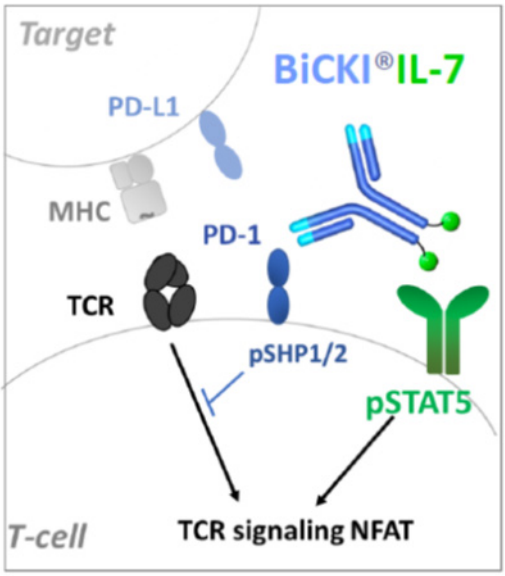
Source: Company's presentation, SITC 2019

While the anti-PD-1 therapies had significant success in the clinic, there is still a large fraction of cancer patients that do not respond to these therapies due to various reasons, including innate and acquired resistance. Thus, combination of target therapies (anti-PD/L-1 and another target) became a particular popular approach with over 2000 ongoing clinical trials. Meanwhile, BsAbs have certain advantages compared to combination of respective single-target monoclonal antibodies. One of the most attractive BsAbs features is a unique ability to simultaneously engage both targets at the same time in the same place. Such preciseness of the immunotherapies becomes particularly important when they are designed to target multiple signalling pathways, each of which has an ample role in regulation of immune system. BiCKI

platform aims to expand the reach of anti-PD-1 therapy through simultaneous engagement of the second target, which could potentially extend the spectrum of responding patients.

Currently, OSE has disclosed the first BiCKI-based design: 1) antagonize PD-1 and 2) simultaneously stimulate interleukin-7 (IL-7) signaling (Fig. 9). Interleukins, including IL-7, are key to T-cell function, survival, and proliferation and have long been of interest in cancer immunotherapy. IL-7 binds to the IL-7 receptor (IL-7Rα) on the surface of T cells, triggering their proliferation and long-term survival. Importantly, IL-7 induces naïve and memory T cells proliferation without expansion of immuno-suppressive T regs, which differentiates it from more known IL-2. While IL-2 spurred the most interest in the field, it also induces the proliferation of cancer-promoting T regs. IL-7, on another hand, was shown to selectively increase the number of T effectors, as its receptor IL-7Rα is poorly expressed on T regs. Thus, we see a lot of scientific rationale for combination of IL-7 agonist and anti-PD-1 therapy. Notably, while there are few IL-7-based therapies in the clinics (such as Hyleukin-7 from NeoImmuneTech), the challenge has been to deliver the signaling directly to T cells. In our view, the bispecific approach of BiCKI platform could help to overcome this issue.

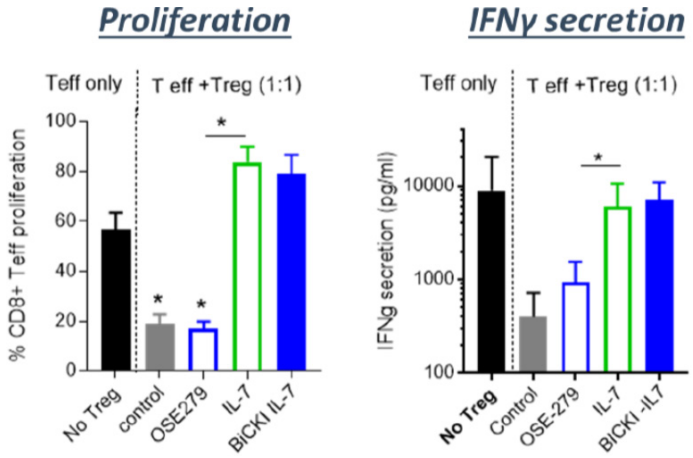
Fig. 9: First BiCKI-derived drug candidate



Source: Company's presentation, SITC 2019

In preclinical studies administration of BiCKI IL-7 resulted in increased expansion of T effs, but not Tregs. Decreased Treg cell frequency is an important differentiation factor for IL-7-based therapies (Fig. 10). Additionally, BiCKI IL-7 increased mucosal migration of T cells, suggesting a potential to promote tumor permeability by T cells. We also note that BiCKI IL-7 was more effective compared to OSE-279 alone (Fig. 10).

Fig. 10: Anti-tumor activity of BiCKI IL-7



Source: Company's presentation, SITC 2019

Overall, based on discussed scientific rationale and OSE's preclinical data, we believe that BiCKI IL-7 could become a first-in-class new generation CPI due to potential synergy between both arms (anti-PD-1 and IL-7). In our view, potential ability to mediate a potent memory response and T effs persistence could position BiCKI IL-7 well among other BsAbs and CPIs.

Moreover, OSE has put in place a collaboration with Léon Bérard Cancer Center (CLB) to identify novel targets that are associated with the resistance to anti-PD-1/L1 therapies based on the different patient cohorts and tumor biopsies. According to agreement, CLB would provide an Artificial Intelligence tool to identify clinically relevant therapeutic targets, which then will be integrated into BiCKI platform. Thus, considering unmet medical need in patients that do not respond to or progressed after anti-PD-1 treatment, we believe that BiCKI platform has a great potential to build upon the progress in scientific understanding of tumor resistance to anti-PD/L-1.

BICKI - IN THE RIGHT PLACE AT THE RIGHT TIME

We believe that BiCKI platform could significantly expand OSE's immuno-oncology pipeline in the near future, as well as provide attractive out-licensing opportunities as BsAbs are gaining momentum. In this regard, several recent deals with significant upfront payments are worth mentioning. In early 2019, Roche bagged Xencor's XmAb24306, an IL-15/IL-15Rα bispecific, for $120M in upfront payment, as well as up to $160M in additional milestone payments and substantial royalty rate on future sales. Moreover, Merck KGaA and GlaxoSmithKline signed a collaboration for the development of M7824, a PD-L1/TGF-ß bispecific, for €300M up front and up to €3.7B in total deal value. Overall, we believe that after the decades of basic research and optimization of the production process, persistence and safety, BsAbs platform technologies have reached the stage of certain maturity, encouraging large pharma players to respond by inking sizable deals.

Driven by the clinical success of CPI, there are currently few BsAbs in the clinic that are also targeting PD-1/PD-L1/2 axis (Fig. 12). Generally speaking, this class of antibodies could be

divided into two: 1) combination of immuno-regulatory arm with tumor-targeting arm (such as anti-PD-L1-based BsAbs); 2) combination of two immuno-regulatory arms aiming at lymphocytes (such as anti-PD-1 based BsAbs).

Fig. 11: Bispecific antibodies based on PD-1/PD-L1 backbone

Company	Product	MOA	Indication	Development Stage
Macrogenics	MGD019	PD-1 X CTLA-4	Solid cancers	Phase 1
	MGD013	PD-1 X LAG3	Solid (liver, gastric) and haematological cancers	Phase 1/2
Xencor	XmAb20717	PD-1 X CTLA-4	Solid cancers	Phase 1
	XmAb23104	PD-1 X ICOS	Solid cancers	Phase 1
AstraZeneca	MEDI5752	PD-1 X CTLA-4	Solid (lung) cancers	Phase 1
Akesobio	AK104	PD-1 X CTLA-4	Solid (gastric, melanoma) cancer	Phase 1/2
Roche	RG7769	PD-1 X TIM3	Solid (lung, melanoma) cancers	Phase 1
Alphamab	KN046	PD-L1 X CTLA-4	Solid (breast, lung, gastric) cancers and lymphoma	Phase 2
F-Star	FS118	PD-L1 X LAG3	Solid cancer	Phase 1
Innovent / Eli Lilly	IBI318	PD-1 X Undisclosed	Solid cancers	Phase 1
Inhibrx	INBRX-105	PD-L1 X 4-1BB	Lymphoma and solid cancers	Phase 1
Merus / Incyte	MCLA-145	PD-L1 X 4-1BB	Solid cancers	Phase 1

Source: Labrijn et al., Nature Reviews, 2019, and Bryan Garnier Research

The second immuno-regulatory arm could target another immune checkpoint receptor, including CTLA-4, LAG3, TIM3, with a rationale that it would remove additional breaks on the immune cells. Albeit such approach could be compromised by safety profile (as seen in combination of anti-PD-1 and anti-CTLA-4 therapies), requiring a further optimization of such BsAbs. On the other hand, the second arm of anti-PD-1 based BsAbs could positively regulate T cell activation through agonistic receptor (such as IL-7R). In this case, the rationale is to increase the proliferation of tumor-reactive T cells and to overcome their exhaustion at tumor site. For instance, PD-1/ICOS BsAb from Xencor, XmAb23104, aims to promote tumor-selective T-cell activation in order to treat advanced solid malignancies. As well as MCLA-145 from Merus, which signed a $200M development agreement with its bispecifics with Incyte. We also note that while BsAbs development for solid tumors could be more challenging due to low antigen expression, immuno-suppressive TME and exhaustion of T cells, there is more room for newcomers compared to hematologic malignancies. Additionally, the tight competition among novel BsAbs in hematologic cancers revolves around the same set of targets, leaving a limited space for differentiation.

Overall, we believe that BiCKI platform could fuel OSE's pipeline with novel bispecific CPIs against solid tumors, providing an attractive licensing opportunity for large-pharma players and generating value for the company. We currently expect the first asset generated through BiCKI platform, BiCKI IL-7, to be ready for clinics in 2021, making it even more attractive for a potential development partner. **Considering the terms of the recent deals in BsAbs space and OSE's track record of early-stage partnership agreements, we believe that the company has all cards in hands to secure another landmark deal, which can become a significant catalyst or the stock. We currently expect the first partnership agreement in association with BiCKI IL-7 in 2021.**

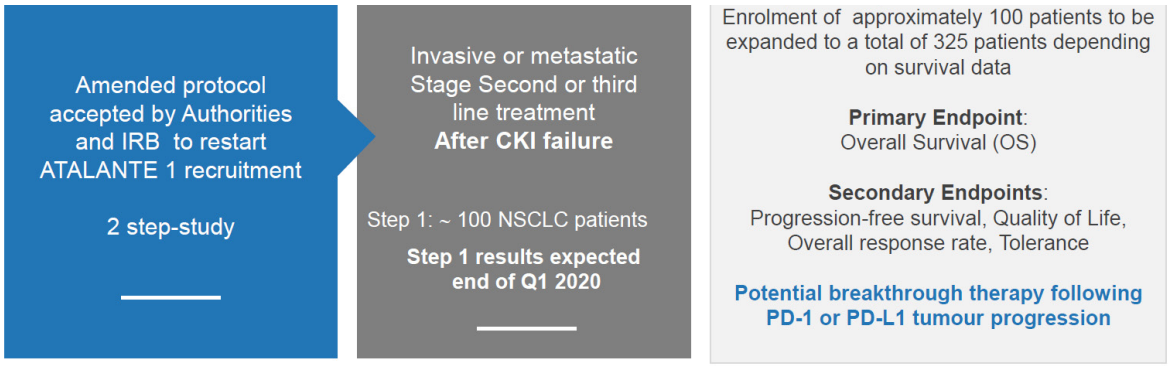
Interim results from Atalante 1 could help to define Tedopi's future

OSE's immuno-oncology franchise also includes a late-stage program, Tedopi, which is currently being evaluated in a phase III Atalante 1 study. Tedopi is a cancer vaccine comprised of 10 neoepitopes, which are the immunogenic parts of cancer-specific antigens. Specifically, Tedopi combines nine epitopes from known tumor-associated antigens MAGE2, MAGE3, carcinoembryonic antigen (CEA), HER2/neu, and P53 with a PADRE helper epitope designed to improve T cell response. Through delivering a range of neoepitopes that are commonly expressed by tumor cells, Tedopi could "alarm" the cytotoxic T lymphocytes and direct them to fight tumor cells that express the corresponding antigens.

Atalante 1 is evaluating Tedopi as a treatment for patients with non-small cell lung cancer (NSCLC), who are HLA-A2+ and progressed after CPIs. Although the survival rate for lung cancer in the US and EU has doubled in the last fifteen years it remains the most common cause of cancer-related death. An approval of Keytruda (an anti-PD-1 from Merck & CO) in combination with chemotherapy in the first-line NSCLC setting, as well as other CPIs, changed the standard of care in this indication, albeit nearly 50% of patients develop innate or acquired resistance and do not see significant clinical benefits. Atalante 1 study was initiated prior to these recent CPIs approvals and, consequently, the protocol of the study was modified to only include NSCLC patients that progressed after previous anti-PD/L-1 therapy.

Currently, Atalante 1 has a two-step design with a stopping point at the first step (n=100), which could either give a green light to proceed to the second step (n=325) or trigger the discontinuation of the study (Fig. 13). The first step analysis will be based on 12-month survival rate (number of patients that survived past 12 months), which is required to meet a pre-specified threshold. Atalante 1 also includes the control arm that is treated with chemotherapy (CT), as CT is currently the only available option for NSCLC patients post CPI. As of now, OSE reported that the Independent Data Monitoring Committee (IDMC) recommended the continuation of patients' recruitment due to absence of safety concerns. Additionally, OSE recently announced collaboration with HalioDX to perform biomarker analysis on the biopsy samples from Atalante 1 study, which, we believe, could help to identify the responding group more precisely.

Fig. 12: Atalante 1 study design



Source: Company's presentation, 2019

CLINICAL DATA SUGGEST TEDOPI'S ACTIVITY IN SOME PATIENTS

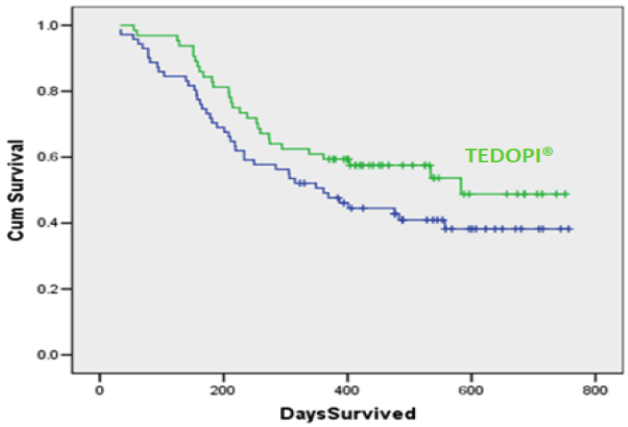
In the previous single-arm phase II study completed in 2008 by Epimmune (prior to Tedopi's acquisition by OSE), 63 patients with advanced, unresectable, HLA-A2+ NSCLC were treated with Tedopi for up to 2 years. While few patients achieved complete (2%) or partial (2%) response, majority of the patients (86%) reached stable disease for 3 months or longer. 12-month survival for all treated patients was about 59% and the median survival (mOS) was 17.3 months (Fig. 13). While these results could indicate survival benefits of Tedopi treatment, we note that they were achieved in CPI-naïve patients and it is difficult to directly translate these results into post-CPI setting.

Additionally, early signs of Tedopi's clinical activity from Atalante 1 study were presented at AACR conference in March, 2019. Presented data also showed low response rate: out of 18 evaluated patients in the investigational arm, 3 had clinical benefits (1 partial response and 2 stable disease). Although in 3 responded patients, Tedopi as a third-line treatment achieved progression free survival (PFS) of 4 - 18 months and survival after treatment initiation of more than 20 months. While we are encouraged by Tedopi's clinical activity in the responded patients, we note the low rate of responses and that patients also received CT after the treatment. There is also a lack of historical data to provide an estimate for a control arm performance (CT only).

We expect the results from the first step of Atalante 1 study in 1Q20, albeit at this time, we are staying on the sidelines regarding the expectations from this readout. Currently, we believe that Tedopi could be more suitable for combination therapy, as well as might be more efficient in a subset of CPI-refractory patients that step one of the study could help to identify.

We also note that in 2019, OSE announced a licensing agreement with Korean Chong Dang Pharmaceutical Corporation (CKD) for Tedopi's commercialization in South Korea. Financial terms of the deal include upfront and short-term payment of €1.2M and up to €4.3M in total milestone payments, as well as double-digit royalties on sales. We are encouraged by Tedopi's endorsement from Korean partner, albeit we also note relatively small size of the Korean oncology market and lower prevalence of HLA-A2 in Asian population.

Fig. 13: Tedopi showed promising results in phase II study



MEDIAN OVERALL SURVIVAL (p=0.086)
- **TEDOPI®: 17.3 MONTHS** vs. CONTROL* (HLA-A2-) : **12 months**

ONE YEAR SURVIVAL (p=0.063)
- **TEDOPI®: 59%** vs. CONTROL* (HLA-A2-) : **49%**

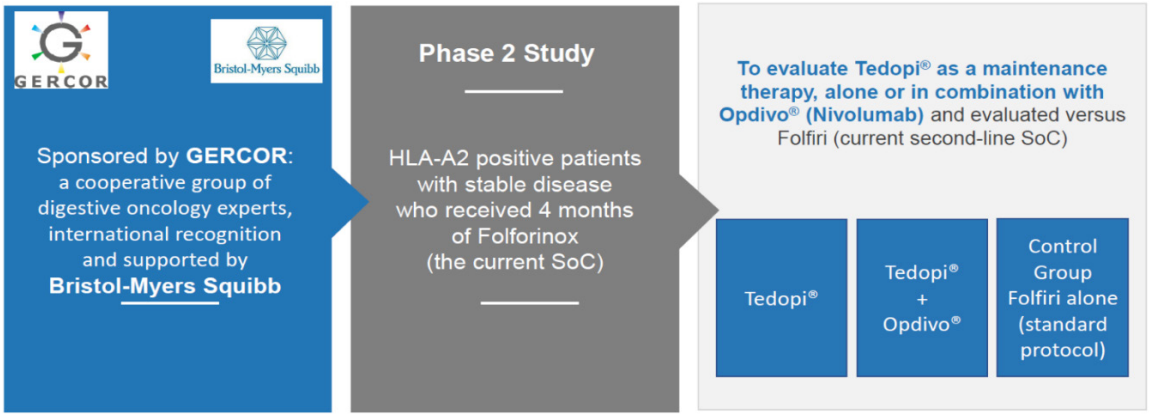
Source: Company's presentation, 2019

Overall, we project Tedopi to reach the market in the US and the EU in 2024, generating royalty revenues of €149M by 2029.

PANCREATIC CANCER AS A POTENTIAL EXPANSION INDICATION FOR TEDOPI

Additionally, Tedopi is being evaluated in the phase II TEDOPaM study in combination with Opdivo (an anti-PD-1 from Bristol-Myers Squibb) as a maintenance therapy in patients with advanced pancreatic cancer (Fig. 15). Pancreatic cancer is a notoriously cold tumor that lacks tumor infiltrating lymphocytes. Considering Tedopi's mechanism of action, it could prime the immune response against tumor, which could be augmented further by CPIs. Thus, there is a potential for synergistic effects between two therapies that TEDOPaM study is aiming to explore.

Fig. 14: Phase II TEDOPaM study in pancreatic cancer



Source: Company's presentation, 2019

TEDOPaM is conducted by Oncology Physician Network GERCOR and has begun patient accrual in 1H19. Since TEDOPaM is an investigator-sponsored study, we currently lack visibility on the recruitment process and potential interim readouts. Currently, *ClinicalTrials.gov* is listing only one medical center as 'recruiting' (out of 28 expected clinical sites) and the estimated primary completion date in December, 2022. Thus, we expect GERCOR to report the top-line results from this study in 2023. **If positive, we believe that OSE could potentially secure a licensing agreement for Tedopi in this indication, which would provide an additional upside to our current estimates.**

Part 3 : Autoimmune franchise de-risks clinical pipeline

Considering OSE's expertise in immune regulation and a critical role of immune regulation in autoimmune diseases, it seems rather natural that OSE's pipeline also includes assets within this therapeutic area. FR104 and OSE-127 are both in early stages of clinical development and both target novel immune mechanisms.

OSE-127 – a promising asset in UC and Sjögren's syndrome

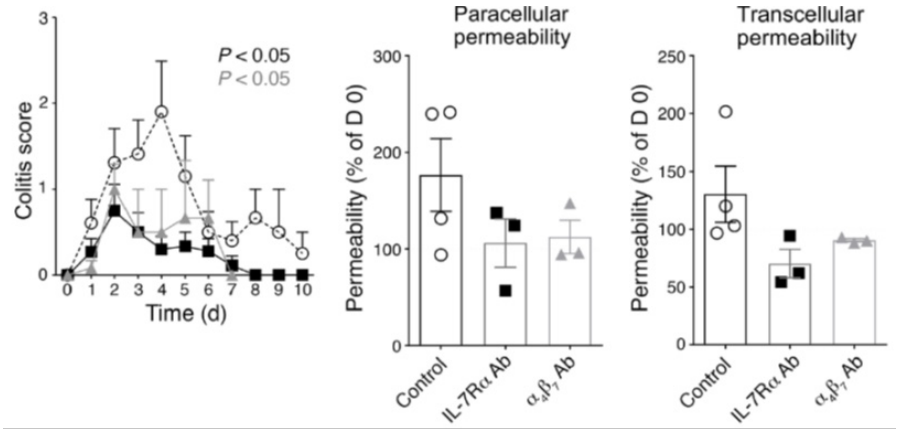
OSE-127 is a humanized monoclonal antibody that blocks the interaction between IL-7 and its receptor, IL-7Rα, found on the cytotoxic T-cells. As discussed earlier, IL-7 drives proliferation of T cells, but excessive signaling could lead to over proliferation, chronic inflammation and, as a result, autoimmune diseases. Therefore, blockade of the interaction between IL-7 and IL-7Rα could reduce inflammatory autoimmune attacks, making it an attractive target to treat autoimmune conditions such as ulcerative colitis (UC) and Sjögren's syndrome (SS). While the cause of both diseases is unknown, both UC and SS are associated with increased levels of IL-7.

SS involves the autoimmune destruction of patients' saliva and tear glands, leading to the disease's characteristic symptoms of dry mouth and dry eyes. Patients can also suffer systemic symptoms including fatigue and chronic pain. There is no disease-modifying therapy, and patients are managed mostly by using eye drops and painkillers. It is estimated that only in the US, SS affects about 4 million patients, however disease diagnosis could be difficult as the disease is associated with a broad range of symptoms. Moreover, in nearly 20% of patients SS coincides with other autoimmune diseases, further complicating the diagnosis of primary SS (pSS) and its treatment. Notably, IL-7 expression is increased in gland tissue of pSS patients and IL-7 levels correlate with increased markers of inflammation, suggesting that targeting IL-7 could be an effective therapeutic approach for these patients.

Ulcerative colitis (UC) is another debilitating autoimmune disease associated with increased levels of IL-7. UC is one of the two major types of inflammatory bowel disease, which is characterized by long-lasting inflammation and ulcers in the digestive tract (colon and rectum). UC symptoms including diarrhoea, bleeding, and severe abdominal pain, and nearly 1 million people in the US is suffering from this condition. Currently, there is no curative treatment for UC, and while flares could be managed with immunosuppressants, clinical symptoms ultimately come back. Colon tissue of UC patients who do not respond to immunosuppressants (including corticosteroids, anti-TNFα, or anti-α4β7 therapies) was shown to overexpress IL-7R, highlighting the importance of this signalling pathway in UC.

Importantly, preclinical studies suggested that both indications UC and pSS could be treated by blocking IL-7/IL-7R signalling. IL-7 blockade reduced human T cell homing to the gut and colonic inflammation in humanized mouse models, and altered effector T cells in colon explants from UC patients grown *ex vivo* (Fig. 16). In the preclinical model of pSS, anti-IL-7R antibody was able to ameliorate SS symptoms, including hyposalivation and leukocyte infiltration in the glands. Thus, we believe that disruption of IL-7/IL-7R signalling is a promising therapeutic approach in these autoimmune indications.

Fig. 15: Anti-IL-7R antibody reduces colitis in humanized mice



Source: Belarif et al., 2018

We also note that OSE signed a licensing deal with Servier for the development of OSE-127 in UC and pSS. According to the agreement, OSE is responsible for OSE-127 development in UC up to the completion of a phase II study, after which Servier is expected to take over the drug's development, and Servier is responsible for the clinical studies in SS. Currently, OSE has completed phase I study of OSE-127, which showed favorable safety profile, and the start of both phase 2 studies is planned for 2020.

While OSE-127 has shown promising results in the preclinical studies, the asset remains relatively unproven. We believe that the collaboration with Servier significantly lowers the development risk for OSE, especially taking into account tight competition and high clinical failure rate in both UC and SS. Thus, we believe that Servier's commitment to OSE-127 program, despite an early stage of development, further validates its potential. Importantly, this agreement also confirms OSE's ability to secure the deals for early-stage assets in autoimmune space. **Upon the completion of the phase II studies, which we expect in 2022, Servier could decide to fully exercise its licensing option, potentially providing another catalyst for the stock. Currently, we expect OSE-127 to reach the market in 2025, generating royalty revenues of €139M by 2030.**

FR104 is an attractive in-licensing asset

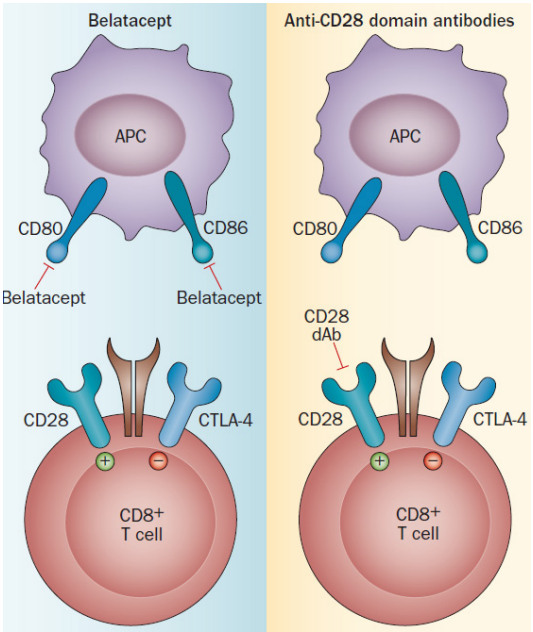
We believe that another OSE's asset in the autoimmune franchise, FR104, is an attractive out-licensing opportunity as well. FR104 is a humanized PEGylated antibody that blocks CD28, a stimulatory receptor of T cell lymphocytes. CD28 is an important co-stimulatory receptor expressed on T cells that delivers signals essential for full Teffs activation and for the development and homeostasis of suppressive Tregs. CD28 binds to its ligands CD80 and CD86 on antigen-presenting cells and positively regulates pro-inflammatory signaling. Note that CD80 and CD86 can also modulate the activity of co-inhibitor CTLA-4, which delivers anti-inflammatory

signaling. FR104 was designed to selectively inhibit CD28 signaling in Teffs, but not CTLA-4 pathway. In preclinical studies, FR104 was able to suppress the activity of aggressive Teffs and promote function of Tregs in order to enhance tolerance of the immune system.

FR104 was previously out-licensed to Janssen Biotech (a subsidiary of Johnson & Johnson) for development in autoimmune diseases and transplantation. Albeit Janssen terminated the licensing agreement in November, 2018 due to reprioritization of its pipeline. Consequently, OSE regained all the data, regulatory filings and intellectual property developed around FR104. After regaining the rights for FR104, OSE is evaluating the options for continuing the development of this program, including worldwide partnering opportunities.

Considering a paramount role of immune reactivity in transplant rejection, inhibition of CD28 signaling have been expensively studied in this indication. Rejection occurs when the body's defense system (immune cells) recognizes the transplant as a foreign object and a standard medical practice require patients, who underwent organ transplantation, to take immunosuppressive therapy. Moreover, belatacept (Nulojix from Bristol-Myers Squibb), a CD80/CD86-blocking antibody was approved for kidney transplant rejection. However, patients treated with belatacept experienced a higher incidence and severity of acute rejection episodes 1 year after transplantation. Notably, belatacept blocks not only CD28 signaling, but CTLA-4 as well (Fig. 17). It was suggested that the efficacy and safety profile of belatacept could be augmented by selective blockade of CD28, which might ultimately improve the control of Teffs differentiation and Treg-mediated suppression. In murine model of graft-versus-host disease (GVHD), FR104 was better at GVHD prevention compared to belatacept. Moreover, FR104 prevented alloimmunization and allowed minimization of CNI (standard of care for transplant recipients) in nonhuman primate renal allograft.

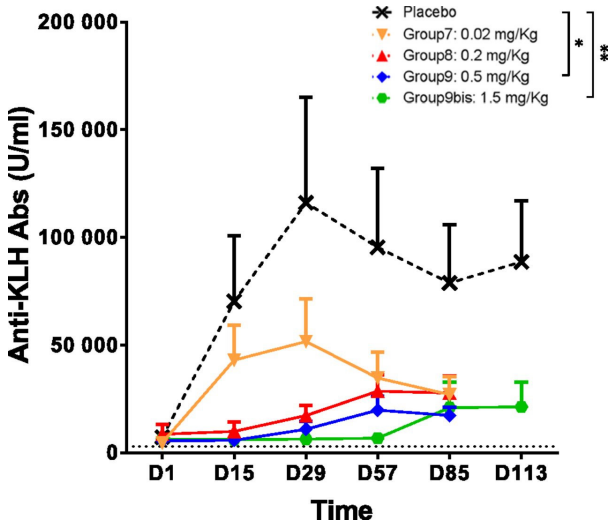
Fig. 16: Selective CD28 blockade does not affect co-inhibitory CTLA-4 signaling



Source : Ford M. et al, Nature Review, 2014

Notably, early attempts to design anti-CD28 therapy resulted in unwanted agonist activity, which lead to cytokine storm syndrome. FR104, on another hand, is a blocking nonactivating agent that was shown to safely and specifically block CD28, while sparing CTLA-4 co-inhibitory signals. Moreover, OSE has conducted a phase I study of FR104 in 46 healthy volunteers, which showed that the drug was well tolerated and had a promising biological activity, inhibiting both reactivity and severity of the immune reaction (Fig. 18).

Fig. 17: FR104 suppresses immune reaction in healthy volunteers



Source : Poirier et al, J Immunol, 2016

During 2018, nearly 22,000 kidney transplantations were performed in the US only and more than 240,000 were living with kidney transplant, according to registry of transplant recipients. Most patients that undergo organ transplantation would receive a combination of two or three immunosuppressive drugs to prevent organ rejection, which have to be taken daily over the lifetime. Donor-specific antibodies that can lead to organ failure are estimated to develop in 11% of patients during the first year after kidney transplantation and in 20% of patients by 5 years after transplantation. Thus, although kidney transplant recipients do relatively well during the first 5 years, there is a high unmet need to find novel therapies to prevent organ rejection and ameliorate the safety profile of current immunosuppressive regimens. Considering favorable safety profile and preclinical evidence, we believe that FR104 is a promising drug candidate to prevent organ rejection after kidney transplantation.

While global market of immunosuppressive drugs for kidney transplantation has reached nearly $2.5B is 2019 and expected to grow with CAGR of 3.3%, there is a tight competition in the space with 10 drugs currently approved and 12 in the phase 3 clinical studies. Notably, Nulojix did not meet expectation to become a blockbuster and currently peak sales of the drug is projected to reach $216M in 2020, with biosimilars driving sales down post 2020. The commercialization hurdles were associated with safety profile, inherent caution among transplant specialists and high pricing. Interestingly, BMS is also developing specific CD28 antagonist, lulizumab pegol

(BMS-931699). The drug is currently is the phase I/II study as a combination therapy for kidney transplant recipients. Therefore, we believe that FR104 is still an attractive in-licensing opportunity for pharmaceutical companies that seek an expansion of their autoimmune franchises.

We note that back in 2016, Janssen has exercised its option to license FR104, with €10M upfront payment and up to €155M in additional milestone payments as well as royalties on sales. Additionally, Janssen was responsible for all clinical development and commercialization costs for FR104. Moreover, in 2019, OSE was able to strengthen the IP protection of FR104, with new patents issued by the Canadian Intellectual Property Office and the USPTO, covering the use of FR104 in the treatment of T-lymphocyte-mediated chronic inflammatory diseases until 2031. **We expect OSE to secure a new partnership agreement for FR104 in 2H20 – 1H21, providing a next catalyst for the stock. We currently project FR104 to reach the market in the US and EU in 2025, generating €43M in royalty revenues for OSE by 2030.**

Part 4: Valuation

Our valuation is based on 4 clinical programs: Tedopi, BI 765063, OSE-127 and FR104. While we emphasize the potential of BiCKI platform we currently do not include it in our financial valuation due to early stage of development and uncertainties associated with potential licensing opportunity. To value OSE shares we use a sum of the parts analysis based on risk-adjusted net present value (rNPV) of each asset, with estimates out to 2031. In order to reflect the significant risks associated with drug development, we are risk-adjusting NPV of respective programs based on the development phase and probability of reaching the market. To account for uncertainties in operations and drug development, we are assuming a 15% discount rate in our analysis, in-line with the discount rate we use for other similar development-stage biotech companies.

We estimate company's available cash position at €25M at the end of 2019, which we believe is sufficient to fund operations until the end of 2020. Furthermore, we believe that potential milestone payments from the pharma partners could become a significant source of non-dilutive funding in the near-term, albeit not guaranteed.

IP POSITION

OSE holds a portfolio of worldwide patents protecting BI 765063, Tedopi, OSE-127 and FR104. These include patents covering the composition of matter, drug targets, methods of use, and humanized antibody formulations with expiration dates from 2029 to 2032. The company licensed exclusively FR104 and OSE-127 from Inserm, and Tedopi from Takeda. Accordingly, to stay conservative in our estimates we project potential royalty revenue stream from OSE's clinical programs until 2031.

rNPV ANALYSIS

In our financial model, we assume that OSE would seek a commercial partner to market Tedopi in the US and EU in treatment of NSCLC post ongoing Atalante 1 study. We expect the potential partner to share the costs associated with further clinical development and to be fully responsible for costs associated with regulatory filings, as well as manufacturing, sales and marketing costs. Therefore, we project the OSE to receive milestone payments and royalty revenue on Tedpoi's sales. We project Tedopi to reach the market at the end of 2024, generating peak sales revenues of €596M by 2029. Taking in account the changes in study design, we conservatively model 15% PoS for Tedopi, in-line with phase II assets in oncology. Positive interim readout of Atalante 1 study could lead us to increase PoS to match the phase III status of the program.

Fig. 18: rNPV of Tedopi

	2020	2021	2022	2023	2024	2025	2026	2027	2028	2029	2030	2031
Royalties	0	0	0	0	5	49	96	131	149	125	70	20
Milestone payments	0	0	20	0	30							
Total revenues	0	0	20	0	35	49	96	131	149	125	70	20
Royalties to Takeda	0.0	0.0	-0.4	0.0	-0.7	-1.0	-1.9	-2.6	-3.0	-2.5	-1.4	-0.4
Total revenues to OSE	**0**	**0**	**20**	**0**	**34**	**48**	**94**	**129**	**146**	**123**	**69**	**19**
R&D	-10	-10	-5	-5	0	0	0	0	0	0	0	0
G&A	-3	-3	-3	-3	-3	-3	-3	-3	-3	-3	-3	-3
EBIT	-13	-13	12	-8	31	45	91	126	143	120	66	16
Taxes	3	3	-1	2	-5	-7	-14	-19	-21	-18	-10	-2
FCF	**-10**	**-10**	**11**	**-6**	**26**	**38**	**78**	**107**	**122**	**102**	**56**	**14**
Discount rate	0.87	0.76	0.66	0.57	0.50	0.43	0.38	0.33	0.28	0.25	0.21	0.19
Discounted FCF	**-8**	**-7**	**7**	**-3**	**13**	**16**	**29**	**35**	**35**	**25**	**12**	**3**
Discount rate (WACC)	15%											
Sum of FCF	156											
Terminal growth	-80%											
Terminal value	3											
Discounted TV	0.5											
NPV	**156**											
PoS	15%											
rNPV	**23.4**											

Source : Bryan Garnier & Co estimates

As the exact indications are yet to be defined, our estimates for BI 765063 are based on the clinical studies of competing assets that target the same pathway. Thus, we take end-stage colorectal, ovarian and bladder cancers as potential disease indications for BI 765063. We currently project the launch of the drug for end of 2025 and potential peak sales of €1.2B by 2030. We also note that BI is responsible for all development and commercialization costs. We expect the next milestone payment from BI in 2021 in association with either phase I/II readout or the start of the phase II study. We assign 10% PoS to BI 765063 program, in-line with the phase I programs in oncology.

Fig. 19: rNPV of BI765063

	2020	2021	2022	2023	2024	2025	2026	2027	2028	2029	2030	2031
Royalties	0	0	0	0	0	2	16	34	70	101	121	121
Milestone payments		20	20			50	50					
Total revenues to OSE	**0**	**20**	**30**	**0**	**0**	**52**	**66**	**34**	**70**	**101**	**121**	**121**
R&D	0	0	0	0	0	0	0	0	0	0	0	0
G&A	-1	-1	-1	-1	-1	-1	-1	-1	-1	-1	-1	-1
EBIT	-1	19	29	-1	-1	51	65	33	69	100	120	120
Tax	0	-3	-4	0	0	-8	-10	-5	-10	-15	-18	-18
FCF	**-1**	**16**	**25**	**-1**	**-1**	**43**	**55**	**28**	**59**	**85**	**102**	**102**
Discount rate	0.87	0.76	0.66	0.57	0.50	0.43	0.38	0.33	0.28	0.25	0.21	0.19
Discounted FCF	**-1**	**12**	**16**	**0**	**0**	**19**	**21**	**9**	**17**	**21**	**22**	**19**
Discount rate (WACC)	15%											
Sum of FCF	178											
Terminal Growth	-80%											
Terminal Value	22											
Discounted TV	4											
NPV	**182**											
PoS	10%											
rNPV	**18.2**											

Source : Bryan Garnier& Co estimates

We also assume that Servier will fully exercise its licensing option for OSE-127 in 2022 and will be fully responsible for all costs associated with phase III studies in UC and SS, as well as all regulatory filings, manufacturing, sales and marketing costs. We also note that nearly half of the clinical development costs for OSE-127 allocated to OSE is paid by BPI. We expect OSE-127 to reach the market for SS by the end of 2025 in the US and in 2026 in the EU and to secure marketing authorization for UC treatment in 2026 in the US and EU. We project OSE to receive double-digit royalties on the sales, as well as milestone payments. Conservatively, we assume PoS of 15%, lower than 21% for phase II assets in autoimmune diseases, due to historically high rate of failure of clinical studies in UC and SS.

Fig. 20: rNPV of OSE-127

	2020	2021	2022	2023	2024	2025	2026	2027	2028	2029	2030	2031
Royalties	0	0	0	0	0	3	30	62	94	138	139	96
Milestone payments												
pSS			10		20	30						
UC		0	10			20	30					
Total revenues to OSE	**0**	**0**	**20**	**0**	**20**	**53**	**60**	**62**	**94**	**138**	**139**	**96**
R&D	-5	-5	0	0	0	0	0	0	0	0	0	0
G&A	-1	-1	-1	-1	-1	-1	-1	-1	-1	-1	-1	-1
EBIT	-6	-6	19	-1	19	52	59	61	93	137	138	95
Tax	2	2	-3	0	-3	-8	-9	-9	-14	-21	-21	-14
FCF	**-4**	**-4**	**16**	**-1**	**16**	**45**	**50**	**52**	**79**	**117**	**117**	**81**
Discount rate	0.87	0.76	0.66	0.57	0.50	0.43	0.38	0.33	0.28	0.25	0.21	0.19
Discounted FCF	**-4**	**-3**	**11**	**0**	**8**	**19**	**19**	**17**	**23**	**29**	**25**	**15**
Discount rate (WACC)	15%											
Sum of FCF	158											
Terminal Value	17											
Terminal Growth	-80%											
Discounted TV	3											
NPV	**161**											
PoS	15%											
rNPV	**24.2**											

Source : Bryan Garnier& Co estimates

We also expect the company to secure a licensing deal for FR104 in 2H20 – 1H21, with upfront and milestone payments (mostly backloaded), as well as double-digit royalties. In accordance with the terms of previous partnership agreements, we expect the potential licensor to be eligible for all development and commercialization costs associated with the program. We project FR104 to reach the market for kidney transplantation in 2025 in the US and 2026 in the EU, generating peak sales of €286M by 2031. We assume 21% PoS for FR104, in-line with the phase II program in autoimmune diseases.

Fig. 21: rNPV of FR104

	2020	2021	2022	2023	2024	2025	2026	2027	2028	2029	2030	2031
Royalties	0	0	0	0	0	1	10	14	27	37	43	43
Milestone payments	5		20		30							
Total revenue to OSE	5	0	25	0	50	1	10	14	27	37	43	43
Royalties to Inserm	-0.1	0.0	-0.5	0.0	-1.0	0.0	-0.2	-0.3	-0.5	-0.7	-0.9	-0.9
Total revenues to OSE	**5**	**0**	**25**	**0**	**49**	**1**	**9**	**14**	**26**	**36**	**42**	**42**
R&D	0	0	0	0	0	0	0	0	0	0	0	1
EBIT	**5**	**0**	**25**	**0**	**49**	**1**	**9**	**14**	**26**	**36**	**42**	**43**
Tax	-1	0	-4	0	-7	0	-1	-2	-4	-5	-6	-7
FCF	**4**	**0**	**21**	**0**	**42**	**1**	**8**	**12**	**22**	**31**	**35**	**36**
Discount rate	0.87	0.76	0.66	0.57	0.50	0.43	0.38	0.33	0.28	0.25	0.21	0.19
Discounted FCF	**4**	**0**	**14**	**0**	**21**	**0**	**3**	**4**	**6**	**8**	**8**	**7**
Discount rate (WACC)	15%											
Sum of FCF	74											
Terminal Value	8											
Terminal Growth	-1											
Discounted TV	1											
NPV	**75**											
PoS	21%											
rNPV	**15.8**											

Source : Bryan Garnier& Co estimates

Assuming a 15% discount rate and a -80% terminal growth rate, we arrive at total rNPV of €80.7M. To this we add the €23M in net cash and cash equivalents held by OSE at the end of 2019 to arrive at a price target of €7.0 per share. Note, that we do not treat governmental loans as debt as the company is not paying interest.

Fig. 22: OSE

Product	Indication	Market Share	Peak Sales (in €M)	PoS	rNPV (in €M)	Per Share	% EV
Tedopi	NSCLC (post CPI)	20%	596	15%	23.4	1.6	22%
BI 765063	r/r Solid tumors (CRC, Ovarian, Bladder)	30%	1214	10%	18.23	1.2	18%
OSE-127	UC	7%	799	15%	23.3	1.6	22%
OSE-127	SS	10%	593	15%			0%
FR104	Kidney transplant	20%	286	21%	15.8	1.1	15%
Net Cash	End of 2019				23	1.56	22%
Fair Value						**7.0**	100%

Source : Bryan Garnier& Co estimates

Potential upsides to our estimates include: 1) earlier-than-expected market approvals; 2) additional indications for valuated programs; 3) market approvals in territories outside of the US and the EU; 4) higher-than expected product pricing or market uptake; and 5) launch of additional programs that we have not included in our current projections.

FINANCIAL SUMMARY

Net income and EPS. At the end of 1H19 the company reported €16M in revenues, mainly resulting from €15M milestone payment from BOEHRINGER INGELHEIM, and net profit of €0.5M. We note that due to IFRS revenue recognition standards €10M from Servier will be recognized in-line with occurred costs. In September, 2019, the company reported a payment from Bpifrance, associated with BI 765063 program, which was recorded as a loan of €4.8M and €0.6M of income. OSE has also received a grant of €0.8M from French National Research Agency. As a result, we project revenues of €16.4M and a net loss of €2.35M, or -€0.2M per share.

Cash. OSE held cash and cash equivalents of approximately of €26.5M and €6M in debt at the end of 1H19. We note that debt mainly constitutes government loans, on which the company does not pay the interest. In September, the company announced additional payments of €6.2M from government funds (BPI and French National Research Agency). Considering €10M milestone payment from Servier, which is booked on the balance sheet, we believe the company has sufficient funds to maintain operations and continue product development until the end of 2020.

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