Belgium | Pharmaceuticals | www.ucb.com

BUY

Price (08/09/2020)	EUR 5.90
Target price	11.00
Risk	High
Reuters	OSE.PA
Bloomberg	
Shares number (m)	15.01
Market cap. (m)	89
Cash Position 12/20e (m)	7
1 year price perf.	64.3%
Diff. with Euro Stoxx	69.6%
Volume (sh./day)	39;804
H/L 1 year	7.48 - 2.99
Free Float	56.2%
Emile Loria	23.7%
Dominique Costantini	13.2%
Alexis Peyroles	4.0%
Other management	3.0%

Company description

OSE is a clinical stage biotech focusing on the development of immunotherapy candidates adressing unmet medical needs in oncology and auto-immune diseases.

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OSE Immuno

Eat me !

A leader in immuno-modulation with 3 main assets:

- 1) Tedopi, a therapeutic vaccine with potentially opening a new therapeutic option to patients refractory to checkpoint inhibitor (CPI) in NSCLC;
- 2) OSE-127, an anti-IL-7 partnered to Servier and developed in ulcerative colitis (UC) and Sjogren's syndrome (SjS);
- 3) BI 765063, a first-in-class SIRP-α inhibitor developed in solid tumours.

Tedopi strikes back

- OSE has announced encouraging efficacy data of Tedopi in IO-refractory lung cancer patients. The 1-year survival rate reached 46% (n=63) versus 36% in the chemotherapy arm. At ESMO, on 18th September, OSE will announce the full read out.
- We believe that these results will be enough to attract a potential partner ready to lead a registration study. This trial has a decent chance of success given the docetaxel comparator arm which typically yields a low response rate (7-10%) and a short-lived mPFS (c. 4 months) in this patient population. If successful, we believe Tedopi may reach EUR 600m in sales by the late 2020s.

OSE-127 goes SjS

- We deem the restructuration of the deal with Servier could lead to a prioritization of the development of OSE-127 from UC towards SjS, an indication with less competitive pressure but with more clinical risks.
- Phase II in UC and SjS are expected to start in 2H20 but the timeline depends on the evolution of the pandemic. For now, we estimate that the combined peak sales for these two indications may reach EUR 1.3bn.

One drug with potential best-in-class status in many cancers

- BI 765063 (OSE-172) is a potential blockbuster drug targeting the CD47/SIRPα axis. This pathway has attracted significant investments and started to be clinically validated thus de-risking this asset developed in collaboration with Boehringer Ingelheim.
- OSE plans to release with BI the first clinical evidence in 1H21 (probably at the ASCO in June). We expect OSE shares to outperform as clinical data of BI 765063 will be released either in mono or in combination with other IO agents.

Initiation with a BUY, TP EUR 11

Our rNPV-based valuation (WACC: 15%) points to an enterprise value of EUR 150. Hence, we initiate coverage with a BUY and EUR 11 TP, offering 80% upside.

EUR	12/16	12/17	12/18	12/19	12/20e	12/21e	12/22e
Revenues	0.4	6.7	24.5	26.0	0.0	25.0	15.0
R & D	-5.1	-14.6	-15.1	-21.7	-21.5	-22.9	-25.9
EBIT	17	-13	5	-1	-27	-3	-17
Decl. profit	20.6	-10.4	5.5	-4.7	-27.1	-7.1	-18.0
EPS	1.44	-0.72	0.37	-0.31	-1.80	-0.47	-1.19
EV/Revenues	nm	7.6	1.5	2.4	nm	4.7	7.5
EV/R & D	-19.1	-3.5	-2.4	-2.9	-4.7	-5.2	-4.3
P/E	4.9	nm	9.2	nm	nm	nm	nm
Net Cash	16.0	7.6	8.0	16.1	-2.4	-7.9	-24.3

Degroof Petercam

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Resting on strong partnerships to prepare next-gen IO

Brief history of OSE

OSE was set up in June 2016 with the merger between Effimune and OSE Pharma. This led to the creation of a leading local player in immunotherapy with a diversified range of therapeutic candidates. The diversification of its pipeline allowed the company to reduce the development risk inherent to immunotherapy. Thanks to the merger, OSE is now developing its portfolio both for autoimmune and oncology diseases.

Today, OSE is a clinical stage company that is developing therapies in oncology and auto-immune diseases to target various immunological mechanisms. The company's leading asset is Tedopi, a therapeutic vaccine provided by OSE Pharma. Recently, Tedopi successfully demonstrated benefits to patients who have progressed post a treatment with a checkpoint inhibitor (CPI).

Effimune provided the rest of the portfolio, which is made up of: 1) BI 765063, an anti-SIRPα out-licensed to Boehringer-Ingelheim; 2) OSE-127, a clinical-stage candidate developed in autoimmune diseases and out-licensed to Servier; and 3) FR104, a clinical-stage candidate developed in transplantation and autoimmune diseases.

Besides these legacy assets, OSE unveiled in 2019 the development of its bi-specific platform "BiCki" which lay on the key backbone component anti-PD-1 and innovative targets selected by OSE. We estimate the first candidate from this platform, "BiCki Il-7", to enter in clinical stage within two years. Finally, based on its multi-epitope vaccine technology, OSE announced that its aims to develop a therapeutic vaccine against the SARS-CoV2.

Exhibit 1 **OSE immunotherapeutics pipeline**

PROGRAM	Indication	Pre-Clinical	Phase 1	Phase 2	Phase 3
IMMUNO-ONCOLOGY					
Tedopi® Neoepitopes	NSCLC				Positive Step-1 results Primary endpoint met
Tedopi®	Advanced pancreatic cancer			Combo with PD1 Opdivo® Ongoing	GERCOR / Bristol-Myers Squibb
BI 765063 (OSE-172) SIRPα-CD-47	Various cancers		Ongoing	Boehringer Ingelheim	
BiCKI® Bispecific anti-PD-1 & Innovative Targets	Various cancers	2020			
AUTO-IMMUNE DISEASES					
FR104 CD28	Autoimmune diseases & Transplantation			Phase 2 planning ongoing	
OSE-127 IL-7R	Ulcerative Colitis Sjögren's syndrome		Positive Phase 1 Results Q4 2019	2020	SERVIER
PROPHYLACTIC VACCINE PROGRAM					
CoVepiT Optimized Neoepitopes of SARS-CoV-2	COVID-19 vaccine	2020	Expected before end of 2020		

Source: OSE

A strategy focused on partnerships and rapid monetization

Since its inception, the strategy of OSE has been to elaborate partnerships in early-stage to provide a quick value-creation. Hence, the company decided to partner up for most of its clinical stage assets with large pharmaceutical companies:

- BI 765063: this promising oncology asset has been licenced-out in preclinical stage to Boehringer-Ingelheim for a total deal value of more than EUR 1.1bn in 2018.
- OSE-127: this clinical-stage candidate developed in Sjögren's syndrome (SjS) and ulcerative colitis (UC), has been partnered to Servier in December 2016.
- Similarly, FR104 was partnered with J&J, providing a EUR10m opt-in option payment to OSE Immunotherapeutics in July 2016. However, due to strategic considerations, J&J decided to return the rights to the asset to OSE in November 2018.
- Finally, OSE has inked several academics collaboration to pursue the development of its portfolio at a lesser cost or to strengthen its internal capabilities.

Exhibit 2 **Academic collaboration of OSE and consortiums**

Asset	Academic partners and consortiums	Comments
Tedopi	GERCOR, Bristol-Myers Squibb	OSE signed a collaboration agreement with the GERCOR in 2017, a non-for-profit academic group to study Tedopi in association with Opdivo in pancreatic cancer.
OSE-127	EFFI-MAB	Consortium that includes the French public medical research organisation (INSERM) and several public hospitals (APHP, Lille Hospitals). The budget allocated to Effimab is EUR 20m, of which EUR 9.1m is provided by the French public investment bank BPI to finance OSE-127 until the end of phase II. At the end of the phase II program, Servier will be entitled to opt-in.
BI 765063 (OSE-172)	EFFI-CLIN	Before being out-licensed to BI, BI 765063 was developed as part of the EFFI-CLIN consortium, set up in July 2017, which is still involve in early stage clinical development. The budget allocated to this consortium, funded by the Public French Investment bank (BPI), is EUR 9.2m.
OSE-703	Memorial Sloan Kettering Cancer Center	OSE entered into a research collaboration agreement with the memorial Sloan Kettering Cancer Center for the development of OSE-703 in June 2017 to assess OSE-703 in NSCLC preclinical-stage models.
BiCki	MAbSilico	In February 2020, OSE entered into a collaboration with MAbSilico, a deeptech startup. This deal aims to capitalize on artificial intelligence and deep learning to develop therapeutic monoclonal antibodies, including novel bispecific antibodies for BiCki and COVID-19 vaccine candidate.

Source: Degroof Petercam estimates

This strategy allowed OSE to provide a low-diluted R&D based economical model making OSE self-sufficient in term of cash since 2015 and the IPO of OSE Pharma. Hence, OSE received so far more than EUR 60m down-payments from its partnerships (Servier – EUR 25m, BI – EUR 30m, J&J – EUR 10m). This has allowed the company to build a solid preclinical stage pipeline and to continuously innovate.

Never give up in oncology while tackling innovative pathways

Within this segment, OSE develops Tedopi, its cancer therapeutic vaccine. OSE is currently developing this asset in checkpoint-refractory HLA-A2 NSCLC patients. We believe targeting this group of patients is attractive given the lack of available treatment options and the significant size of the population as it is estimated that >45,000 HLA-A2 NSCLC patients in the US/EU/Jap/China will be second-line checkpoint-refractory by 2021e. The full data package of the on-going phase III will be released on 18th September at ESMO. While top line results were already announced, the details regarding the duration of response and the survival rate at one year will be some of the key endpoints to watch out.

Next, OSE is pursuing the development of its early-stage pipeline. Most exciting program is BI 765063, a SIRPα monoclonal antibody targeting the CD47/SIRP pathway, developed in partnership with Boehringer Ingelheim. This program has had a dramatic increase in visibility as of late given its commercial potential and the fact that it has long been pursued by many other companies, including Forty Seven (bought by Gilead for USD 4.9bn) and I-MAB (inked a USD 2bn deal in September 2020 with Abbvie for their anti-CD47). The first upcoming clinical data that will be mostly disclosed at the ASCO'21 meeting in June next year will be therefore highly regarded and a key catalyst for OSE. As a clinically validated pathway with significant upside opportunity, we view BI 765063 as the key asset of OSE.

The fortune came back for Tedopi

Tedopi is OSE's leading asset. This therapeutic vaccine is currently developed in two indications:

- In NSCLC, after progression with a PD-1/PD-L1 checkpoint inhibitors.
- In pancreatic cancer, as an adjuvant therapy post-treatment with Folforinox for patients with stable diseases.

Tedopi has hit in its second shot in NSCLC

The cancer vaccine therapies are a notorious high-risk field with many setbacks seen in the past. While Tedopi failed to achieve a benefit in its initial targeted NSCLC population (2L post-chemotherapy), OSE recently announced positive results the subgroup of IO refractory/relapsing lung adenocarcinoma.

A multiepitope vaccine with positive phase IIb data

Tedopi is a therapeutic vaccine comprising nine epitopes that address five well-known tumour-associated antigens (TAAs). The tenth synthetic peptide is the pan-DR epitope (PADRE), a well-known helper T-Lymphocyte (HTL) epitope included to increase the magnitude and duration of cytotoxic T-lymphocyte (CTLs) responses. All these epitopes were designed to selectively recognise the HLA-A2 serotype. This serotype is present in c. 45% of the population according to OSE.

Accordingly, Tedopi was designed to induce CTL responses against TAAs frequently observed in NSCLC (non-small cell lung cancer), such as:

- Carcinoembryonic antigens (CEA): glycoproteins involved in cell adhesion, which is present in c. 70% of lung cancers.
- P53: Protein p53 is common in cancer and gained functions that help to contribute to malignant progression. The mutation of p53 is present in 40-50% of NSCLC (Korst & Crystal, 2003).

- HER-2/neu: HER2 is a member of the human epidermal growth factor receptor (HER/EGFR). This glycoprotein is involved in cellular expansion for some cancer cells. This mutation is found in 22-50% of NSCLC (Korst &Krystal, 2003).
- MAGE-2 (melanoma-associated antigen 2): MAGE-2 genes are almost universally expressed in body tissues.
- MAGE-3 (melanoma-associated antigen 3): Like MAGE-2, this antigen is expressed in many types of cancer.

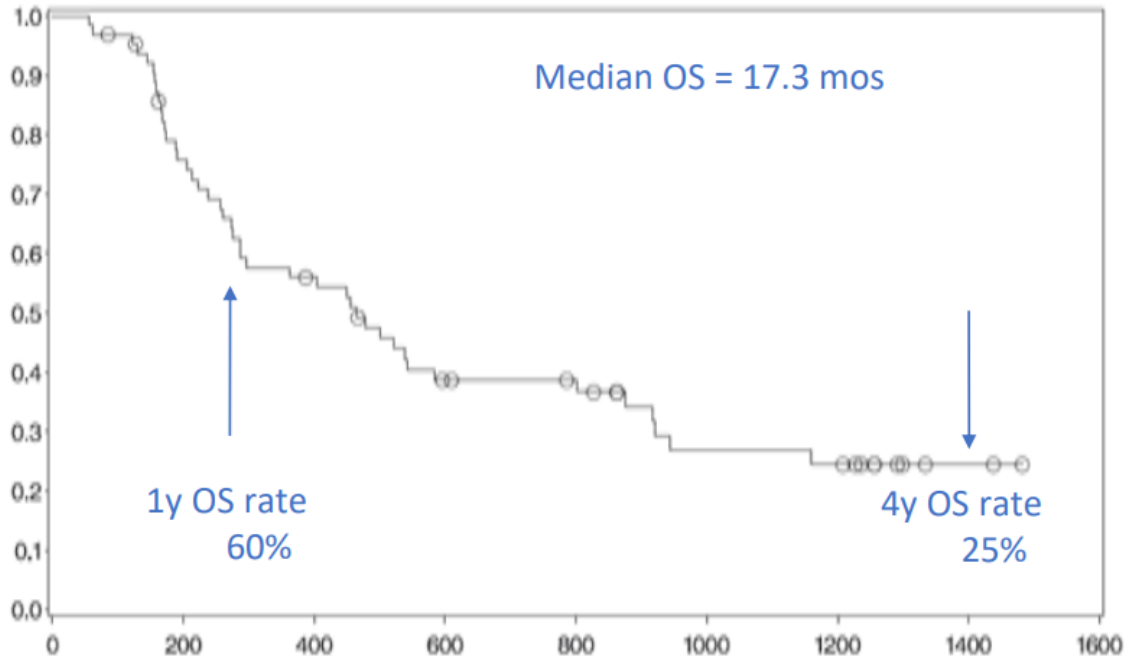
All these mutations are considered as poor prognosis factors for the development of the cancer, notably in NSCLC. **The originality of Tedopi's formulation is its ability to generate an immune response against ten epitopes present in five antigens while traditional therapeutic vaccine formulations only target one antigen.** This differentiation seems to provide further benefit to Tedopi compared to monovalent cancer therapeutic vaccines.

A positive phase IIb that has conducted to an ambitious phase III

In phase IIb Tedopi demonstrated an activity while the design of the study was unconventional. Thus, the trial was an open-label study, with a single arm that enrolled 135 patients, of which 64 were found to be HLA-A2+. These patients received Tedopi while the HLA-A2 negative patients served as observation. The 64 patients HLA-A2+ patients either failed to chemotherapy (92%) and a tyrosine kinase inhibitor (34%). Most of the patients had a NSCLC at a stage IV meaning the cancer has spread from where it started to at least one other body organ.

In phase IIb, Tedopi showed a promising median overall survival of 17.3 months with a manageable safety profile in pre-treated HLA-A2 positive patients with advanced NSCLC.

Exhibit 3 **Phase II results in pre-treated NSCLC patients (n=64)**



Source: M. Barve et al, Journal Clin. Oncol. 2008

One patient had a complete response and one had a partial response. While these results were promising, given that HLA-2 negative patients have generally a better prognosis, it was not enough to demonstrate a significant improvement versus the chemotherapy-controlled arm.

Exhibit 4

Arm	Number of patients	Median survival (months)	p-value	1-year survival	p-value	Response rate (complete and partial)
HLA-A+	64	17.3	not significant (p=0.063)	59%	not significant (p=0.089)	3%
HLA-A-	72	12		49%		n.a.

Source: OSE

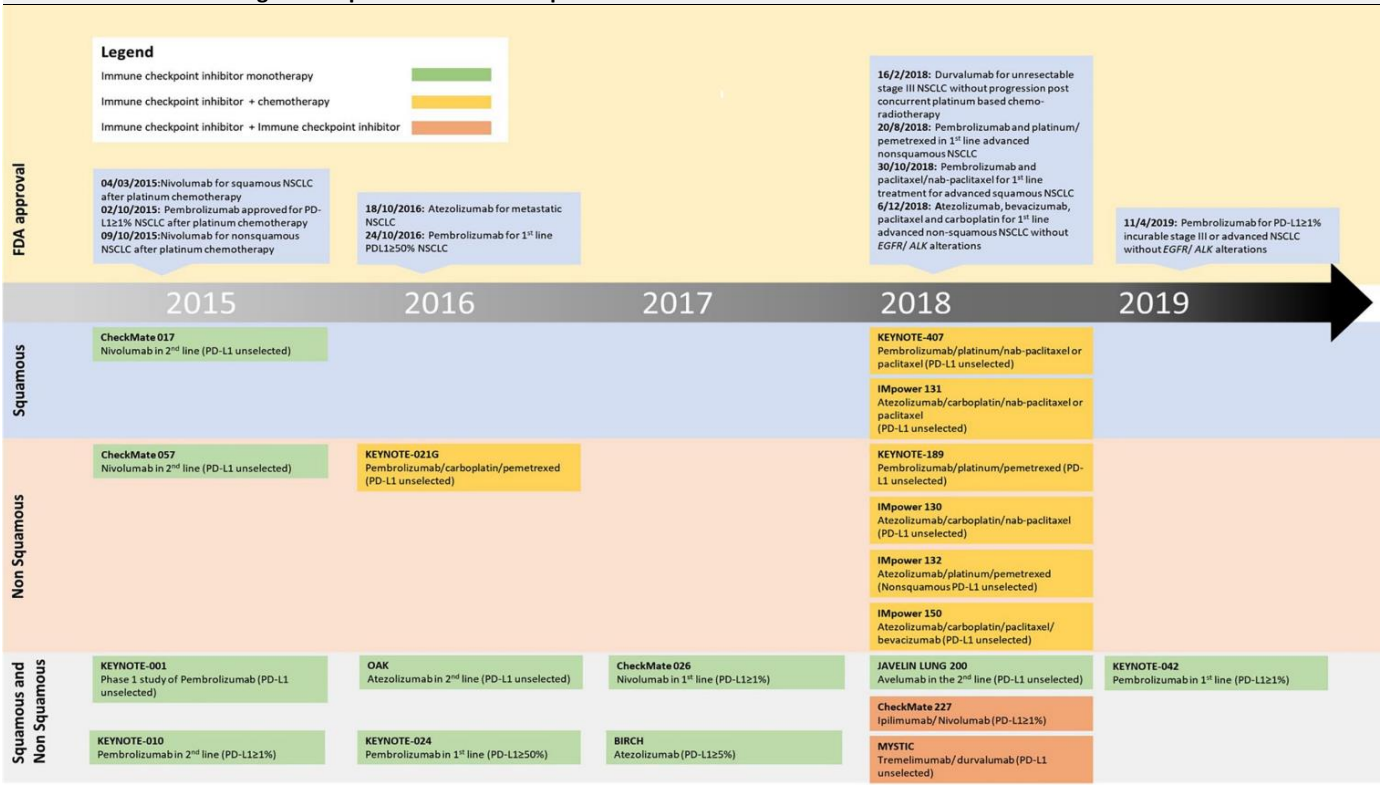
A fortunate phase III strategy for ATALANTE-1

Thanks to these results, OSE Pharma was established to launch a phase III, ATALANTE-1, assessing the efficacy of Tedopi in HLA-A2+ patients having progressed with a chemotherapy or a checkpoint inhibitor (CKI).

At the time of the launch of the trial, the chemotherapy was still the standard of care in first line and the CKIs were approved solely in second line.

Now, CKIs (namely Merck's Keytruda and Roche's Tecentriq, and more recently BMS's Opdivo[1]) are the standard of care in NSCLC for front line treatment either for squamous or non-squamous NSCLC regardless of PD-L1 status.[2]

Exhibit 5 The evolving landscape of immune checkpoint inhibitors in advanced NSCLC



Source: JL Low, RJ Walsh et al., 2019

Despite the advances in patient outcomes demonstrated by approved IO monotherapies in PDL1-high patients (KEYNOTE-024: 45% ORR and 10.3 mPFS) and IO/chemotherapy combinations in frontline non-small cell lung cancer (KEYNOTE-189: 48% ORR with 8.8 mPFS in all-comers), significant unmet need remains as (1) the percentage of patients who respond to approved CKIs treatment is still relatively low, with at least half of patients not achieving a

[1] https://news.bms.com/press-release/corporatefinancial-news/us-food-and-drug-administration-approves-opdivo-nivolumab-ye-1

[2] https://www.ncbi.nlm.nih.gov/pmc/articles/PMC6716180/

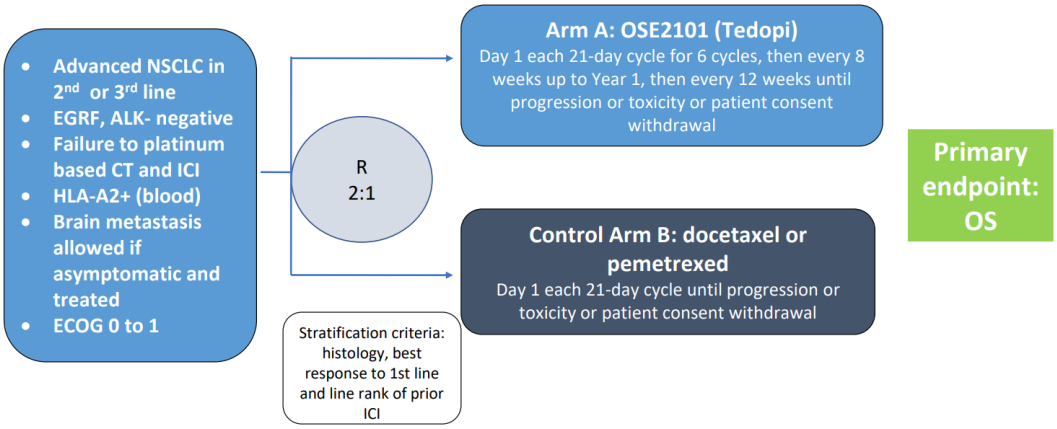
response (refractory patients), and (2) even in the patients who do initially respond, a large fraction will likely experience disease progression within a year (relapsing patients).

Therefore, a significant portion of patients that do not response or progress after an initial response are waiting for additional options.

ATALANTE-1 design

ATALANTE-1 initially enrolled patients with advanced NSCLC without EGFR or ALK mutations with progressive disease to chemotherapy or second-line immune checkpoint inhibitors (IC) were eligible. Patients had to be HLA-A2 positive. Treated and asymptomatic brain metastases were allowed. Patients were randomized 2:1 to receive 1ml SubQ TEDOPI Q3W for 6 cycles, then Q2M for the reminder of the year and finally Q3M or standard treatment with chemotherapy.

Exhibit 6 **ATALANTE-1 design study**



Source: OSE Immunotherapeutics

OSE had planned as well to realise sub-analysis regarding the histology (squamous vs. non-squamous NSCLC) as well as on the PD-L1 status. The trial was launched in January 2016 and aimed to initially enrolled 500 patients in Europe and in the US.

Unfortunately, in June 2017, following an IDMC meeting (Independent Data Monitoring Committee), OSE announced the suspension of the inclusion of patients within its trial due to a lack of efficacy of Tedopi in the chemotherapy-refractory arm.

Thanks to the initial design of ATALANTE-1, OSE shifted its strategy toward patients that failed CPIs treatment and could resume the recruitment of patients in this population in December 2017. The new designed of ATALANTE-1 comprised of two-step study. Part A planned to enrol 100 patients. The second step of the study would have recruited 225 patients accordingly to a prespecified median overall survival determined by the IDMC.

Part A data are promising but a confirmatory study is needed

In April 2020, OSE announced the positive topline results of Atalante-1 Part A study. Data demonstrated an improvement in the one-year survival rate in the Tedopi versus the controlled arm. The 12-month survival rate of 46% had a lower limit (33%) of the 95% confidence interval above the pre-specified futility boundary of 25%. Thus, OSE considered these preliminary data as successful.

Exhibit 7 **Step-1 results of Atalante-1**

Arm	Number of patients	1-year survival rate	p-value
Tedopi	63	46%	undisclosed, HR undisclosed, 95% CI [33% - 59%]
Control (chemotherapy)	36	36%	

Source: OSE

No other details have been disclosed and full read out is expected to be release at the ESMO congress in September. However, due to the COVID-19 pandemic, OSE decided to withdraw the second step of the study despite this positive first part. In case of success, the second step would have allowed to a conditional approval or even a full approval. Thus, gathering data on 100 patients similarly to the phase IIb is likely to be insufficient in our view to get the nod from the regulatory agencies.

Thus, the current prospect of Tedopi remains to be eluded in a stepping competitive environment. New treatments for patients that have progressed or did not respond despite the use a of checkpoint inhibitor in first line NSCLC treatment are indeed consider as a next key growth area.

A large unmet need in checkpoint-refractory NSCLC

According to company data most of the Stage IV NSCLC patients (~85%) progress on or after checkpoint therapy, which amounts to roughly 140,000 patients in the major markets.

These patients are an heterogeneous population that, with respect to checkpoint therapy, either; (1) never had an immune response or had an immediate adaptive resistance; or (2) sustained a clinical response, but then developed clonal resistance or acquired resistance over time. As patients become refractory to first-line PD1/PD1 plus chemo therapies, they are left with limited treatment options.

Based on HLA-A2 population, we estimate that roughly 40,000 patients may be addressed by Tedopi in the major pharmaceutical markets. We believe this market will growth as more patients are treated with CKIs in first line.

An intensifying competitive environment

Due to the recent launch of checkpoint inhibitors in front line NSCLC, few options have been tested for patients post anti-PD-1/PD-L1 setting so far, and most of the current tested options are still in clinical research setting. We discuss here below some of the most recent clinical outcome in ICI-refractory/relapse NSCLC patient population.

Initial early clinical trial readouts have begun to emerge involving novel IO combinations (either in second or third line).

In small sets of NSCLC patients post-anti-PD-1/PD-L1 treatment, Altor BioScience, a Nantworks company, (ALT-803 IL-15R agonist in combination with nivolumab) and Syndax (entinostat HDAC inhibitor in combination with pembrolizumab for monocyte high patients) have reported response rates in the 20-30% range while Merck (vorinostat HDAC inhibitor in combination with pembrolizumab) and Corvus (CPI-444 A2AR antagonist in combination with atezolizumab) have reported a modest response rate of ~10%.

At ASCO 2018, Garon et al, reported an ORR of 5%, an mPFS of 1.8 months, and an mOS of 8.4 months for 78 ICI-pretreated NSCLC patients who were treated with AstraZeneca's durvalumab plus tremelimumab. In an update abstract disclosed at the ASCO 2020, Leight et

al, concluded that the combination durvalumab and tremelimumab was not effective in ICI-refractory squamous lung carcinoma.[3]

The lack of 1-year survival data unfold by peers make it difficult to compare Tedopi with other tested candidates. However, based on historical data of chemotherapy there is a clear room for improvement on NSCLC CPI-refractory patients.

[3] https://meetinglibrary.asco.org/record/184935/abstract

Exhibit 8 **Existing data of ICI-refractory patients**

Company	Candidate	Target	Phase	Study	Subtype/ Group	PD-L1 status	Therapy	Dose	n	mOS (months)	mPFS (months)	ORR	Other
Mirati Therapeutics	Sitravatinib	TKIs	II	MRTX-500	All comers	All comers	sitravatinib + Opdivo	sitravatinib QD + nivo 240/480 mg IV Q2W:Q4W	73	18.1[4]	n.a.	29%	Median DOR: 9.2months
Roche / Exelixis	cabozantinib	TKIs	Ib/II	COSMIC-021 - cohort 7	All comers	All comers	Cabometix + Tecentriq	Cabo 40 mg PO QD and Atez 1200 mg IV Q3W	30	n.a.	4.2	27%	
Vaccinex	pepinemab	SEMA4D	Ib/II	NCT03268057	All comers	All comers	pepinemab + Bavencio	10 mg/kg pepinemab +10 mg/kg ave Q2W	29	n.a.	n.a.	7%	
Merck	vorinostat	HDAC	Ib	NCT02638090	All comers	All comers	Vorinostat + Keytruda	Vor 200/400 mg PO QW + pembro 200 mg IV Q3W	24	n.a.	n.a.	12%	PFS in ICI-refractory: 2.8months/ ICI-relapse: 4.6 months; OS in ICI-refractory: 6.8months/ ICI-relapse: 7.3 months
Syndax[5]	entinostat	HDAC	Ib	ENCORE-601 - cohort 2	All comers	All comers	Ent + Keytruda	Ent T 5 mg PO QW + pembro 200 mg IV Q3W	72	n.a.	2.8	10%	mPFS (5.3 months) and ORR (21.1%) to ent + pembro was improved for patients with elevated classical monocytes Median duration or response of 8 months (range 3-18 months)
ImmunityBio	ALT-803 (N-803)	IL-15	Ib	NCT02523469	Squ (10%) /non-Squ (90%)	All comers	Alt-803 + Opdivo	nivo 3mg/kg Q2W + ALT-803 Q1W	21	17.4	9.4	29%	PL-L1+ ORR: 75% (PD-1>50%); 0% (PD-1 >1-49%) ; PD-L1 - : 30%

Source: Company data, Degroof Petercam

[4] Note: Patients with PCB (Prior Benefit Cohort) on a checkpoint inhibitor as part of their last treatment regimen prior to enrollment. PCB is defined as either complete response, partial response or stable disease for ≥12 weeks. Subset of PCB patients (n=73) who received the combination as either 2nd or 3rd line of therapy after progressing on treatment with a checkpoint inhibitor.

As discussed above, with no details on Tedopi's patient population (number of prior therapies, PD-1 status, histology of tumours and whether or not tumour progression was documented) and really few unfold data seen so far from peers, it is significantly difficult in interpreting the clinical data across to the Atalante-1 trial. Keeping it mind, a 46% 1-year survival data rate seems a decent number for Tedopi to be achieved in ICI-refractory population.

Next step for Tedopi in NSCLC

Based on the results obtained from these first 100 patients we believe Tedopi is unlikely to be approved for the time being. A larger phase III will be needed. We think OSE will first try to find a partnership as the company won't take the risk for a large clinical trial. To appropriately power the trial for overall survival, the key clinical endpoint for cancer studies, we believe the study will have to recruit c. 600 patients, similarly to what it is seen with the on-going phase III of Mirati Therapeutics for instance (first read out expected by YE21).

Pancreatic cancer remains a difficult indication

Besides the ICI-refractory population, Tedopi is also developed in pancreatic cancer. This cancer is a notoriously difficult one to address. Despite being less common than many other tumour types, pancreatic cancer is now the third leading malignancy in terms of mortality with an estimated ~44K deaths expected in the US in 2019.

Approximately 80% of patients present with unresectable disease, inclusive of roughly 50% with metastatic disease and another 30% with locally advanced unresectable disease. Pancreatic cancer mortality rates lead all other cancers, with five-year survival rates of just about 8%.

The front-line treated metastatic patient population consist of approximately 28k patients in the US and 36k patients in Europe. An adjuvanted-treatment post chemotherapy for patients with stabilised disease could offer an attractive market to Tedopi if it proves to increase the overall survival.

However, the barriers to drug development and clinical trial success have been significant and include (1) the aggressive course of disease, (2) a dense fibrotic stroma that prevents optimal drug delivery or diffusion of active immune cells, and (3) a willingness to start Phase III studies based on small, often uncontrolled Phase II trials data in selected healthier populations.

The pancreatic cancer is therefore a notoriously hard-to-treat cancer with many setbacks seen over the past couple of years.

Exhibit 9 **Pancreatic cancer past clinical developments**

Product	Company	MOA	Setting	Results	outcome
Lynparza	Astra/Merck	PARPinh	BRCA+, 1L maintenance	Met PFS (7.4 vs 3.8 months (HR 0.53 [95% CI 0.35-0.82]; missed OS (18.9 vs. 18.1 months; (HR 0.91; 95% CI, 0.56 to 1.46; P=0.68).	Approved in Dec. 2019.
Masisinib	AB Science	TKI	1L +/- gemcitabine	n.a.	Final analysis planned in 2020
Graspa	Erytech	Asparaginase receptor stimulant	2L	n.a.	Interim analysis due Q3'20
Pamrevlumab	FibriGen	anti-VTGF	1L	n.a.	Primary completion 2022
PEGPH20	Halozyme	recombinant PH20 hyaluronidase	1L +/- Abraxane/gem	mOS (11.2 months compared to 11.5 months, HR=1.00, p=0.9692)	Failed
Imbruvica	J&J	BTK inh	1L +/- Abraxane/gem	n.a.	Failed
napabucasin	Sumitomo Dainippon	STAT3 inh	1L +/- Abraxane/gem	n.a.	Discontinued post futility analysis
pegilodecakin	Eli Lilly	PEG IL-10	2L +/- Folfox	n.a.	Failed
Jakafi	Incyte	JAK inh.	2L capcitabine +/- ruxolitinib	n.a.	Discontinued post interim analysis
Algenpantucel-L	Lumos Pharma	Cancer vaccine	Resected, adjuvant, Gem +/- algenpantucel-L	median OS 30.4 vs 27.3 months	Failed

Source: Degroof Petercam, company data

Of note, ICIs have also failed as standalone therapy to show any responsiveness to locally advanced or metastatic pancreatic cancer. The on-going combination include agents targeting TAMs, to modulate the tumour immune-suppressive environment notably.[6] However, this approach also shown some poor success so far.[7]

Phase II design the adjuvanted market post-chemotherapy with Folforinox

In 2018, OSE entered in partnership with the GERCOR (a leading clinical research association that is sponsoring the trial) and BMS (that provides Opdivo), to assess Tedopi in pancreatic cancer.

OSE's Phase 2 trial, called TEDOPaM, is intended to evaluate Tedopi alone or in combination with Opdivo as a maintenance therapy for patients who achieved stable disease after receiving standard 1L chemotherapy, Folfirinox, for four months (8 cycles of treatment per Folfirinox).

Exhibit 10 **Design of the TEDOPaM study**

Arm A (reference): FOLFIRI (n = 52)	IV; FA 400 mg/m^2, Iri 180 mg/m^2, 5FU bolus 400 mg/m^2 + continuous 2400 mg/m^2/46h
Arm B: Tedopi (n = 52)	Subcutaneous injection on D1 Q3W/6 doses then Q8W until month 12 [M12] then Q12W up to M24
Arm C: Tedopi + nivolumab (n = 52)	Tedopi + nivolumab 360 mg IV on D1 Q3W/6 doses then 480 mg Q4W up to M24

Source: Annals of Oncology

[6] https://molecular-cancer.biomedcentral.com/articles/10.1186/s12943-019-0966-6/tables/1

[7] https://immuno-oncologynews.com/2020/03/06/cabiralizumab-plus-opdivo-failed-halt-progression-advanced-pancreatic-cancer-phase-2-trial/

In the trial, Tedopi-based approaches will be compared with Folfiri, a standard maintenance chemotherapy. The primary endpoint is the OS at 1 year. An interim analysis is planned after the enrolment of 60 patients (20 per arms).

The study started in early-2019. However, due to the COVID-19, the enrolment of new patients has been halted since March 2020. We therefore expect topline results from this study in late-2021 at the earliest.

Folfirinox is a highly active chemotherapy but with greater safety concerns

FOLFIRINOX is a combination of chemotherapies that includes oxaliplatin, irinotecan, fluorouracil, and leucovorin. While Folfirinox has been shown to be more efficacious than gemcitabine and the GemAb (Gemcitadine + Abraxane) combination, it has a poor market penetration in the US/EU5 owing to its low tolerability (neutropenia, thrombocytopenia, nausea, diarrhea, neuropathy). Moreover, the lack of commercial promotion (all APIs are already genericised) make this chemotherapy combination reserved for the healthier patients. We believe this equates to roughly a third of currently front-line treated mPDAC patients.

To the opposite, the combination Gemcitadine/Abraxane (approved in September 2013 in the US)) is now considered the standard of care for frontline treatments of pancreatic cancers.

Thus, we believe the currently addressable population of Tedopi will need to be extended with follow up clinical trials as an adjuvanted therapy post the combination Gemcitadine/Abraxane or post a surgical-resection in order to expand its opportunity in pancreatic cancer. For now, besides a high clinical risk, we also consider the target population to be limited (c. 3,000 patients in the US/EU5).

A differentiated covid-19 vaccine leveraging Tedopi technology

Using its multi-epitope technology Memopi which was used to develop Tedopi, OSE announced in May 2020 it was working on a prophylactic COVID-19 vaccine candidate. The development of this vaccine is being made in collaboration with MAbSilico to accelerate thanks to an artificial intelligence program the screening of potential epitope that would trigger an immune response. First preclinical results of this vaccine are expected by year-end.

While we view this approach as interesting, we believe that OSE is far behind the on-going competitive race in the search for a COVID-19 vaccine. The company received EUR 200k from the Nantes Métropole to develop this vaccine. This is to put into perspective with multi-billions grants and funding revived by other developers of traditional (i.e Novavax) or next-generations platform peers (i.e. Moderna, BioNtech, AstraZeneca etc..).

First preclinical results have been released in August, highlighting the interest of this multivalent candidate. A first clinical study is expected by year-end. However, while competition is already starting their phase III programs, this program will need to quickly attract some interests from partners in order to prove its viability in a highly competitive environment.

BI 765063: a potential best-in-class CD47/SIRP axis candidate

BI 765063 belongs to the category of candidates targeting the CD47/ SIRPα pathway. This is a myeloid checkpoint pathway transforming myeloid immune suppressive cells into effector cells within the tumour micro-environment (TMEs).

In April 2018, OSE partnered with BI to develop BI 765063 for a total deal value of up to EUR 1.1bn, making that deal one of the largest-ever for a pre-clinical stage asset in oncology.

A phase 1, dose finding study, is ongoing in patients with advanced solid tumours. BI 765063 is either administered as a single agent and in combination with BI's PD-1 proprietary antagonist BI 754091.

While OSE do not retain anymore the commercial rights on this asset, we believe the cash-inflow from BI will allow to develop its early-stage pipeline (i.e. the BiCki platform), hence further contributing to the creation of value for existing shareholder without dilutive effect.

The myleloid-derived cells modulation: a strong rational in tumours

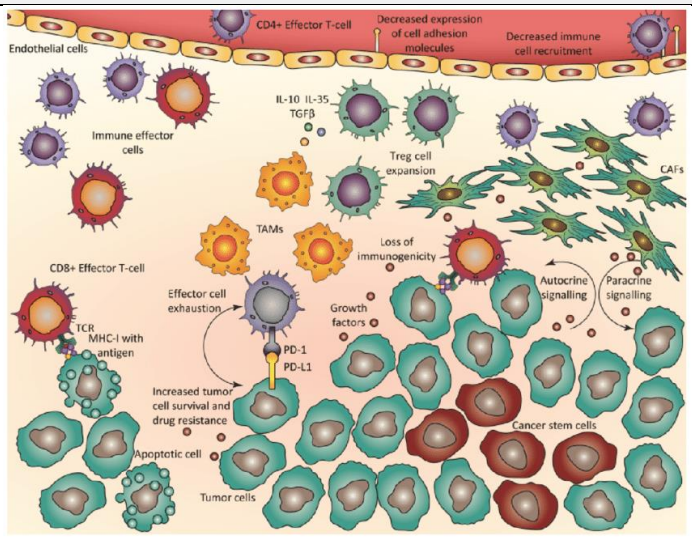
The innate and adaptive components of the human immune system form a complex organization of tissues, cells and proteins that serve to protect the body from invading pathogens. For the body to mount an effective response to a foreign cell or a cancer cell, the innate and adaptive immune systems must generally work in concert.

It is becoming increasingly evident that non-malignant immune cells in the TMEs can promote tumour growth by secreting pro-angiogenic factors and growth factors, and by weakening effector T-cell function by secreting immunosuppressive cytokines.

TMEs comprise innate and adaptive immune cells such as T cells, dendritic cells and macrophages.

Among these immune cells, recent preclinical and early clinical evidence has shown that the presence of myeloid-derived suppressor cells (MDSCs), tumour-associated macrophages (TAMs), regulatory T cells (Tregs) are associated with poor prognosis in a number of solid tumour indications. The presence of these cells is also correlated with a reduced benefit from checkpoint inhibitors. Indeed, these cells are recruited the cancer cells to create an immuno-suppressive environment turning the cancer toward a "cold-tumour".

Exhibit 11 **The tumour microenvironment**



Source: Tarasov et al, 2019

Overall, suppressive immune cell type in the microenvironment of solid tumours are often found, where they promote tumour growth, metastases, angiogenesis, while inhibiting anti-tumour immune responses.

Thus, while recently approved immune-modulator for the treatment of cancer primarily explored the T-cell mechanisms (i.e. PD-1/PD-L1 axis), existing research focus on the cells surrounding the TMEs to improve T cells effectiveness.

Macrophages as effector cells

Macrophages, a key component of the innate immune system, serve as a first line of immune defence and initiate an immune response based on non-specific signals of foreign or abnormal cells. They also play a key role in alerting cells of the adaptive immune system (i.e. T cells) to the presence of potential targets such as cancer cells.

Macrophages are a type of white blood cell typically broken down into two subtypes depending on their role in either healing (M2) or immune function (M1).

In a classic immunologic model, M1 macrophages carry out their immune function by recognising foreign pathogens, engulfing them in a process known as phagocytosis and initiating a cascade of chemical signals that promote inflammation and recruit the adaptive immune system (i.e. T cells).

Healing macrophages, classified as M2 macrophages, typically arrive a couple of days after the initiation of an immune response to decrease inflammation, increase the formation of new blood vessels, and facilitate tissue following the elimination of pathogens.

Role of myeloid suppressive cells in TMEs

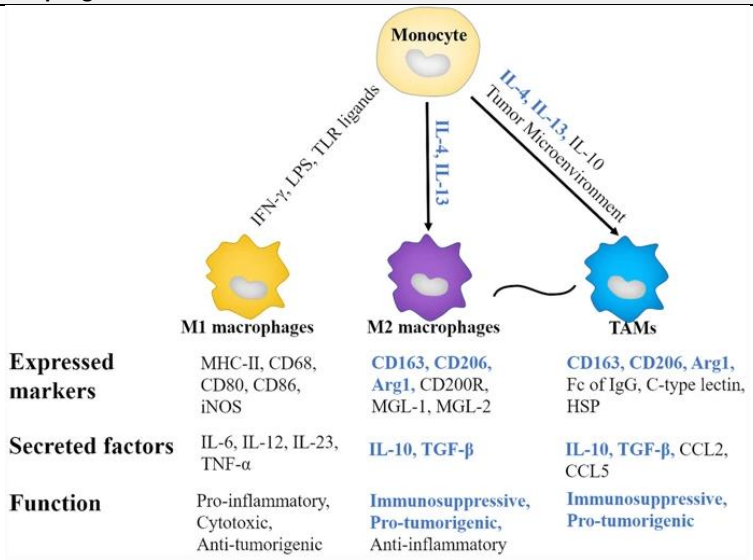
Normally, common myeloid progenitor (also called immature myeloid cells) migrate to different peripheral organs, where they differentiate exclusively into dendritic cells and macrophages type M1 or polymorphonuclear cells (also named granulocytes).

However, several factors produced in many pathological conditions, notably cancers, promote the accumulation of immature myeloid cells (M2 macrophages), prevent their differentiation and induce their activation. These cells exhibit immunosuppressive functions after activation and were named MDSC (Gabrilovich et al, 2007).

The second category of myeloid cells found in TMEs, called TAMs, is involved in almost all of the tumour development pathways:

- Tumour cell proliferation.
- Angiogenesis (formation of cancer vessels).
- Tumour cell invasion.
- Immunosuppression through Treg induction and T cell inactivation.

Exhibit 12 **Macrophage differentiation and their characteristics**



Source: Chen et al, 2019

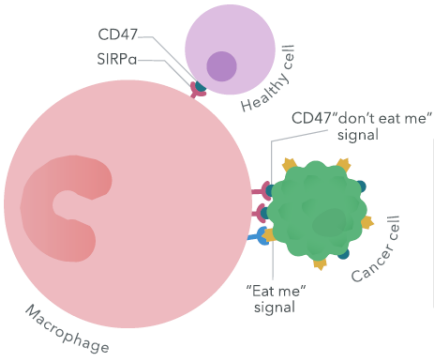
TAMs are the most abundant population of tumour-infiltrating immune cells in TMEs and are increasingly seen as an important target form tumour suppression.

BI 765063 mechanism of action: a dual innate / adaptive immune promoting effect

Myeloid cells selectively express SIRPα, an tyrosine associated inhibitory receptor (also named CD172a), which controls myeloid functions. The ligand of SIRPα is CD47. CD47 is expressed in almost all normal human cells.

In case of tumour, it may be an up-regulation of CD47 which leads to several adverse effects for the immune-control of the tumour. Expression of CD47, and its interaction with SIRPα, leads to the inhibition of macrophage activation and protects cancer cells from phagocytosis (the macrophage aims to "eat" the cancer cells). Thus, cancer cells use CD47, a "don't eat me" signal, in order to evade detection by the immune system and subsequent destruction by macrophages.

Exhibit 13 **Macrophage differentiation and their characteristics**



Source: Forty Seven

Targeting the CD47-SIRPα pathway could eliminate tumour cells via multiple mechanisms. Blocking CD47-SIRPα interactions has been shown to promote the destruction of cancer cells by phagocytes, including macrophages and neutrophils.

Furthermore, there is growing evidence that targeting of the CD47-SIRPα axis **may also promote antigen-presenting cell function through dendritic cells and thereby stimulate direct tumour cell killing via antibody-dependent cell-mediated cytotoxicity**.

According to the literature, it also promotes human subtype M1 macrophages, which has anti-tumour effects (pro-inflammatory), while decreasing the M2 subtype (pro-tumoural effect).

Finally, as the CD47 has been found in multiple tumours hence, this target holds strong promise for multiple indications. However, CD47 is also ubiquitously expressed at low levels on normal cells. This poses potential safety challenges.

Competitive environment

CD47 has emerged as one of the most promising immuno-oncology targets. It has attracted many players and an intensive investment. Most recently, Gilead acquired Forty-Seven for USD 4.9bn strengthening the sentiment that TMEs-modifying candidates are a hotspot for large pharmaceutical companies.

Most of the current are focusing on binding the CD47 pathway. This is the case for:

- Forty Seven's magrolimab
- Celgene's CC-90002
- TG-Therapeutics's TG-1801

Exhibit 14 The CD47-SIRPα hotspot pathway

Company name	TRILLIUM				Forty Seven		ALX ONCOLOGY		Celgene		arch oncology	TG Therapeutics	OSE IMMUNOTHERAPEUTICS
Candidate	TTI-621		TTI-622		Magrolimab	FSI-189	ALX148		CC-90002	CC-95251	AO-176	TG-1801	OSE-172
Molecule	WT SIRPαFc fusion protein		WT SIRPαFc fusion protein		CD47 mAb	SIRPα mAb	High aff. SIRPαFc fusion protein		CD47 mAb	SIRPα mAb	CD47 mAb	Bi-spec. Ab CD47/CD19	SIRPα mAb
FC istoype	IgG1		IgG4		IgG4	IgG1 (dead Fc)	Inert IgG1		IgG4	-	IgG2	IgG1	IgG4
RBC binding	No		No		Yes	No	Yes		Yes	No	Mnimal	No	No
Monotherapy CRs	Yes		No		No	-	No		No	No data	No data	No data	No data
First-in-human	Feb-16		Jun-18		Aug-14	2Q20	Feb-17		Mar-15	Jan-19	Feb-19	Mar-19	Jun-19
Development stage	P1b		P1a	P2	P1	Preclinical	P1		P1	P1	P1	P1	P1
Indications	R/R Heme (IV dosing) - monotherapy	Solid (intratumoural dosing +/- ICIs) - monotherapy	Heme (r/r lymphoma) - monotherapy	Heme (1L AML[5]/ 1L MDS[6]) + aza	Solid (mono; combo w/ avelumab, cetuximab)	-	NHL (combo w/ritu)	Solid tumors (mono; combo w/ pembro, trastu)	NHL (+ritu)	Solid + heme	Solid tumour	Heme B-cell malignancies	Solid tumour (single agent; combo w/ BI754091)
Status	Study recruiting	In planning	Study recruiting	Study recruiting	Study recruiting	-	Study recruiting		Active, not recruiting	Study recruiting	Study recruiting	Study recruiting	Study recruiting
Highest dose	0.5 mg/kg QW	0.5 mg/kg QW	8 mg/kg QW	30 mg/kg QW	45 mg/kg QW (priming dose of 1mg/kg)	-	15mg/kg QW	30 mg/kg Q2W	4 mg/kg QW	n.a.	n.a.	n.a.	n.a.
n	c. 200	n.a.	19	24 MDS + 22 AML	34 (ave)+78 (cetu)	-	33	89	24	230	90	16	116
ORR	CTCL-MF[1]: 23% CTCL-SS[2]: 13% PTCL[3]: 18% DLBCL[4]: 29%	n.a.	11% (1 CR + 1 PR (DLBCL))	+ aza: 92% MDS / 64% AML	- + ave (ovarian): 0% - + cetu (mCRC, KRASwt): 6% / KRASm: 0%	-	+ ritu: 45% (average 10/15 mg/kg)	+pembro 2L HNSCC[7]: 40% in ICIs naïve and 0% in ICIs refractory + trastu HER2+ Gastric: 21% + pembro 2L NSCLC: 5%	+ritu: 13%	n.a.	n.a.	n.a.	n.a.

Source: Degroof Petercam, notes: 1) CTLC-MF: cutaneous T-cell lymphoma mycosis fungoides; 2) CTLC-SS: cutaneous T-cell lymphoma Sezary syndrome; 3) PTLC: Peripheral T-cell lymphoma; 4) DLBCL: Diffuse large B-cell lymphoma; 5) AML: acute myeloid lymphoma; 6) MDS: myelodysplastic syndrome; 7) HNSCC: head and neck squamous cell carcinoma

Other CD47 player from China are also emerging such as I-mab biopharma's TJC4 (acquired in September 2020 by Abbvie in a USD 2bn deal) and Innivent's IBI188. **The only companies that are developing a dedicated SIRPα mAb in clinical stage are:**

- OSE/BI's BI 765063
- Forty Seven's FSI-189
- Celgene's CC-95251

No data have been published so far by these SIRPα mAb candidates. Moreover, while CD47 inhibitors have shown a clear success in hematologic cancer, notably in combination with azacytidine for magrolimab, the data in solid tumours are still limited.

CD47s have shown potent activity in haematologic cancer

The only CD47 inh. that has shown a strong activity (but in hematologic cancers) in monotherapy so far are from Trillium. Magrolimab showed a strong efficacy but through a combination study with azacytidine in MDS/AML. Trillium's TTI-621 is a fusion protein consisting of a SIRPα receptor linked to an IgG1 Fc region that binds to CD47 on tumour cells and abrogates the suppressive "don't eat" signal. In addition to suppressing CD47, TTI-621's Fc region also acts as a pro-phagocytic signal though binding to the SIRPα. At this stage, TTI-621 has been the only CD47- or SIRPα-targeting therapeutic to demonstrate complete responses as a monotherapy in clinical testing, but still in heme cancers.

Data in solid tumours are still limited

In solid tumours, Forty Seven's magrolimab has released some data with poor outcome in combination with Erbitux (cetuximab) in mCRC (6% ORR). In ovarian cancer in combination with Bavencio (avelumab) magrolimab also showed some limited activity (0% ORR).

Similarly, another CD47, ALX Oncology was tested in solid tumours in combination with Keytruda with limited success so far compared to what is seen with Keytruda in targeted indications except for HNSCC 2L (40% vs 18%[8] ORR). That did not prevent that company to raise USD 185m in July 2020 during its IPO.

BI 765063 holds several advantages versus the competitors

We hold the view that BI765063 offers key competitive advantages compared to its peers:

1. A potential better safety profile.

The first wave of clinical-stage CD47 antibodies were found to bind to red blood cells (RBCs) and cause significant hematologic adverse effects, such as severe anaemia and cytopenia. Indeed, CD47 is widely expressed and is most highly expressed in hematopoietic cells and tumour cells. For hematopoietic cells, CD47 protects young cells from being destroyed although eventually the cell becomes less able to prevent phagocytosis and older cells get eaten by macrophages in the spleen. By blocking these CD47 receptors, the CD47 mAb induce the innate immune system (mainly macrophages) to destroy the hematopoietic cells.

Thus, Forty Seven initially observed high rates of grade 3 anaemia in dose escalation and has addressed this phenomenon by initiating patients on a 1 mg/kg "priming" dose to reduce anaemia incidence when administering a therapeutic dose of 30-45 mg/kg. Despite this priming dose, any-grade anaemia on magrolimab is on the order of 40% event.

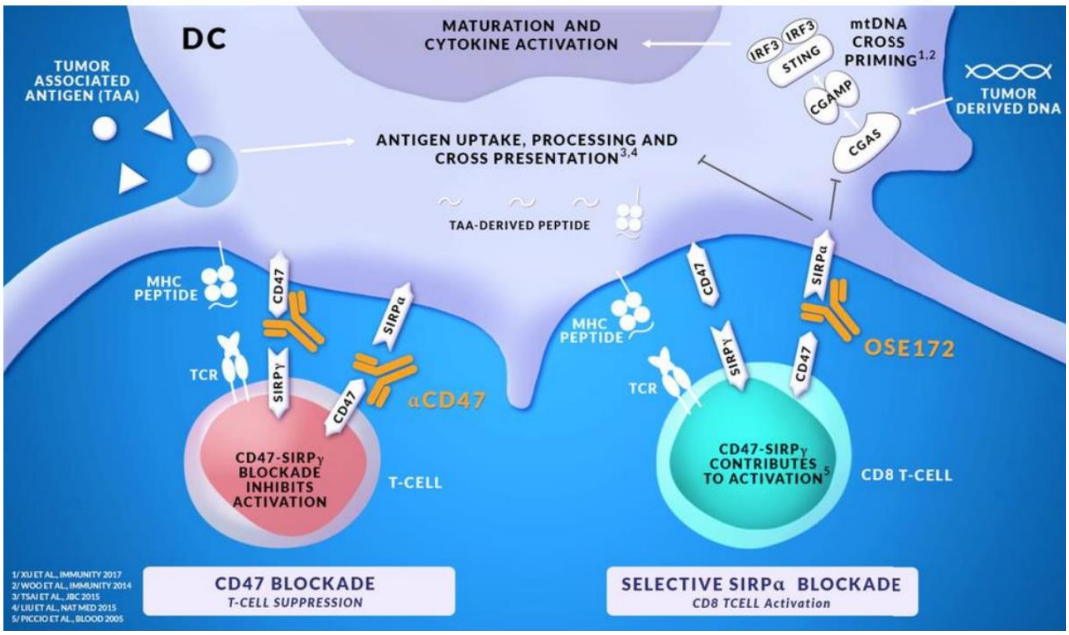
[8] https://www.ncbi.nlm.nih.gov/pmc/articles/PMC6048158/

By selectively inhibiting the SIRPα axis, less haematological side effects should be observed with BI7650663.

2. BI765063 does note block T cell responses and increase the ADCC response.

BI7650663 do not disturb the SIRPγ-CD47 axis to the opposite of the anti-CD47. The SIRPγ-CD47 has described to play a role in human T cell proliferation and migration. It is expressed on T-cells and activated NK cells and binds CD47 at low affinity. Preclinical works have shown that that blocking the SIRPγ-CD47 interaction with specific antibodies against either CD47 or SIRPγ impaired T-cell activation by CD47+ antigen presenting cells.[9]

Exhibit 15 BI765063 as an antagonist to SIRP do not blockade T cell activation



Source: OSE Immunotherapeutics

Thus, by preventing this blockade though CD47, BI7650663 is likely to strengthen the immune response versus anti-CD47. To the opposite, the selective inhibition of SIRPα has been shown to potent the anti-tumour activity[10].

A landmark partnership with BI an ambitious emerging IO player

The interest of the SIRPα was confirmed in early-2018 when BI acquired the global rights of OSE-172 (now BI 7650663) for up to EUR 1.1bn in milestone payments, plus EUR 15m upfront with royalties on future net sales. The first-in-man study announced in June 2019 triggered a EUR 15m payment to OSE.

Preclinical studies in murine tumour models have demonstrated the clinical benefit of BI 7650663 as monotherapy, but suggest clinical outcomes may be enhanced through combination therapy with an adaptive PD-1 ICI, or with a costimulatory agent (such as anti-4-1BB monoclonal antibody[11]), to provide dual activation of innate and acquired immunity.

[9] Piccio L, Vermi W, Boles KS, Fuchs A, Strader CA, Facchetti F, et al. Adhesion of human T cells to antigen-presenting cells through SIRPβ2-CD47 interaction costimulates T-cell proliferation. Blood. 2005;105(6):2421–7

[10] Liu et al (2016) Inhibition of SIRPa in dendritic cells potentiates potent antitumour immunity. Oncoimmunology 5, e1183850

[11] Gauttier V, et al. Cancer Res 2018;78(13 Suppl.): Abstract 1684.

Hence, while few details have been disclosed on the clinical development of BI 7650663, we note that multiple combination may be planned, foremost, with the anti-PD1 developed by BI, BI 754091.

Exhibit 16 Combination contemplated by OSE/BI for BI7650663

INTERNAL	PARTNERSHIPS
Anti-PD1 Anti LAG 3 SMAC mimetics IL23-i	Vira Therapeutics (oncolytic viruses) CureVac (mRNA vaccines)

Source: OSE Immunotherapeutics

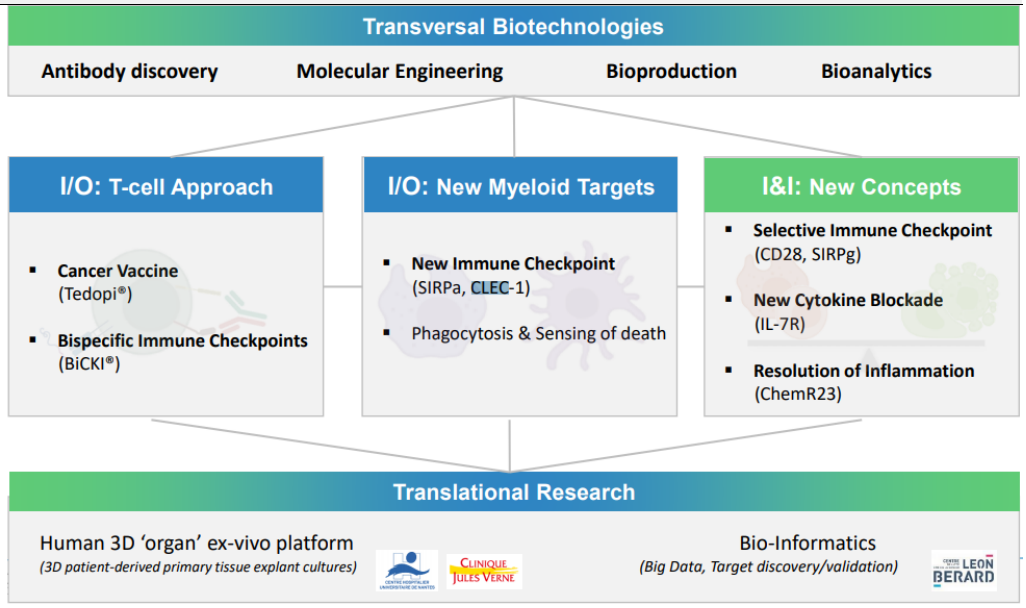
Thanks to this partnership with BI, OSE bears almost no further cost for the development of BI 7650663. Moreover, as part of the consortium Effi-CLIN, OSE was eligible to EUR 9.2m grants of which EUR 5.4m were vested in September 2019 further limiting the cash outflow for this project.

Early-stage oncology programs already prepare the field

OSE's key know-how is to bring multiple candidates in clinical stage and partnered them in order to limit its risks. To that purpose, the company is developing two other preclinical oncology assets (a CELC-1 and an anti-IL-R) as well as a novel bispecific platform called BiCKI.

Further developments are being made with several collaboration partnerships to span the whole spectrum of on-going trends in the elaboration of immune-oncology developments.

Exhibit 17 OSE's Research Pillars



Source: OSE Immunotherapeutics

We view the BiCKI platform as highly promising to create shareholder's value through partnership.

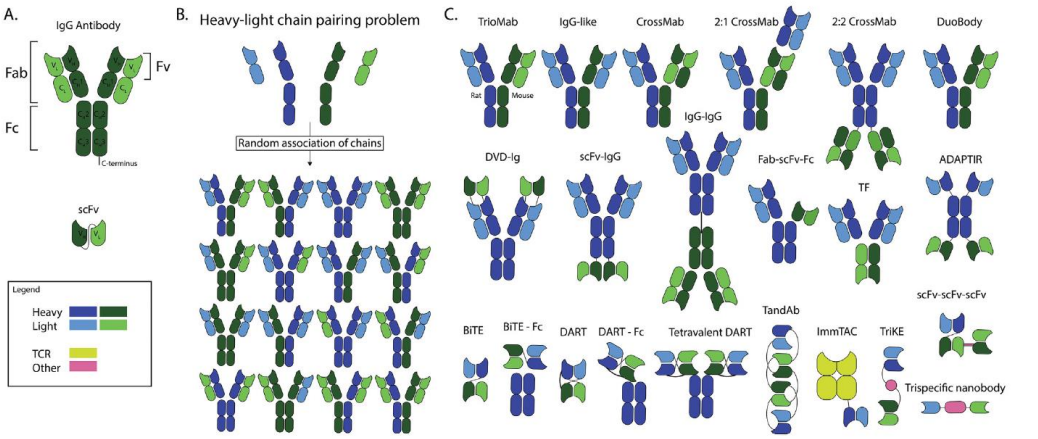
BiCKI is likely to attract interest as bispecific is a highly-regarded technology

Bispecific antibodies BsAbs combine specificities of two antibodies and simultaneously address different antigens or epitopes. The goal of BsAbs can be multiple:

- bsAbs with 'two- target' functionality can interfere with multiple surface receptors or ligands associated, for example with cancer, proliferation or inflammatory processes.
- bsAbs can also place targets into proximity, either to support protein complex formation on one cell, or to trigger contacts between cells. Examples of 'forced-connection' functionalities are bsAbs that support tumour-targeted immune cell recruiters and/or activators.

The advancement in bioengineering made the pharmaceutical industry to produce multiple type of BsAbs with a myriad of potential construction.

Exhibit 18 Schematic overview of the antibody structure and bsAb constructs currently being evaluated in clinical trials.



Source: Suurs et al, 2019

Currently, c. 60 bsAbs are in clinical trials in cancer patients of which c. 40 use the same mechanism of action: engagement of immune cells with tumour cells. Of the remaining c. 20 bsAbs in clinical trials, five deliver a payload to tumours and 14 are blocking signalling in the cancer environment.

A highly-regarded technology

Due to their potency, bsAbs have attracted a large interest from pharmaceutical players with numerous licensing deals overseen in the past couple of years. However, while BsAbs have attracted many investments, a lot of players are entering in that field creating a strong competitive environment. Hence, without a validation of the platform by a large player, we find it difficult to put numbers behind the value of BiCKI.

Exhibit 19 **Licensing deals in the bispecific landscape**

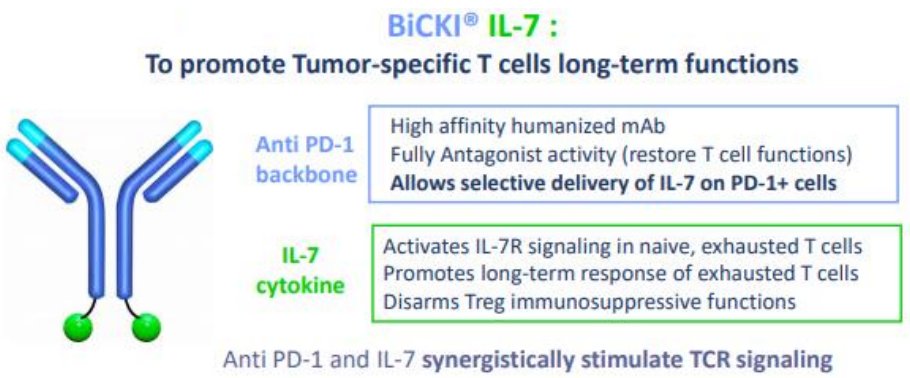
Deal Date	Company	Product	Pharmacological Class	Deal Partner/ Product Source	Status on Deal Date	Deal Value (USDm)
Jun-20	Abbvie	Epcoritamab, DuoHexaBody®-CD37 and DuoBody-CD3x5T4	CD3xCD20	Genmab	Phase I/II	3,900
Mar-19	Roche	XmAb24306	IL-15 & IL-15 receptor alpha (IL-15RA) bsAbs	Xencor	PC	120
Feb-19	AbbVie	TNB-383B	Anti-B cell maturation antigen (BCMA) & CD3 bsAbs	TeneoOne	PC	80
Dec-18	Gilead	AGEN1223 / GS-1423	bsAbs T cell engager (BiTE)	Agenus	PC	30
Nov-18	BeiGene	ZW25	Anti-human epidermal growth factor (HER2/ErbB-2) bsAbs	Zymeworks	Phase I	430
Aug-18	Roche	Affimed-Roche Research Program	Anti-CD30 & CD16A bsAbs	Affimed	Research	5,096
Aug-18	Harbour BioMed	ISB 1302	Anti-human epidermal growth factor (HER2/ErbB-2) & CD3 bsAbs	Glenmark Pharmaceuticals	PC	120
Jul-18	ABL Bio	ABL Bio-I-Mab bsAbs	Anti-cancer bsAbs	I-Mab Biopharma	Research	103
Jul-18	Genmab	Genmab-Immatics Cancer Research Project	Anti-cancer bsAbs	Immatics biotechnologies	Research	604
Jun-16	Novartis	XmAb14045	Anti-CD3 & IL-3 alpha/CD123 bsAbs	Xencor	PC	2,560
Sep-15	Amgen	AMG 424	Anti-CD3 & CD38 bsAbs	Xencor	PC	1,745
May-15	Genmab	BioNTech-Genmab IO Project	Anti-CD40 & CD137 bsAbs	BioNTech	Research	15
Jan-15	BMS	Celgene-Zymeworks bsAbs Program	bsAbs	Zymeworks	Research	164

Source: Degroof Petercam, Evaluate pharma

Preclinical data of BiCKI looks attractive

The first candidate that has been developed from BiCKI is BiCki-IL7 a PD-1/IL-7 BsAbs built with OSE proprietary anti-PD1, OSE-279. IL-7 is an optimal target for immunotherapy to preferentially stimulate effector T-cell (Teff) functions over regulatory T-cells (Treg) due to the differential expression of IL-7R and poor capacity of IL-7 to stimulate Treg proliferation. Moreover, it has been published that PD-1 blockades increase IL-7R expression and improve IL-7 signalling in exhausted T-cells. Hence, IL-7 could act as a booster of PD-1 functionality.

Exhibit 20 **OSE's BiCki first candidate: BiCki IL-7**



Source: Morello et al, 2020

No other PD-1/IL-7 bsAbs are developed to our best knowledge, hence, "BiCki Il-7" could become a fist-in-class candidate. We believe further candidates from the BiCki platform may emerge if "BiCki Il-7" proves to be successful. Several combinations are currently being studied by OSE:

- Cytokines;
- Costimulatory ligands;
- Costimulatory blockers;
- Trap-receptors.

We expect BiCki IL-7 to enter in clinical stage in 2021. While the track record of OSE make us confident in its capacity to develop and monetize this platform, we bear in mind that this program remains at an early stage evolving in a strong competitive environment.[12]

New targets are also underway: CLEC-1 and IL-7R

In earlier-stage, OSE is pursuing two new projects targeting the CLEC-1 receptor on myeloid cells considered as a potential new checkpoint inhibitor for macrophage (similarly to the SIRPα) and the receptor to the IL-7 (OSE-703).

The most exciting project in our view if the CLEC-1, given that the IL-7 mAb should be deprioritised due to the development of BiCki-IL-7.

CLEC-1 an orphan C-type lectin receptor has been designed by OSE as a potential novel myeloid checkpoint inhibitor and "DON'T EAT ME" signal with first results presented at the 2020 AACR virtual meeting. Using an antagonist to this receptor, OSE highlighted the potential role of its candidate in increase tumour cell phagocytosis by macrophages and through the activation of dendritic cells. This pathway was discovered at the INSERM and OSE in-licensed this discovery, thus we presumably guess that OSE is entitled to pay a low-end single digit royalty on net sales to the INSERM.

We view this new mechanism of action as promising if it shows similar success than the CD47/ SIRPα axis but difficult to assess given that OSE is the first company having developed a candidate targeting this pathway.

[12] Siwei Nie et al, Biology drives the discovery of bispecific antibodies as innovative therapeutics, Antibody Therapeutics, January 2020

Autoimmune diseases: novel targets present novel opportunities

Not exclusively focused on cancer therapies, OSE also used its know-how in developing drug candidates to modulate pathways that could be used in auto-immune (AI) diseases. The leading product of its AI portfolio is OSE-127, an anti-IL7R, developed in partnership with Servier. OSE-127 is about to enter in phase II in Sjögren's syndrome (SjS) and ulcerative colitis (UC).

The second candidate of this portfolio is FR104. It was once partnered to J&J, but the large pharmaceutical company decided to return the rights for strategic reasons. The product is currently "phase II ready" and OSE is seeking for a new partner. At this stage, we consider having a limited visibility on the future of this program.

OSE-127: seeking to differentiate by indication

Interleukin 7 (IL7) and its receptor (IL7R) are essential for normal T-cell development and function. However, they can also promote autoimmunity, chronic inflammation and cancer. As mentioned earlier, OSE develops anti-cancer agents (BiCki IL-7 and OSE-703) based on the T-cell promoting effects of the IL-7.

Conversely, this cytokine is known to play a key inflammatory effect in several auto-immune diseases. Thus, an increasing body of both preclinical and clinical evidence suggests that raised expression levels of interleukin (IL)-7 and/or the IL-7 receptor (IL-7R) are associated with various disease states, including primary Sjögren's syndrome[13,14] and colitis.[15].

Sjögren's syndrome is a large opportunity poorly addressed

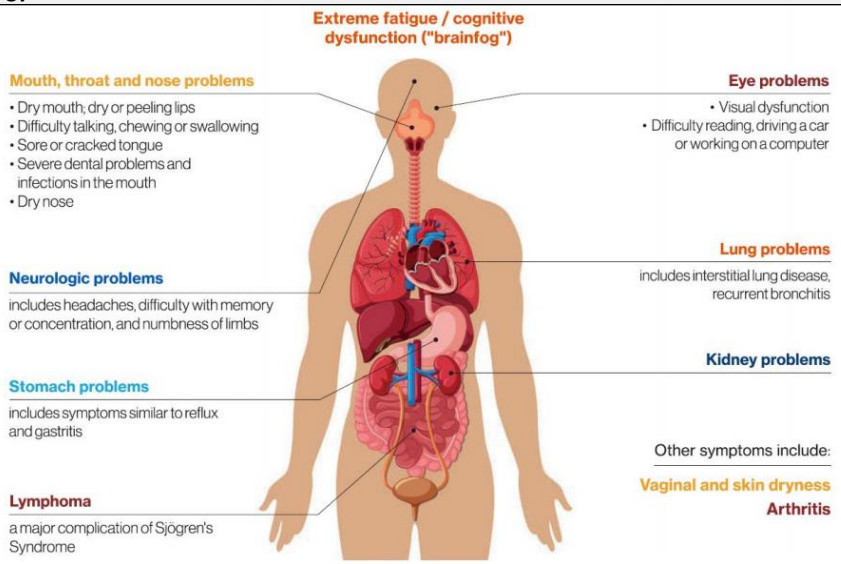
SjS is a chronic, systemic autoimmune disease characterized by lymphocytic infiltration of the exocrine glands such as the lacrimal and salivary glands. The disease frequently leads to keratoconjunctivitis sicca (dry eye) and xerostomia (dry mouth). SjS may occur with other autoimmune diseases, such as SLE or rheumatoid arthritis.

The severity of Sjögren's varies from person to person. Many patients have a mild disease that only affects the eyes and mouth. Others have symptoms that wax and wane in severity or may even go into remission. Severe cases include immune disorders (autoantibodies) and significant loss of function of exocrine saliva and/or lachrymal glands.

No treatments have been shown to alter the course of this disease. Supportive treatment is aimed at relieving dry mouth/dry eye symptoms.

[13] Bikker A, Kruize AA, Wenting M, Versnel MA, Bijlsma JW, Lafeber FP, et al. Increased interleukin (IL)-7Ralpha expression in salivary glands of patients with primary Sjogren's syndrome is restricted to T cells and correlates with IL-7 expression, lymphocyte numbers and activity. Ann Rheum Dis 2012; 71: 1027–33.
[14] Bikker A, Moret FM, Kruize AA, Bijlsma JW, Lafeber FP, van Roon JA. IL-7 drives Th1 and Th17 cytokine production in patients with primary SS despite an increase in CD4 T cells lacking the IL-7Ralpha. Rheumatology (Oxford) 2012; 51: 996–1005.
[15] Willis CR, Seamons A, Maxwell J, Treuting PM, Nelson L, Chen G, et al. Interleukin-7 receptor blockade suppresses adaptive and innate inflammatory responses in experimental colitis. J Inflamm 2012; 9: 39.

Exhibit 21 SjS's pathology



Source: Degroof Petercam

It is estimated that SjS affects 0.2-4% of the population worldwide.[16] In the US, approximately 2 to 4 million people have the disease, though only a million have been definitively diagnosed.[17]

When SjS appears in a previously healthy person, the disease is classified as primary, while patients with concomitant systemic autoimmune diseases are classified as associated (or secondary) SjS.

Clinical scores in SjS

To help patients and their physicians to assess the activity of the disease the European organization EULAR (European League Against Rheumatism) coordinated the creation of scientifically validated scores that are used during clinical trials.

ESSDAI score

The ESSDAI rolls up all of a patient's symptoms into one score reflecting the disease's influence on the body as a whole. If a compound slows the disease process, the score should drop as symptoms across the body abate.

Moderate-to-severe Sjögren's patients are defined with ESSDAI scores over 6. The medical community had agreed that a change of 3 points would be considered meaningful for Sjögren's patients. However, that bar had never been tested because no one had ever before used the ESSDAI score in a randomized clinical trial with positive results.

ESSPRI score

This is the patient-reported index score. The ESSPRI is calculated by averaging the scales for pain, fatigue and dryness.

A long-list of setbacks in SjS

[16] Pierce JL et al; "Swallowing Disorders in Sjögren's Syndrome: Prevalence, Risk Factors, and Effects on Quality of Life." Dysphagia; V.31; No.1; 02/16; p49.
[17] Kassan SS et al; "Clinical Manifestations and Early Diagnosis of Sjögren Syndrome." Archives of Internal Medicine; V.164; 6/28/04; p1275

Similarly, to the SLE, the SjS is a multifactorial disease which involves multiple immune-pathways. As such, a "one-size fit for all drugs" is challenging to develop and multiple setbacks have occurred so far.

Exhibit 22 SjS pipeline

Drug	Company	MOA	Status	Comments
B-cell activation				
Benlysta (belimumab)	GSK	BAFF	phase II	Benlysta+/-Rituxan vs placebo, results expected in 1H20 (NCT02631538)
Tibulizumab	Lilly	BAFF & IL-17A	phase I completed in 2018	Abandoned?
ianalumab/VAY736	Novartis	BAFF-R	Phase II	Expected completion: 2Q20 (NCT02962895)
RC18	Remegen	Fusion protein Blyss/APRIL	Phase II	Topline results expected in 2H20 (NCT04078386)
parsaclisib	Incyte	PI3Kδ inhibitor	Phase II	Completed in 2H19, awaiting results
tirabrutinib	Gilead	BTK inhibitor	Phase II	Large (n=152) study studying filgotinib +/- tirabrutinib (NCT03100942)
T-cell proliferation				
OSE-127	Servier / OSE	IL-7	Phase II	To start in 2020
GSK2618960	GSK	IL-7R	Phase II	Withdrawn (study was stopped for portfolio prioritization, NCT03239600)
Antigen presentation / co-stimulation				
Orencia (abaecept)	BMS	CTLA4	Phase III	Failed (NCT02915159)
lulizumab	BMS	CD28	Phase II	Abandoned - phase II results in 2017 (NCT02843659)
Prezalumab (AMG 557/MEDI587)	Amgen/AstraZeneca	anti-ICOS-L	Phase II	Abandoned - phase II results in 2018 (NCT02334306)
Iscalimab (CFZ533)	Novartis	anti-CD40	Phase II	Phase IIb read out expected in 2021 (NCT03905525)
VIB4920	VielaBio	CD40L	Phase IIb	Enrollment paused in phase IIb due to COVID-19
Proinflammatory cytokines				
Actemra (tocilizumab)	Roche	IL-6R	Phase II/III	Abandoned (NCT01782235)
Remicade (infliximab)	J&J	anti-TNFs	Phase III	Abandoned
Embrel	BMS	anti-TNFs	Phase III	Abandoned (NCT00001954)
Type 1 interferons				
RSLV-132	Resolve therapeutics	RNAse	Phase II	Positive results announced in June 2019 (NCT03247686)
MEDI7734/VIB7734	VielaBio / Astrazeneca	ILT7	Phase I	Development pursued in several autoimmune indications including Sjôgren's syndrome
filgotinib	Galapagos / Gilead	Jaks	phase II	Abandoned

Source: Degroof Petercam, company data

OSE-127 is about to enter in phase II with Servier in SjS

In March 2020, Servier and OSE signed an amendment on their collaboration. Recall that the deal included EUR 272m in development, regulatory and sales milestones. On this amount, EUR 10.25m were perceived as part of an upfront payment in early 2017, and an additional EUR 10m was paid as part of the first part of the two-step option in early-2019.

Under the amended agreement, EUR 5m will be perceived by OSE for the first-in patient in the SjS phase II study and the rest (EUR 15m) will be perceived once both the SjS and the UC studies will be completed, and in priority upon completion of the Phase 2a clinical study in Sjögren's syndrome.

Servier will be responsible for the SjS trial while OSE will conduct the UC trial. We believe this marks a strategic shift of OSE's partner towards the SjS due to the intensive competitive environment in UC due to the emergence of oral therapies (i.e. JAKs notably).

Ulcerative colitis competition is likely to be high

Market size and epidemiology

The global ulcerative colitis (UC) market was valued at USD 6.9bn in 2018 and is expected to grow with a CAGR of 4.9% from 2019 to 2024. Epidemiological studies estimate that the disease encompasses a diagnosed prevalence of around 735,600, 549,700 and 199,700 patients in US, EU5 and Japan, respectively, and is expected to increase annually by approximately 0.6%.

Pathology

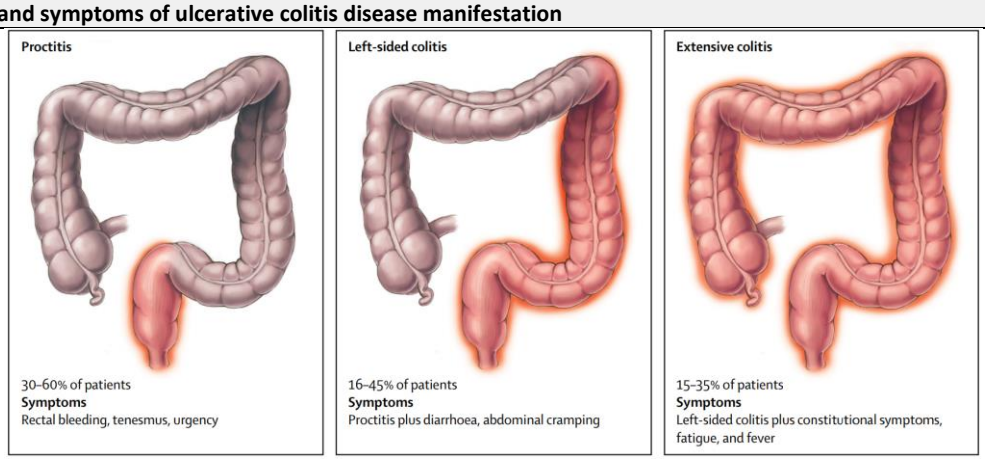
Ulcerative colitis (UC) is a chronic idiopathic inflammatory bowel disease (IBD) that causes inflammation of the inner-lining (mucosa) of the large intestine (colon) in a continuous fashion extending from the rectum to the more proximal colon. The disease is not manifested continuously, but rather via a relapsing and remitting course. The hallmark symptoms of UC include abdominal pain, bloody diarrhoea, faecal urgency. Disease onset typically occurs in young adults in their 20s and 30s. The aetiology of UC is unclear. Increasing evidence suggests the presence of an underlying autoimmune component. Many UC patients experience extraintestinal manifestations (EIMs) that involve multiple organs sharing features with other autoimmune disorders.

Current treatment patterns and unmet need

The goal of pharmacological treatment is achieving symptom control, endoscopic/mucosal healing, limiting/eliminating corticosteroid use and most importantly clinical remission. Pharmacological treatment methodology typically involves an induction therapy in order to achieve clinical remission, followed by a maintenance therapy (often at a lower drug dose) to sustain the clinical response and prevent future disease relapse.

- Patients with moderate-to-severe disease typically undergo initial treatment with conventional oral therapies including aminosalicylates (5-aminosalicylic acid), immunomodulators (azathioprine, 6-mercaptopurine, methotrexate, cyclosporine A) and corticosteroids.

Exhibit 23 **Sites and symptoms of ulcerative colitis disease manifestation**



Source: Ungaro, Ryan et al. "Ulcerative colitis." The Lancet 389.10080 (2017): 1756-1770.

- However, due to unacceptable side-effects and/or inadequate response, more than half of these patients require alternative treatments. Commonly used drugs herein are injectable biologics including anti-Tumour Necrosis Factor (TNF) antibodies (Humira, Remicade, Simponi) and anti-adhesion antibodies (Entyvio). These therapies are limited by several factors including side-effects, increased risk of infection or cancer and loss of clinical benefit over time. KOLs indicated that approximately 30%

of patients per year need to abort anti-TNF treatment due to side-effects and/or inadequate response, and about 50% of patients stop responding within 12 months. The need for biologics to be administered via injection (intravenous or subcutaneous) represents an additional limiting factor, as the invasive nature of the latter impedes patient comfort and often requires hospital visits.

Several biologics and oral small molecules are in late-stage development. A few offer a novel mechanism-of-action, while the majority represents 'me-too' drugs. So far, Xeljanz, a Janus kinase inhibitor (JAKi) is the only oral treatment alternative approved for UC. However, Xeljanz carries a black box warning on cardiovascular and infection risk, which is believed to be dose-dependent. Two additional JAKi's, Galapagos' filgotinib and AbbVie's Rinvoq, are expected to have Phase III readout in 2020 and 2021, respectively. Two oral sphingosine-1-phosphate (S1P) modulators, ozanimod and etrasimod, are expected to have Phase III readout in 2020 and 2021, respectively.

- Approximately 15% of patients ultimately require surgery (colectomy), a rate which has not declined over the past 10 years. KOLs highlighted that this procedure also holds several risks, including post-surgical inflammation risk.

These elements demonstrate that novel oral disease-modifying drugs present a substantial opportunity to improve the clinical outcome and quality-of-life of UC patients.

Drug efficacy measures

The most commonly used clinical measures for assessing UC disease activity, and for evaluating the efficacy of novel treatments is the Total Mayo Score (TMS) and the Ulcerative Colitis Disease Activity Index (UCDAI). Both tools incorporate scorings on stool frequency (reported by patient), rectal bleeding (reported by patient), mucosal findings (via endoscopy) and the physician's assessment of disease activity. Each parameter can register a subscore from 0 up to 3, a higher score implying a more severe grade. The maximum score of both measures is 12. Moderate-to-severe UC can be defined by a TMS and/or UCDAI of 6 or higher.

The added value of the physician's assessment of disease activity to TMS has recently been put into question by KOLs and regulators. New recommendations to exclude this parameter have been brought forward, leading to an adapted clinical measure defined as the modified Mayo Score (MMS). Moderate-to-severe UC is defined by a MMS of 4 or higher.

- Clinical remission, the primary efficacy endpoint recommended by the regulators, is defined by a stool frequency subscore of 0, a rectal bleeding subscore of 0 and an endoscopy subscore of 1 or 0 (mucosal healing). Clinical remission would imply a TMS or UCDAI equal or lower than 2, with no subscore higher than 1.
- Clinical response is typically defined by a TMS or UCDAI reduction of minimal 3 points + 30% decrease from baseline score + a decrease of the rectal bleeding subscore of minimal 1 point OR an absolute score for rectal bleeding not exceeding 1.

Exhibit 24 Total Mayo Score (TMS)

Stool frequency*
0 = Normal no. of stools for this patient
1 = 1–2 stools more than normal
2 = 3–4 stools more than normal
3 = 5 or more stools more than normal
Rectal bleeding†
0 = No blood seen
1 = Streaks of blood with stool less than half the time
2 = Obvious blood with stool most of the time
3 = Blood alone passed
Findings of flexible proctosigmoidoscopy
0 = Normal or inactive disease
1 = Mild disease (erythema, decreased vascular pattern, mild friability)
2 = Moderate disease (marked erythema, absent vascular pattern, friability, erosions)
3 = Severe disease (spontaneous bleeding, ulceration)
Physician's global assessment‡
0 = Normal
1 = Mild disease
2 = Moderate disease
3 = Severe disease

*Each patient served as his or her own control to establish the degree of abnormality of the stool frequency.

†The daily bleeding score represented the most severe bleeding of the day.

‡The physician's global assessment acknowledged the three other criteria, the patient's daily record of abdominal discomfort and general sense of well-being, and other observations, such as physical findings and the patient's performance status.

Source: fda.gov

Exhibit 25 Ulcerative Colitis Disease Activity Index (UCDAI)

1. Stool frequency
   0 = Normal
   1 = 1–2 Stools/day >normal
   2 = 3–4 Stools/day >normal
   3 = >4 Stools/day >normal
2. Rectal bleeding
   0 = None
   1 = Streaks of blood
   2 = Obvious blood
   3 = Mostly blood
3. Mucosal appearance
   0 = Normal
   1 = Mild friability
   2 = Moderate friability
   3 = Exudation, spontaneous bleeding
4. Physician's rating of disease activity
   0 = Normal
   1 = Mild
   2 = Moderate
   3 = Severe

Maximum score = 12

Source: fda.gov

- Regarding biomarkers, faecal calprotectin has proven to be a reliable biomarker to monitor disease activity, with levels above 50 µg/g being correlated with endoscopic disease activity in both UC.

Comparative clinical performance analysis

In order to establish an approximated benchmark, we outlined the efficacy and safety profiles of existing top-selling drugs and late-stage drug candidates for treatment of moderate-to-severe UC, based on the aforementioned drug measures. Note that statistical significance compared to placebo was not always observed for the reported response/remission values. In addition, caution should be taken in conducting cross-trial efficacy comparisons as patient characteristics are most probably not identical in between different trials. The safety/tolerability component is equally important in novel drug evaluation. This especially holds true for anti-inflammatory drugs, as many of currently approved agents hold several safety/tolerability concerns (e.g. anti-TNFs).

Exhibit 26 Comparative clinical efficacy analysis in UC

Class	Drug	Intake	Brand	Company	Status	Readout	Total product sales[1]	Week 6-12 response[2] rate % (placebo-adjusted)	Week 6-12 remission[2] rate % (placebo-adjusted)	Safety concerns
Anti-TNF	Infliximab	Injection	Remicade	JnJ	Approved	NA	USD 5.3bn	33	24	Infection/cancer
	Adalimumab	Injection	Humira	AbbVie	Approved	NA	USD 19.9bn	10	10	Infection/cancer
	Golilumab	Injection	Simponi	JnJ	Approved	NA	USD 2.1bn	21	12	Infection/cancer
Anti-adhesion molecules	Vedolizumab	Injection	Entyvio	Takeda	Approved	NA	USD 1.8bn	21	12	Infection/PML[3]
	Etrolizumab	Injection	NA	Roche	Phase III	2020 (failed)	NA	4	21	NA
	SHP647	Injection	NA	Takeda	Phase III	2020	NA	25	14	NA
	AJM300	Oral	NA	Eisai	Phase III	2020	NA	37	21	NA
JAK inhibitors	Tofacitinib	Oral	Xeljanz	Pfizer	Approved	NA	USD 1.8bn	27	11	Infection/thrombosis
	Filgotinib[5]	Oral	NA	Galapagos	Phase III	2020	NA	NA	11	NA
	Upadacitinib	Oral	Rinvoq	AbbVie	Phase III	2021	NA	32	14	Infection/thrombosis
	TD-1473	Oral	NA	TheraVance	PhaseII/III	2025	NA	NA	NA	NA
Anti-interleukins	Ustekinumab	Injection	Stelara	JnJ	Approved	NA	USD 3.5bn	31	11	Infection/cancer
	Risankizumab	Injection	Skyrizi	AbbVie	Phase III	2020	NA	NA	NA	Infection
	Mirikizumab	Injection	NA	Lilly	Phase III	2020	NA	39	18	NA
	BI 655130	Injection	NA	BI	Phase III	2022	NA	NA	NA	NA
Sphingosine 1-phosphate (S1P) modulators	Ozanimod	Oral	NA	Celgene/BMS	Phase III	2020	NA	18	9	Cardiac
	Etrasimod	Oral	NA	Arena	Phase III	2021	NA	11	8	Cardiac
PDE4 inhibition	Aprelimast	Oral	Otezla	Celgene/BMS[4]	NA[4]	NA[4]	USD 1.6bn	20	18	Depression/weight loss

[1]Inclusion of other indications

[2]Based on Mayo Score

[3]Progressive Multifocal Leukoencephalopathy

[4]BMS plans to divest Otezla as part of the Celgene acquisition

[5]200 mg dose (failed for the 100mg)

Data extracted from company websites, product labels and academic publications

Source: Data extracted from company websites, product labels and academic publications

OSE-127 positioning

As seen, the competitive environment in UC is quite intense and many oral, more convenient candidates are under development. We believe OSE-127 will face significant competition by the time it will be launched.

Moreover, the IL-7 target remains to be validated in a randomised clinical trial. As such, we remain cautious on the potential outlook of OSE-127 in UC.

FR104 is on hold searching for a partner

FR104 is an anti-CD28 which was once partnered to J&J. In November 2018, OSE regained the global rights of this asset as J&J dropped the collaboration for strategic considerations. Since then, the candidate is a phase II ready candidate for the treatment of rheumatoid arthritis as well as for its use in transplantation procedure.

We believe the candidate is no longer a priority for the group and that without a partner, OSE won't take the bet to move forward FR104 by itself. We believe this decision is being made both for cash consideration (prioritisation of early-stage program) and change in therapeutic landscape (intense competitive environment in rheumatoid arthritis).

Sales forecast

OSE is building a dual business model that relies on both license revenues (OSE-127 and BI 765063) and internal developments (Tedopi). Note that we do not include other assets in our top-line model, as they are still at an early stage of development (BiCki and CoVepiT) or deprioritized (FR104).

Thus, we model licence revenues coming from the development and commercialisation of OSE-127 in SjS and UC by Servier. We estimate peak sales of EUR1.2bn for OSE-127 in the aforementioned indications, and estimate that OSE could receive cumulative revenues of c. EUR2.0bn over 2020-40E, with a EUR15m milestone payment due in 2022E (i.e. opt-in option from Servier at the end of the on-going phase II).

Together with BI, OSE is developing a potential best-in-class asset, which began in clinics in 2019. BI will progressively take the lead in the management of the clinical development, taking over full responsibility at the end of the phase I. While the sales potential of this product may be hugged given many potential cancer indications, we solely value the clinical milestones given the lack of visibility of potential targeted indications.

Regarding Tedopi, we value both the pancreatic and NSCLC indication through an out-licencing partnership that may emerge in 2021.

Although we have used a cautious set of assumptions, we would highlight that our forecasts carry a degree of uncertainty stemming from the lack of visibility on pricing, as well as the tough and burgeoning competitive environment in the field of immune-oncology.

With a cash position estimated at c. EUR21m at mid-2019, OSE is fully funded until mid-21. This timeline includes the c. EUR 7m loan agreement guaranteed by the French state.

Credibility gained among big pharma

Strategic deal with BI

In April 2018, OSE started collaborating with BI for the development and commercialisation of BI 765063. Under the terms of the deal, OSE received an upfront payment of EUR 15m. In 2019, following the start of the first clinical study, BI made a second payment to OSE of EUR 15m. OSE will be due up to EUR 1bn in milestone payments. The arrangement also includes royalties on sales.

Partnership on OSE-127 with Servier

In December 2016, OSE has inked a licensing agreement with Servier covering the development and commercialisation of OSE-127. As part of this deal, OSE is eligible for development and commercial milestone payments of up to EUR 272m and double-digit royalties on net sales

It included an upfront payment of EUR 10.25 million and additional payments of EUR 30m upon the exercise of a two-steps option license. The EUR 10m first step option fees were already received in February 2019.

This agreement was amended in March 2020, making OSE eligible to receive a EUR5m milestone payment from Servier upon the enrolment of the first patient in the Phase 2a clinical study in SjS and an additional EUR15m payment upon exercise option completion of both Phase 2 clinical trials, and in priority upon completion of the Phase 2a clinical study in SjS.

Tedopi: NSCLC phase III confirmatory expected with a partner

ATALANTE-1 detailed results will be presented at the ESMO in September. We believe that the small population studied and the closing of the study at the interim analysis due to the COVID-19 pandemic will be insufficient for a conditional filing. Thus, we take the hypothesis that OSE will start to search for a partner by YE-20 in order to launch a confirmatory large phase III study.

We estimate that this trial will start in 1H21. We therefore expect the partner to launch Tedopi for the treatment of checkpoint-inhibitor, refractory, HLA-A2 positive, metastatic NSCLC, in the EU/US in 2025 and in Japan/China in 2026.

We have not counted any sales in peripheral European countries, or in other industrialised countries like Australia, Israel, Russia, South Africa, etc., which could offer upside. We assume commercial exclusivity of ten years from the start of commercialisation.

A conservative peak sales of EUR 0.6bn in NSCLC

Key assumptions

We use the following set of assumptions for our top-line model of Tedopi in NSCLC:

- Peak penetration of 30% in EU5/US and 25% in Japan/China which is realistic given given the unmet medical need identified in those settings. We believe that the top line could experience a quick ramp-up (around five years).
- Price of EUR 60,000 per treatment in the US, well below the list price of CPIs, which stands at c. USD 140,000 (i.e. c. EUR 130,000)[18]. In Europe, prices for recently launched oncology medicines are c. 50% lower than US prices. Note that we are being conservative as the cost of more recent drugs is slightly higher.
- A patent expiry estimated in 2035.

Addressable market

It is estimated that c. 470,000 patients are diagnosed with 1L metastatic NSCLC each year in the US/EU-5/Japan/China combined. Around 160,000 of them do not driver mutations (i.e: EGFR, ALK etc). Of these patients around 30%-60% of patients receive a CPIs as a backbone therapy. Of these patients, we assume that c. 85% will progress, 66% of them will receive a 2L and 48% of these patients are HLA-A2 positive.

Exhibit 27 **Tedopi targeted population in NSCLC**

Market waterfall	EU5	US	Japan	China	Total
Population (in millions)	322.2	330.7	*126.2*	*1386.4*	
1L metastatic NSCLC population drug treated (in thousands)	**126**	**107**	**54**	**184**	**471**
1L metastatic w/o driver mutations (sq and non-sq) - (in thousands)	98	88	*35*	*138*	*359*
1L metastatic w/o driver mutations (sq and non-sq) - CKI treated	**59**	**44**	**18**	**41**	**162**
% coverage	*60%*	*50%*	*50%*	*30%*	*45%*
% of patients progressing post CKI	**50**	**37**	**15**	**35**	**137**
% of CKI-refractory patients	*85%*	*85%*	*85%*	*85%*	*85%*
2L drug-treated	**31**	**27**	**11**	**21**	**90**
% of drug-treated	*63%*	*72%*	*73%*	*60%*	*66%*
HLA-A2 patients	**15**	**13**	**5**	**10**	**43**
% HLA-A2 patients	*48%*	*48%*	*48%*	*48%*	*48%*
Drug treated (000)	**4.5**	**4.1**	**1.8**	**2.5**	**12.9**
% patients	*30%*	*32%*	*34%*	*25%*	*30%*

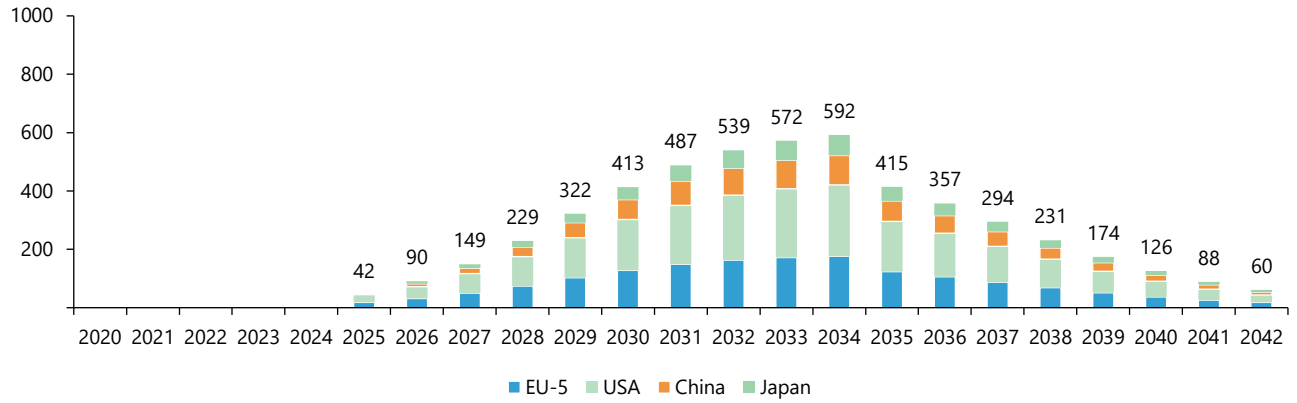
Source: Degroof Petercam

[18] Keytruda: drug information. UpToDate. www.uptodate.com. Published 2020. Accessed January 3, 2020.

Sales forecast

Based on this population, we estimate that Tedopi could generate peak sales of EUR 600m in the treatment of refractory CPIs-treated NSCLC patients.

Exhibit 28 **Tedopi sales in NSCLC (EURm)**



Source: Degroof Petercam

Metastatic pancreatic cancer: peak sales of EUR 120m

Key assumptions

We use the following set of assumptions for our top-line model of Tedopi in metastatic pancreatic cancer:

- Peak penetration of 30% in EU5/US which is realistic given the unmet medical need identified in those settings. We believe that the top line could experience a quick ramp-up (around five years).
- Price of EUR 60,000 per treatment in the US, well above the list price of Onivyde (USD c.45,000 USD before rebate) but below the list price of CPIs, which stands at c. USD140,000 (i.e. c. EUR 130,000).

Addressable market

There were around 51,000 new cases of CRC in the US in 2018 and c. 66,000 in the EU-5, and approximately 70% are at a metastatic or regionally advanced stage and non resectable. Around 75% of these patients receive a 1L treatment on which 30% are under a Folforinox treatment regimen and would be eligible, if HLA-A2 positive, to receive a maintenance treatment with Tedopi.

Exhibit 29 **Tedopi targeted population in PDAC**

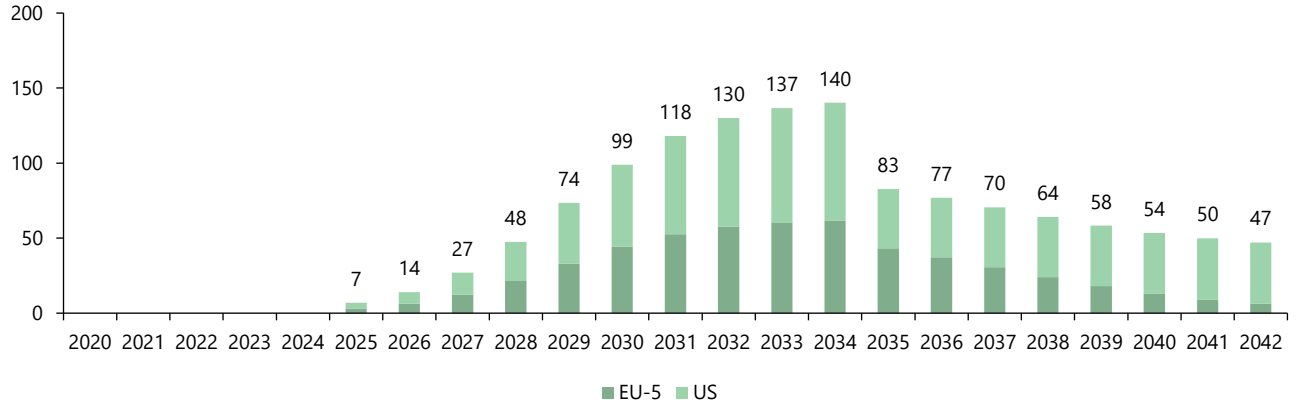
Market waterfall	EU5	US	Total
Population (in millions)	322.2	330.7	
Pancreatic cancer incidence (in thousands)	**66**	**51**	**117**
New ly diagnosed metastatic or regionnaly advanced	48	37	*85*
1L drug treated	**36**	**28**	**64**
% treated	*76%*	*76%*	*76%*
Treated with Folfirinox	**11**	**8**	**19**
% treated with Folforinox	*30%*	*30%*	*30%*
HLA-A2 patients	**5**	**4**	**9**
% HLA-A2 patients	*48%*	*48%*	*48%*
Drug treated (000)	**1.5**	**1.3**	**2.8**
% patients	*30%*	*32%*	*31%*

Source: Degroof Petercam

Sales forecast

Based on this population, we estimate that Tedopi could generate peak sales of EUR 150m in the maintenance of PDAC patients.

Exhibit 30 Tedopi sales in PDAC (EURm)

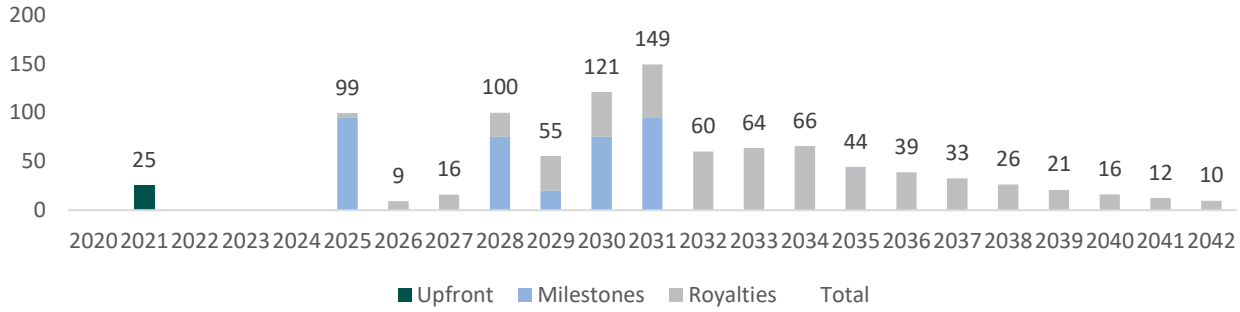


Source: Degroof Petercam

Revenues for Tedopi as per partnership opportunity and full asset value

We take the hypothesis of a global partnership signed by OSE in 2021. We factor into our model: 1) EUR 360m for development, regulatory, and commercial milestones; and 2) cumulative royalties totalling over EUR 950m over 2020-40E based on a royalty rate of 13% applied on global sales.

Exhibit 31 OSE revenues with Tedopi (EURm)



Source: Degroof Petercam

OSE-127: SjS becomes the main opportunity

Due to the intense competitive environment in UC, OSE and its partner Servier operated a shift in strategy on OSE-127 towards the pSjS. Meanwhile, OSE is responsible for the clinical development of OSE-127 until the end of the phase II which should started soon. Thus, we expect the opt-in option of Servier (EUR 15m) to be exercised in 2022.

SjS: peak sales of EUR 900m

Addressable market and top line assumptions

The incidence of pSjS is evaluated at 0.06% patients per year in the US and EU- 5 together[19]. The systemic pSjS accounts for 71% of this population[20]. We assume the product will grab a

[19] Maldini et al., 2014

[20] Ramos-Casals M, Solans R, Rosas J, Camps MT, Gil A, Del Pino-Montes J. et al. Primary Sjogren syndrome in Spain: clinical and immunologic expression in 1010 patients. Medicine. 2008;87:210-9

market share of 40% since it would be the only approved treatment in this setting. We set the price of this therapy at USD 35,000, i.e. the list price of GSK's Benlysta in the US, due to the similarities between SjS and SLE (systemic lupus erythematosus). Hence, we factor into our model a price of EUR 30,000 for the US and EUR 20,000 in the EU-5. Finally, based on the recent patent grant of the USPTO, we estimate a patent expiry in 2035.

Exhibit 32 OSE-127 targeted population in SjS

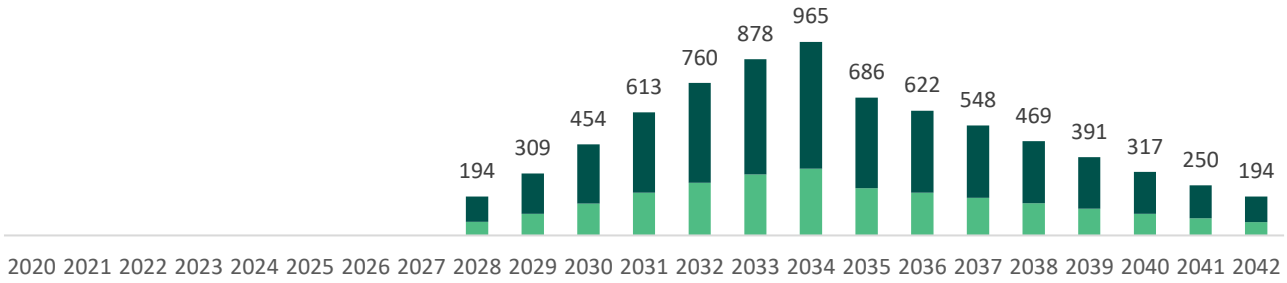
Market waterfall	EU5	US	Total
	322.2	330.7	
pSjS diagnosed prevalence	**194**	**200**	**394**
Patients with active systemic disease	137	142	*279*
Patients treated with biologicals	**41**	**50**	**91**
% treated	*30%*	*35%*	*33%*
OSE-127 treated (000)	**18.5**	**23.4**	**41.9**

Source: Degroof Petercam

Sales forecast

Based on this population, we estimate that OSE-127 could generate peak sales of EUR 900m in systemic pSjS for a launch in 2028.

Exhibit 33 OSE-127 sales in SjS (EURm)



Source: Degroof Petercam

UC: an increasingly limited opportunity due to oral emerging treatments

Given the increasing competitive environment in UC, we believe that the market opportunity for new entrant is becoming tiny unless it shows meaningful clinical effectiveness with an attractive safety profile. We are therefore cautious regarding the commercial potential of OSE-127 especially without any clinical data yet.

Addressable market and top line assumptions

The UC market is large and estimated to reach USD 9bn in 2026 from USD 6bn in 2020 mainly driven by the emergence of JAKs (Abbvie's Rinvoq, Pfizer's Xejianz) and S1P1 (Arena's etrasimod among others).

We believe OSE-127 will have to demonstrate a strong efficacy profile to overwhelm its inconvenient injectable profile. We assume this candidate will grab a market share of 8% in the refractory moderate-to-severe UC patient population.

Exhibit 34 OSE-127 targeted population in UC

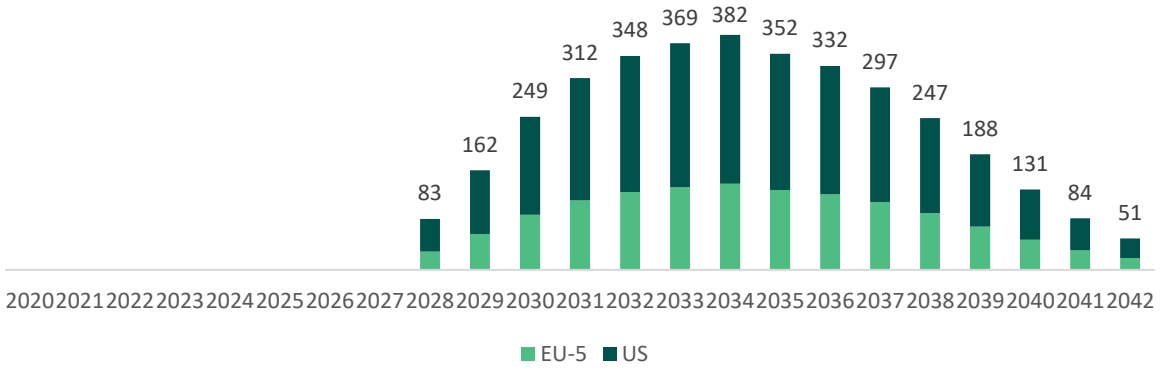
Market waterfall	EU5	US	Total	Source
Population (in millions)	322.2	330.7		
Diagnosed prevalence	**560**	**700**		Decision Ressources Group, Deg
Moderate-to-severe UC	308	399	*707*	
Refractory to 5-ASA	**123**	**160**	**283**	Company data
% refractory	*40%*	*40%*	*40%*	
Patients treated with 2L (biologicals and oral)	**64**	**81**	**144**	Company data
% treated with 2L	*52%*	*51%*	*51%*	
Drug treated (000) at peak	**6.2**	**7.9**	**14.1**	**Degroof Petercam**
% patients	*10%*	*10%*	*10%*	

Source: Degroof Petercam

Sales forecast

Based on this population, we estimate that OSE-127 could generate peak sales of c. EUR 400m in systemic UC.

Exhibit 35 OSE-127 sales in UC (EURm)

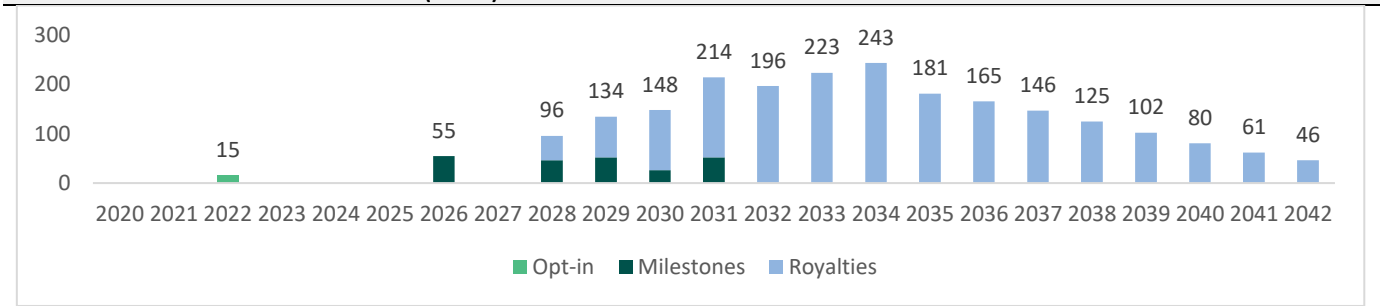


Source: Degroof Petercam

Revenues for OSE-127 as per Servier partnership and full asset value

We factor into our model: 1) EUR 15m opt-in option payment in 2022 at the end of phase II clinical trials; 2) EUR 231m for the remaining development, regulatory, and commercial milestones; and 3) cumulative royalties totalling over EUR 950m over 2020-40E based on a royalty rate of 13% applied on global sales.

Exhibit 36 OSE revenues with OSE-127 (EURm)



Source: Degroof Petercam

OSE-172/ BI 765063: revenues stream will continue

While difficult to assess, we take the hypothesis from Bodan and Villiger to evaluate the revenue stream emerging from the deal with BI.

Exhibit 37 Down-payments weight as per stage of development

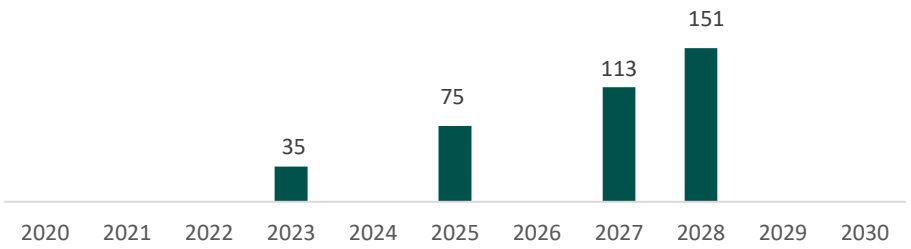
	Preclinical	Phase I	Phase II	Phase III	NDA	Market
Preclinical to market weight of payments	2%	3%	7%	10%	14%	64%
PI to market weight of payments		3%	7%	11%	14%	65%
PII to market weight of payments			7%	11%	15%	67%
PIII to market weight of payments				12%	16%	73%
NDA to market weight of payments					18%	82%

Source: Valuation in life science, Boris Bodan and Ralph Villiger

We factor into our model: 1) 3% of down-payments received at the end of the phase I in 2023; 2) 7% at the end of the phase II in 2025; 3) 15% at the end of a phase III program in 2027; and 4) 15% for the regulatory approval in 2028.

We do not take into consideration any commercial milestone (representing 64% of the potential down payments) for the moment given the lack of visibility of potential future indications. Thus, we consider that there is plenty of room for further upsides once first results will be released.

Exhibit 38 Revenues stream from BI agreement (EUR m) – not risk-adjusted

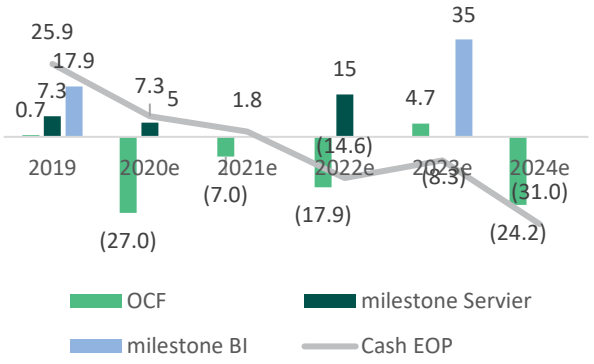


Source: Valuation in life science, Boris Bodan and Ralph Villiger, Degroof Petercam

Fully funded until 3Q-2021 (cash position at mid-20E: c. EUR 21m)

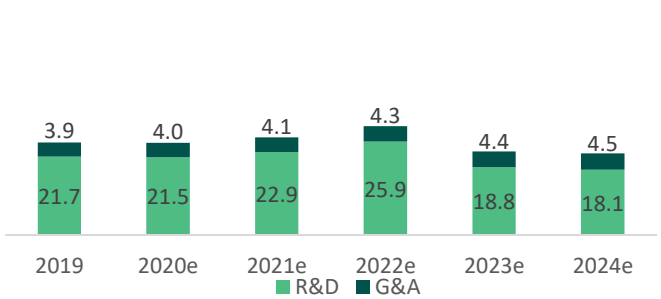
Based on OSE's solid end-19 cash situation (EUR 25.8m) and the proceeds from the loan guaranteed by the French state (c. EUR 7m), as well as the projected trends in G&A costs and R&D expenses (expected to decrease due to slowing down of clinical trial activities), the company is fully funded until 3Q21. This exclude a potential EUR 25m that we estimate the company may receive if a deal is inked on Tedopi in 2021.

Exhibit 39 Cash forecast (EUR m)



Source: Degroof Petercam estimates

Exhibit 40 R&D and SG&A forecasts (EUR m)



Source: Degroof Petercam estimates

Valuation and target price

We derive our valuation of EUR 173m (EUR 11 per share), factoring in OSE-127, Tedopi and BI 765063. We consider all other assets as free options for now, as they are in the early stages of development (BiCki) or on hold of a partner (FR104).

The next twelve months are expected to be a full one for OSE with: 1) the publication of the complete data of Tedopi in NSCLC expected at the ESMO (18th September); 2) the launch of the phase II of OSE-127 in SjS and UC; and 3) the first results of BI 765063 expected in 1H21. Another key catalyst for the shares should be the signature of a partnership on Tedopi that we expect in 2021.

Key valuation assumptions

Methodology and scope

Our valuation of OSE is based on an SOP including Tedopi in CPI-refractory NSCLC and PDAC, OSE-127 in SjS, and BI 765063 (based on clinical milestones). Each asset is valued using an rNPV model built over an explicit timeframe, which runs from 2020E to 2040E. We exclude all other early-stage developments and FR104, thus potentially offering substantial upside.

Cost assumptions

Our valuation model uses the following cost assumptions:

Exhibit 41 Cost assumptions

Assumptions	Tedopi						OSE-127				BI
	NSCLC				PDAC		UC		SjS		Solid tumours
	EU-5	US	Japan	China	EU-5	US	EU-5	US	EU-5	US	n.a.
COGS (as % of sales)	13%	8%	13%	17%	13%	8%	15%	10%	15%	10%	n.a.
SG&A (% of sales)	20%	20%	20%	20%	20%	20%	25%	25%	20%	20%	n.a.
Launch costs (EURm)	160				80		80		80		n.a.
Payment to third parties	6% of royalties on global sales paid to Takeda						none				none
R&D (EURm)	OSE contribution: none Partner contribution: all R&D costs (EUR 50m Phase III then EUR 5m p.a.)				OSE contribution: phase II (EUR 3m) Partner contribution: all R&D costs post phase II (EUR 50m Phase III then EUR		OSE: phase II (EUR9.3m) Servier: phase III (EUR 55m, then EUR 5m p.a.)		OSE: phase IIa (EUR3m) Servier: phase IIb (EUR 7.5m); phase III (EUR 22.5m)		n.a.

Source: Degroof Petercam

As part of the collaboration with BI, we assume that OSE supports no R&D costs. All the costs related to the production or the commercialization of the candidates are supported by BI, even though we believe that OSE may participate to the commercialization effort in Europe at some point of time.

Tax rate

We assume a tax rate of: 1) 25.0% for the profit that will be generated through the direct marketing of products; and 2) 15.0%, based on the French tax policy, for licensing-related revenues including upfront and milestone payments, and royalties.

WACC

We use two different WACCs: 1) 15% for the candidates when they are developed by OSE and 2) 8.8% (the average WACC in the European pharma industry) when the product is developed and commercialised by a partner. This modelling approach aims to take into account the specific risks inherent to the company that develops/markets the product.

Probability of success

Our model also takes into consideration the likelihood of occurrence of each cash flow based on the probability of success (POS) of clinical studies. The likelihood of approval (LOA), taking into account the current stage of development is: 1) 64% for Tedopi in NSCLC and 16% in PDAC; 2) 17% for OSE-127 in UC and 11% in SjS; and 3) 4% for BI 765063 in solid tumours.

Exhibit 42 POS in drug development

Succes rates	Autoimmune diseases		Solid tumours	
	PoS* (%)	LoA** (%)	PoS* (%)	LoA** (%)
Ph. I --> Ph. II	65%	11%	64%	4%
Ph. II --> Ph. III	32%	17%	23%	6%
Ph. III --> Reg.	62%	53%	34%	27%
Reg. --> MA	86%	86%	80%	80%

Source: BIO *Probability of success; **Likelihood of approval

Number of shares

Our TP is calculated on the basis of c. 15.7m shares (fully diluted), including 15.0m outstanding shares and 0.7m potential new shares stemming from the exercise of warrants.

Target price: EUR 11

Our valuation comes out at EUR 166m, corresponding to a EUR11 TP. Under our blue-sky scenario, which ascribes a 100% probability of success to the different phases of development, the company would be worth about EUR 1bn.

Exhibit 43 SOP

Product	Commercial Strategy	Peak Sales EURm	NPV EURm	Discount	rNPV EURm	rNPV / Share EURm
Tedopi / NSCLC refractory	Out-licensing	592	216	69%	67	4.29
Tedaopi / PDAC	Out-licensing	140	48	93%	3	0.20
OSE-127 / UC	Out-licensing	382	120	83%	20	1.28
OSE-127 / pSjS	Out-licensing	965	391	89%	44	2.82
OSE-172	Out-licensing	na	170	72%	48	3.07
Non-allocated costs	ns	ns	-27	0%	-27	-1.71
EV			917	83%	157	10
Net debt at mid-2020			-16		-16	-1.02
Equity			933	81%	173	11.0

Source: Degroof Petercam estimates

Upcoming catalysts

While 2020 is a year of trial launch for OSE-127, detailed results of Tedopi in NSCLC will be disclosed at the ESMO in September. It is the main catalyst to come. Other important event trigger will be the release of first results on BI 765063 expected in 1H20.

Exhibit 44 News flow

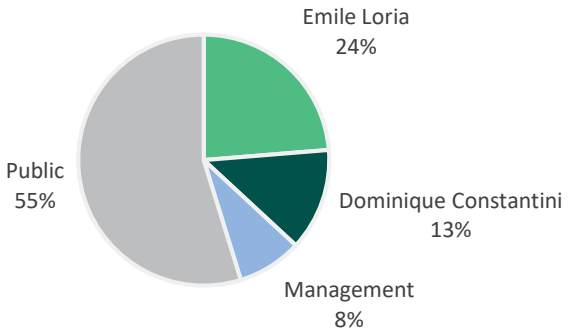
Date	Product	Event	Level of importance
18th September	Tedopi	Presentation of positive Step-1 results of PIII in NSCLC at ESMO congress	High
2H20	CoVepiT	First preclinical results of COVID-19 vaccine program If positive, potential entry into clinical phase end 2020	Medium
Q4 20	OSE-127	Initiation of two Phase 2 in UC (OSE sponsor) and Sjögren's syndrome (Servier sponsor)	Low
1H21	BI 765063/OSE- 172	First Results of PI in solid tumors	High
4Q21	Tedopi	Results of PII in pancreatic cancer	High

Source: Degroof Petercam estimates

Appendix: Shareholders and key people

Shareholding structure

Exhibit 45 Key shareholders



Source: Degroof Petercam

Management team

Dominique Costantini, MD, Chairman and director of development

Dr Costantini held a number of management positions at HMR (now Sanofi) where she led medico-marketing activities to commercialise products (notably in immunology, endocrinology, infectious illnesses and oncology). While there, Ms. Costantini also participated in the development of various medicines, from conception to the product approval and commercialisation stages. In 1997, Costantini founded BioAlliance Pharma, where she held the position of Chief Executive Officer until 2011. During her tenure there she led BioAlliance Pharma's IPO on Euronext (2005). BioAlliance Pharma originated Livatag, an anticancer nanotechnology in primary liver cancer, currently in phase-III development (in Europe and the US). In the past, Costantini was instrumental in forming many international industrial partnerships (Europe, the US, China, Japan and South Korea). To date, BioAlliance Pharma (renamed Onxeo in 2014) is the only French biotechnology company to have two FDA drug approvals. In 2012, Costantini co-founded and led OSE Pharma as CEO from 2012 to 2018.

Emile Loria, founder

Dr Emile Loria has more than 25 years of experience in the pharmaceutical and biotechnology sectors, having held key positions notably at Ciba-Geigy and Sanofi Pharma. He was a researcher at the Cancer Institute in Villejuif and then at the Cancer and Immunogenetics Institute (INSERM-CNRS) founded by Professor Georges Mathé. In 1986, he founded MS Medical Synergy, a drug management company. He then joined Biovector Therapeutics where he became Chairman and Chief Executive Officer.

In 2001, Loria was appointed Chief Executive Officer of Epimmune and developed the epitopes technology, from which stemmed the Tedopi epitopes. He ran this programme from the initial research stage to the start of the phase-II clinical trial. In 2012, he purchased, via OPI (Geneva), all of the Memopi assets and co-founded OSE Pharma.

Alexis Peyroles, CEO

Alexis Peyroles has more than 20 years of international management and financial control experience, having served in multiple related positions. He joined Sanofi-Aventis in 1996 as Financial Controller in Japan before becoming Head of Financial Control for the Baltic States. He was subsequently named Head of activities for Business Development in Eastern Europe.

In 2005, Alexis joined the Guerbet group (a leader in the field of contrast products, especially in medical imaging) as Financial Control Manager and in 2009 became Chief Executive Officer for Latin America, based in Brazil.

Since 2013, Alexis Peyroles has been involved in OSE Pharma, both as Chief Financial Officer and in charge of Business Development. From May 2016 (the date of the merger of OSE Pharma with Effimune) to April 2018, he served as Chief Operating Officer of OSE Immunotherapeutics, in charge of Finance, Business Development and Operations.

Alexis Peyroles graduated from EDHEC Business School and holds an Executive MBA from Imperial College in London.

Profit & Loss (EUR m)	12/16	12/17	12/18	12/19	12/20e	12/21e	12/22e
Revenues	**0.4**	**6.7**	**24.5**	**26.0**	**0.0**	**25.0**	**15.0**
(of which Sales)	0.4	6.7	24.5	26.0	0.0	25.0	15.0
(of which Other revenues)	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Gross profit	**0.4**	**6.7**	**24.5**	**26.0**	**0.0**	**25.0**	**15.0**
Operating costs	8.6	19.3	19.5	27.4	27.0	28.5	31.7
(of which R & D)	5.1	14.6	15.1	21.7	21.5	22.9	25.9
EBIT	**17.5**	**-12.6**	**4.8**	**-1.5**	**-27.0**	**-3.5**	**-16.7**
Net Financial Result	0.1	-0.1	-0.1	0.0	-0.1	0.0	0.0
Pre-tax result	**0.0**	**0.0**	**0.0**	**0.0**	**0.0**	**0.0**	**0.0**
Taxes	3.1	2.2	0.8	-3.2	0.0	-3.5	-1.1
Except. / Discont. operations	0.0	0.1	0.0	-0.1	-0.1	-0.1	-0.1
Associates	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Minorities	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Net declared earnings	**20.6**	**-10.4**	**5.5**	**-4.7**	**-27.1**	**-7.1**	**-18.0**

Cash Flow (EUR m)	12/16	12/17	12/18	12/19	12/20e	12/21e	12/22e
EBIT	17.5	-12.6	4.8	-1.5	-27.0	-3.5	-16.7
Depreciation	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Amortization	0.1	0.1	0.1	0.6	0.0	0.0	0.0
Impairment	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Changes in provision	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Changes in working capital	0.4	3.2	-4.6	8.6	0.0	0.0	0.0
Others	-14.2	3.5	0.7	1.5	-0.1	-3.6	-1.2
Operational Cash Flow	**3.7**	**-5.8**	**1.1**	**9.2**	**-27.0**	**-7.0**	**-17.9**
Tax expenses	-3.1	-2.2	0.0	-0.1	0.0	0.0	0.0
Dividends from associates	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Net interest charges	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Others	0.0	0.0	0.0	0.0	0.0	0.0	0.0
CF from operating activities	**0.6**	**-8.0**	**1.1**	**9.1**	**-27.0**	**-7.0**	**-17.9**
CAPEX	0.0	-0.4	-0.6	-0.3	-0.3	-0.3	-0.3
Investments in intangibles	3.1	0.0	0.1	2.9	2.9	2.9	2.9
Acquisitions	-	-	-	-	-	-	-
Divestments	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Others	3.1	0.1	0.0	-0.2	-0.2	-0.2	-0.2
CF from investing activities	**6.1**	**-0.3**	**-0.6**	**2.4**	**2.4**	**2.4**	**2.4**
Dividend payment	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Minor. & pref. dividends	-	-	-	-	-	-	-
Equity financing	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Others	-1.3	3.1	-0.6	4.8	6.1	-0.9	-0.9
CF from financing activities	**-1.2**	**3.1**	**-0.6**	**4.8**	**6.1**	**-0.9**	**-0.9**
Changes in consolidation scope	-	-	-	-	-	-	-
Exchange rate impact	-	-	-	-	-	-	-
Net debt/cash change	**5.5**	**-5.2**	**0.0**	**16.3**	**-18.5**	**-5.5**	**-16.4**

Notes -

Balance Sheet (EUR m)	12/16	12/17	12/18	12/19	12/20e	12/21e	12/22e
Fixed assets	**53.0**	**53.4**	**53.9**	**55.9**	**55.9**	**55.9**	**55.9**
Tangible fixed assets	0.1	0.4	0.9	1.0	1.0	1.0	1.0
Goodwill	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Other intang. assets	52.6	52.6	52.6	52.6	52.6	52.6	52.6
Financial fixed assets	0.1	0.1	0.1	0.3	0.3	0.3	0.3
Other fixed assets	0.2	0.3	0.3	2.0	2.0	2.0	2.0
Current assets	**36.5**	**24.0**	**23.0**	**33.1**	**14.6**	**9.0**	**-7.3**
Inventories	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Trade receivables	12.3	0.1	2.3	0.7	0.7	0.7	0.7
Other current assets	6.5	11.3	8.3	6.5	6.5	6.5	6.5
Cash & Equivalents	17.8	12.5	12.4	25.8	7.3	1.8	-14.6
Discontinued assets	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Total assets	**89.5**	**77.4**	**76.9**	**88.9**	**70.4**	**64.9**	**48.5**
Total Equity	**64.5**	**55.4**	**61.8**	**58.5**	**40.0**	**34.5**	**18.1**
Equity	64.5	55.4	61.8	58.5	40.0	34.5	18.1
Minorities & preferred	-	-	-	-	-	-	-
Provisions	5.2	3.1	2.2	5.4	5.4	5.4	5.4
Provisions for pensions	0.2	0.2	0.2	0.4	0.4	0.4	0.4
Deferred taxes	5.0	2.9	2.0	5.1	5.1	5.1	5.1
Other provisions	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Other LT liabilities	1.2	4.3	3.8	10.6	10.6	10.6	10.6
LT interest bearing debt	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Current liabilities	**18.7**	**14.5**	**9.1**	**14.3**	**14.3**	**14.3**	**14.3**
ST interest bearing debt	0.6	0.6	0.6	0.9	0.9	0.9	0.9
Accounts payables	4.3	8.8	6.6	6.9	6.9	6.9	6.9
Other ST liabilities	13.8	5.1	1.9	6.6	6.6	6.6	6.6
Discontinued liabilities	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Total liabilities	**89.5**	**77.4**	**76.9**	**88.9**	**70.4**	**64.9**	**48.5**

EV and CE details (EUR m)	12/16	12/17	12/18	12/19	12/20e	12/21e	12/22e
Market cap.	**101.2**	**55.8**	**50.4**	**55.5**	**88.5**	**88.5**	**88.5**
+ Net financial debt	-16.0	-7.6	-8.0	-16.1	2.4	7.9	24.3
(of which LT debt)	0.0	0.0	0.0	0.0	0.0	0.0	0.0
(of which ST debt)	0.6	0.6	0.6	0.9	0.9	0.9	0.9
(of which Cash position)	17.8	12.5	12.4	25.8	7.3	1.8	-14.6
+ Provisions (pension)	5.2	3.1	2.2	5.4	5.4	5.4	5.4
+ Minorities (MV)	-	-	-	-	-	-	-
- Peripheral assets (MV)	-0.1	-0.1	-0.1	-0.3	-0.3	-0.3	-0.3
+ Others	-	-	-	-	-	-	-
Enterprise Value	**98.5**	**50.8**	**36.4**	**63.1**	**101.6**	**118.0**	**111.8**
Equity (group share)	**64.5**	**55.4**	**61.8**	**58.5**	**40.0**	**34.5**	**18.1**
+ Net financial debt	-16.0	-7.6	-8.0	-16.1	2.4	7.9	24.3
+ Provisions (pension)	0.2	0.2	0.2	0.4	0.4	0.4	0.4
+ Minorities	-	-	-	-	-	-	-
- Peripheral assets	-0.1	-0.1	-0.1	-0.3	-0.3	-0.3	-0.3
+ Others	-	-	-	-	-	-	-
Capital employed (for ROCE)	**53.6**	**50.8**	**55.9**	**47.6**	**47.6**	**47.6**	**47.6**
+ Accumulated goodwill amortiz.	-	-	-	-	-	-	-
CE (for ROCE grossed gdwll)	**53.6**	**50.8**	**55.9**	**47.6**	**47.6**	**47.6**	**47.6**

Notes -

Per Common Share (EUR)	12/16	12/17	12/18	12/19	12/20e	12/21e	12/22e
Declared EPS	**1.44**	**-0.72**	**0.37**	**-0.31**	**-1.80**	**-0.47**	**-1.19**
Declared EPS (fully diluted)	1.44	-0.72	0.37	-0.31	-1.80	-0.47	-1.19
CFS	20.70	-10.29	5.58	-4.16	-27.13	-7.10	-17.96
Dividend	0.00	0.00	0.00	0.00	0.00	0.00	0.00
Book Value	4.52	3.83	4.17	3.90	2.67	2.30	1.21
Shares (m)							
At the end of F.Y.	14.290	14.489	14.817	15.006	15.006	15.006	15.006
Average number	14.290	14.489	14.817	15.006	15.006	15.006	15.006
Fully diluted Average number	14.290	14.489	14.817	15.006	15.739	15.739	15.739

Ratios	12/16	12/17	12/18	12/19	12/20e	12/21e	12/22e
P/E	4.9	nm	9.2	nm	nm	nm	nm
P/CF	0.3	nm	0.6	nm	nm	nm	nm
P/BV	1.6	1.0	0.8	0.9	2.2	2.6	4.9
EV/Revenues	257.3	7.6	1.5	2.4	-	4.7	7.5
EV/R & D	-19.1	-3.5	-2.4	-2.9	-4.7	-5.2	-4.3
EV/EBIT	5.6	-4.0	7.5	-42.9	-3.8	-34.0	-6.7
EV/CE	1.8	1.0	0.7	1.3	2.1	2.5	2.3
Dividend yield	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%

Notes -

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Investment rating system: The Degroof Petercam stock ratings are based on the estimated performance relative to the Degroof Petercam Benelux coverage universe. The total return required for a given rating depends on the risk profile relative to this universe. This risk profile is represented by the Beta, as estimated by the analyst. Low risk stocks have an estimated Beta below or equal to 0.9, Medium risk stocks have a Beta between 0.9 and 1.3 and High risk stocks have a Beta equal to or above 1.3. The required relative performance for a given rating is indicated below. The price targets given and the expected relative performance are always based on a 12 month time horizon.

	SELL	REDUCE	HOLD	ADD	BUY
High Beta >= 1.3	RP<-15%	-15%<=RP<-6%	-6%<=RP<+6%	+6%<=RP<+15%	RP>=15%
Medium 0.9 < Beta > 1.3	RP<-10%	-10%<=RP<-4%	-4%<=RP<+4%	+4%<=RP<+10%	RP>=10%
Low Beta <= 0.9	RP<-6%	-6%<=RP<-2%	-2%<=RP<+2%	+2%<=RP<+6%	RP>=6%

RP : Relative Performance against Degroof Petercam coverage universe

Information about rating distribution and analyst remuneration system can be found at: https://sellside-research.degroofpetercam.com
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