EDISON

Oncology Venture

Initiation of coverage

Optimising oncology drug development

Pharma & biotech

Oncology Venture (OV) is a cancer-focused pharmaceutical company that in-licenses discontinued oncology drugs at low cost and uses its mRNA-based drug response predictor (DRP) technology to identify and treat previously unidentified patient populations most likely to respond. OV conducts these focused Phase II trials with an aim to sell or out-license the Phase III-ready drugs with their respective DRPs. OV has in-licensed six assets to date, the most advanced of which is dovitinib from Novartis. Our initial valuation is SEK823.8m or SEK59.56 per share.

Year end	Revenue (SEKm)	PBT* (SEKm)	EPS* (SEK)	DPS (SEK)	P/E (x)	Yield (%)
12/16	1.3	(40.5)	(3.33)	0.0	N/A	N/A
12/17	2.1	(64.9)	(5.28)	0.0	N/A	N/A
12/18e	1.7	(127.9)	(8.81)	0.0	N/A	N/A
12/19e	1.7	(250.0)	(16.39)	0.0	N/A	N/A

Note: *PBT and EPS are normalised, excluding amortisation of acquired intangibles, exceptional items and share-based payments.

Gene expression and the DRP

The DRP uses the transcriptome, which is the collection of the RNA sequences in a cell to identify patients most likely to respond to an anticancer therapy. These active gene readouts are used to characterise the cell lines that are sensitive and resistant to the drug in question to target a subpopulation of cancer patients most likely to respond to the drug.

Six in-licensed assets

Before in-licensing an asset, OV first develops a DRP biomarker model in vitro with the drug and evaluates the model using clinical biopsies and blinded patient response data. If the retrospective evaluation is successful, the two parties enter into a licence agreement, granting OV exclusive rights to develop the asset. OV has in-licensed six assets and plans to start trials targeting the top 10% to 30% of patients who have the highest likelihood of response in focused clinical trials.

Current focused Phase IIs

OV is investigating LiPlaCis for the treatment of the top two-thirds of patients with metastatic breast cancer (mBC) identified by the DRP. So far 17 patients (out of the 20-patient recruitment goal) have been enrolled. Last year, OV enrolled the first patient in the APO010 trial for multiple myeloma (MM) and received Danish Medicines Agency (DMA) approval to initiate the irofulven trial for prostate cancer.

Valuation: SEK823.8m or SEK59.56 per share

We arrive at an initial valuation of OV of SEK823.8m or SEK59.56 per share based on a risk-adjusted NPV analysis of the underlying assets and OV's proportional ownership. As per the business model, each programme is in Phase II development and therefore has significant financing needs (SEK610m by 2020) to reach commercialisation. Based on our estimations, the 2X-111 programme has the highest value (SEK17.58 per OV share).

4 May 2018

Price	**SEK16.2**
Market cap	**SEK224m**
	US$0.12/SEK
Net cash (SEKm) as of December 2017 and January 2018 rights issue	56.7
Shares in issue	13.8m
Free float	67%
Code	OV.SS
Primary exchange	AktieTorget
Secondary exchange	N/A

Share price performance

%	1m	3m	12m
Abs	(4.4)	(29.6)	(56.2)
Rel (local)	(9.1)	(28.4)	(52.7)
52-week high/low	SEK40.0	SEK15.5	

Business description

Oncology Venture is a Denmark-based biopharmaceutical company focused on oncology. Its patent-protected mRNA-based drug response predictor platform enables the identification of patients with gene expression highly likely to respond to treatment. To date the company has in-licensed six drug candidates with the intent to conduct focused Phase II clinical trials and then out-licence the revamped drugs.

Next events

Phase II 2X-121 in breast cancer readout	H218
Phase II LiPlaCis trial completion in breast cancer	H119
Phase II 2X-111 in breast cancer readout	H119

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Investment summary

Company description: Cancer-focused biotech

OV is a clinical-stage pharmaceutical company focused on developing anticancer drug therapeutics targeting patient sub-populations with gene expression most likely to respond to treatment. The company was incorporated in 2012 as a spinout from the Medical Prognosis Institute (MPI), a precision medicine company traded on the Nasdaq First North in Sweden. OV serves as the drug development arm of MPI. It acquires the rights to previously abandoned oncology products and conducts focused Phase Ib/II clinical trials in patients selected for high response rates using MPI's messenger RNA (mRNA)-based DRP. OV's strategy is to either sell or out-license the Phase III ready revamped drugs with its DRP to create opportunities for investor exit. The company has in-licensed six oncology programmes during a range of development phases from small private and large pharmaceutical companies alike. In January this year, OV in-licensed dovitinib, a Phase III tyrosine kinase inhibitor (TKI) from Novartis, making it the most clinically advanced asset in the pipeline.

Valuation: SEK823.8m or SEK59.56 per share

We value OV at SEK823.8m or SEK59.56 per share. This is based on a risk-adjusted NPV analysis of each in-licensed anticancer drug. We value that the 2X-111 programme has the highest value (at SEK17.58 per share of OV) because of the unmet need and lack of effective therapies for glioblastoma multiforme patients ($212.6m in peak sales). We value the remaining assets in a range of SEK50.9m-155.0m with 29% to 100% ownership. Valuations reflect the phase of development across the board, with probabilities of success ranging from 20% to 35%.

Financials: SEK610m needed over three years

OV ended FY17 with SEK12m in cash and equivalents and raised SEK44.7m (2.7m shares at SEK16.30 per share) in January 2018. Our forecasts model SEK610m needed to bring all six anticancer programmes to Phase III for potential out-licensing. We forecast increases in combined R&D expenditure to SEK74m in 2018 and SEK194m in 2019 primarily associated with the advancement of at least four assets into Phase IIa trials. We expect the company to become cash-flow positive by 2023. In March 2018, OV and MPI announced their plans to merge; OV will own 51% of the combined group, however, we do not consider this material to the operations of the company.

Sensitivities: Clinical risks partially mitigated by the DRP

There are inherent risks in clinical development. This case is particularly unique because the respective assets were previously discontinued at various stages in development, which may be indicative of poor pharmacokinetics, miscalculated pharmacodynamics, considerable toxicity profiles and insignificant activity. The core premise of OV's business model is that these risks are mitigated by the use of DRP technology to identify patient populations where the drug may provide a clinical benefit, which therefore may increase the probability of success of clinical trials. However, this also means the success of each programme is heavily dependent upon the accuracy of the unique DRP developed for the particular asset and indication. It is important to note that the DRP may also be highly selective and therefore restrict market potential. Lastly, OV faces significant partnering risk: the company may successfully complete any number of focused Phase II trials demonstrating the use of drug-specific DRP's improves patient outcomes and not identify a partner either out-license or exit to.

Optimising oncology drugs with DRP technology

OV's core strategy is to in-license oncology drugs that demonstrate activity in previously ignored patient sub-populations using its DRP biomarker technology. The goal is to conduct focused Phase Ib/Phase II clinical trials in those patients who have the highest likelihood of response and then either out-license or sell the Phase III-ready drugs with the respective DRP. The DRP biomarker platform assigns each patient a pre-treatment gene expression score that predicts the probability of either sensitivity or resistance to an anticancer agent. These values are derived from transcriptome analysis of cultured cell lines and normalised on a scale of 0 to 100, where a higher number is indicative of a greater likelihood of response. Patients are then elected to participate in a focused Phase Ib trial (eight patients to start) and OV may decide to either discontinue development or enrol more patients to complete the Phase IIa portion of the trial (approximately 20 patients) based on these initial results.

OV listed in July 2015 and has raised approximately SEK140m ($16m) paid in capital. Since its inception, OV has in-licensed six anticancer products that have been previously discontinued during a range of clinical phases and is developing a unique DRP for each asset. OV's parent company MPI granted OV exclusive rights to the DRP technology strictly for drug development for three years (ending in December 2019). The original terms of the agreement also permitted MPI to continue to use the DRP for precision medicine purposes. Bearing in mind evident conflicts of interest for both parties, MPI and OV announced their plans to merge, thus extending the runway for DRP drug development.

OV's pipeline consists of six oncology products that target a variety of cancers with distinct mechanisms of action. OV is developing LiPlaCis, a liposomal formulation of cisplatin, for the treatment of mBC as well as other solid cancers in collaboration with Cadila Pharmaceuticals. OV's ownership of this entire programme is 29%, assuming Cadila fully participates in development. OV is also developing irofulven, a cytotoxic DNA binding agent, and APO010, a Fas receptor agonist. Moreover, OV incorporated two subsidiaries in 2016, 2X Oncology (~92% owned by OV) and OV-SPV2 (~40% owned by OV, 10% owned by MPI, 50% owned by Sass & Larsen Aps) with $4.0m (SEK36.5m) in seed financing primarily from existing OV shareholders. 2X Oncology has two development programmes, a novel liposomal formulation of a chemotherapy that encourages drug delivery across the blood-brain barrier (BBB) as well as a dual poly ADP ribose polymerase (PARP) and tankyrase (TNKS) inhibitor. 2X Oncology is headquartered in Cambridge, MA and Dr Buhl Jensen of MPI and OV serves as chairman of the board. Furthermore, OV formed OV-SPV2 as a spin-out focused on the development of dovitinib, a TKI that was in-licensed from Novartis. OV is fully devoted to the advancement of these subsidiary oncology programmes internally.

Exhibit 1: OV pipeline

Product (company, year acquired)	Indication	Mechanism	Stage	Ownership	Notes
LiPlaCis (LiPlasome Pharma, 2016)	mBC, prostate, head and neck, oesophageal, and skin cancers	Alkylating agent (liposomal formulation of cisplatin)	Phase II	29%	Received CE mark for LiPlaCis DRP in January 2017. Phase II trial in mBC ongoing with 17 patients enrolled (out of 20 patient recruitment goal). OV is responsible for conducting Phase II trial in mBC. Collaboration agreement in place with Cadila Pharmaceuticals since 2016. Cadila is responsible for one Phase III randomised trial (in mBC) and four Phase II trials (in prostate, head and neck, oesophageal, skin cancers). OV will own approx. 29% of the programme after Phase III.
Irofulven (Lantern Pharma, 2015)	Metastatic prostate cancer	Cytotoxic DNA binding agent	Phase Ib/II	100%	Developed by University of California San Diego and previously investigated by MGI Pharma and Eisai (via acquisition of MGI). Received US patent in August 2017 for irofulven DRP. Received allowance from DHMA and ethics committee to conduct Phase II trial in prostate cancer in December 2017. To be initiated in Q218. Lantern will receive licence fees, milestone payments and royalties, and OV will receive shares in Lantern. The financial terms of this agreement have not been disclosed.
APO010 (Onexo, 2012)	MM	Fas receptor agonist	Phase Ib/II	100%	Developed and investigated by TopoTarget/Onexo before in-licensing. Patient recruitment began in May 2017 and dose escalating portion of the trial is ongoing. Following these results the trial is expected to complete within 12 months. OV was granted full rights to APO010, however, further details have not been disclosed.
2X-121 (Eisai, 2017)	mBC and ovarian cancer	PARP/ TNKS inhibitor	Phase II	92%	Owned via 2X Oncology subsidiary. Initiated mBC Phase II trial Q118, IND filing expected in Q218, Phase II mBC and ovarian cancer data readout H218 and Phase II prostate and pancreatic cancer data readout H119. Terms of the agreement have not been disclosed.
2X-111 (2-BBB, 2017)	BMBC, GBM	Anthracycline (GSH PEGylated liposomal doxorubicin)	Phase II	92%	Owned via 2X Oncology subsidiary. Obtained US IND in June 2017. Plans to initiate Phase II trials in Q218 and expects data readout in H119. Terms of the agreement have not been disclosed.
Dovitinib (Novartis, 2017)	Renal and liver cancers	TKI	Phase Ib/II	40%	Owned through OV-SPV subsidiary. Previous Dovitinib activity in Novartis Phase III trial demonstrated PFS of 3.7 months in third-line treatment of metastatic renal cell carcinoma patients versus Bayer's sorafenib activity (PFS 3.6 months). Terms of the agreement have not been disclosed.

Source: Oncology Venture. Notes: The company operates as a unified entity; Fas=first apoptosis signal; BMBC=brain metastasis from breast cancer; DHMA= Danish Medicines Agency; MM= multiple myeloma; GBM=glioblastoma; GSH=glutathione; PARP=poly ADP ribose polymerase; TNKS: tankyrase; TKI=tyrosine kinase inhibitor; PFS=progression free survival.

Development and validation of the DRP algorithm

MPI formed OV in 2012 to serve as its drug development arm with an exclusive licence to its DRP biomarker platform. MPI's DRP uses the transcriptome, which is the collection of the RNA sequences, or active gene readouts and splice variants, in a cell to identify patients most likely to respond to a particular anticancer therapy. The platform was developed in vitro using an established panel of 60 human tumour cell lines from the National Cancer Institute (NCI-60) to correlate the genetic expression profile of a tumour to either sensitivity or resistance to an anticancer drug. Gene expression profiles of the NCI-60 cancer cell lines are derived from a microarray (commercially available Affymetrix Gene Chips) to quantify the level of mRNA transcribed from a nucleic acid molecule that identifies biomarkers. A biological relevance filter is then applied such that only markers previously known to interact are used to reduce the number of false positives. This process generates a list of genes characterising the cell lines that are sensitive and resistant to the drug in question, which is subsequently used to identify a subpopulation of cancer patients most likely to respond to the drug in vivo.

These cell panels are further validated using patient tumour samples or diagnostic formalin-fixed paraffin-embedded biopsies (note these are highly variable sample sets). Gene expression in patients' cells is determined in the same manner as in the cell lines previously described. The sum of the expression levels of the patient's biomarkers is compared to the median of the sums derived from the training set population with the same tumour type to predict either sensitivity or resistance

to the anticancer agent and provides a sneak peek into how the drug will perform in the more variable clinical setting.

The efficacy of the DRP system has been supported in over 25 retrospective studies for a variety of cancers and therapies. One such study evaluated the development of a gene expression score that predicts response to fulvestrant in patients with locally advanced oestrogen receptor-positive (ER+) breast cancer. The prediction score was based on baseline gene expression in the presence of fulvestrant where 103 genes showed increased expression in sensitive cell lines and 311 genes showed increased expression in non-responding cell lines.[1] The DRP was then used to predict patient sensitivity to fulvestrant based on the expression of each gene in the response profile of pre-treatment tumour biopsies obtained from AstraZeneca's Phase II study that investigated neoadjuvant endocrine therapy for women with ER+ breast cancer. These data are combined to produce a predictor score. The patients who clinically responded (ie partial response) to fulvestrant demonstrated a significantly higher sensitivity predictor score than the non-responders (ie stable disease and disease progression) (p=0.01). Moreover, the addition of clinical covariates obtained from the study such as tumour stage and percentage of tumour cells demonstrated a significant difference (p=0.003) between responders and non-responders. Within this trial the positive predictive value was 88% and the negative predictive value was 100%.

In an external validation of the DRP system in collaboration with the MD Anderson Center, the test was evaluated in three distinct datasets including patients treated with epirubicin monotherapy for breast cancer, ABVD (doxorubicin, bleomycin, vinblastine, and dacarbazine) chemotherapy for Hodgkin's lymphoma, and methotrexate for acute lymphoblastic leukaemia. MD Anderson independently selected datasets that satisfied specific conditions set by MPI (ie at least 100 distinct patients receiving the same treatment and availability of treatment outcomes) and sent the list of drugs used to treat the patients to MPI to develop a predictive model in vitro for each drug using the NCI-60 cell lines. MD Anderson then applied the model and compared the predictions with primary patient responses from existing records to evaluate the performance of the DRP. The prediction score in all three cases significantly predicted patient response (p=0.02).[2] However, the paper concluded that although the sensitivity scores based on in vitro models predicted patient response better than chance, the results are not quite compelling enough to change clinical practice,[3] and that there may be an opportunity to develop the DRP for drug development purposes where existing clinical variables are not yet established, to predict the likelihood of patient response. Nonetheless, the DRP also has its limitations. In one retrospective trial, MPI developed DRPs based on in vitro assays to predict patient response (relapsed free survival) to irinotecan treatment for metastatic colorectal cancer. The irinotecan DRP identified 38 positively correlated genes.[4] The irinotecan DRP was unable to predict patient response to irinotecan (p=0.450). The paper discusses that the DRP most likely failed in this case because no significant effect was found with irinotecan treatment and that the population who did benefit from the drug may have been too small to detect using the available patient samples.

The DRP method is patented for more than 70 anticancer agents including vincristine, cisplatin, carboplatin, rituximab etc.[5] While MPI wants to sell the test to determine best treatment in clinical practice, OV is mining discontinued drugs for development to increase the probability of success of

[1] Knudsen S, et al. (2014) Development and Validation of a Gene Expression Score That Predicts Response to Fulvestrant in Breast Cancer Patients. *PLoS ONE* 9(2): e87415.

[2] Wang, W., et al. (2013). Independent Validation of a Model Using Cell Line Chemosensitivity to Predict Response to Therapy. *JNCI: Journal of the National Cancer Institute,105*(17), 1284-1291.

[3] Wang, W., et al. (2013).

[4] Buhl, I. K., et al. (2016). Cell Line Derived 5-FU and Irinotecan Drug-Sensitivity Profiles Evaluated in Adjuvant Colon Cancer Trial Data. *Plos One,11*(5).

[5] US Patent No. 8,445,198

clinical trials by only treating the patient population most likely to respond. Before in-licensing an asset, OV first develops a DRP biomarker model in vitro with the drug and evaluates the model using clinical biopsies and blinded patient response data. If the evaluation is successful, the two parties enter into a licence agreement granting OV exclusive rights to further develop the asset. OV's plan is to include only the top 10% to 30% of patients who have the highest likelihood of response in focused Phase I/II clinical trials.

OV's clinical trial design is considerably cost saving. The company aims to screen approximately 100 potential patients for each new trial; it identifies those most likely to respond, and waits to enrol those patients identified as soon as the patient relapses. Furthermore, the company thoroughly investigates interim data (on approximately eight patients) to determine whether to continue to develop the asset. These methods effectively save time, money and resources.

LiPlaCis: Liposomal cisplatin chemotherapy

Platinum-based chemotherapy drugs (commonly called platins, ie, cisplatin, oxaliplatin and carboplatin) are widely prescribed alone or in combination with other drugs for the treatment of solid tumours since the early 1970s.[6,7] Platins are DNA crosslinking agents that exert antitumor activity by interfering with transcription and/or DNA replication mechanisms.[8] Platins also induce mitochondrial damage, hinder ATPase activity and disrupt cell transport mechanisms that subsequently trigger cytotoxic effects and apoptosis (or cell death).[9]

Nonetheless, platinum-based drug cytotoxicity is not limited to cancer cells and is consequently associated with severe dose-related cell damaging effects, immunosuppression, bone marrow suppression, ototoxicity, peripheral neurotoxicity and, most notably, renal toxicity. Platins inherently bind to extracellular and intracellular proteins, such as serum albumin, which inactivates enzymes and affects drug metabolism, efficacy, and distribution throughout the body.[10] This leads to relatively short blood circulation times and inadequate pharmacokinetics.[11] Limitations such as these have motivated the development of liposomal platinum reformulations and targeted therapy to improve therapeutic efficacy and reduce toxicity.

A number of encapsulated platinum-based formulations have entered the clinic, however, commercialisation has not yet been achieved largely due to inferior response rates in comparison to free platins (Exhibit 2). The development of Aroplatin[12] and SPI-077[13] have similarly been discontinued essentially due to drug inactivity in early dose escalation trials,[14] while the most

[6]Fuertes, M., et al. (2003). Cisplatin Biochemical Mechanism of Action: From Cytotoxicity to Induction of Cell Death Through Interconnections Between Apoptotic and Necrotic Pathways. *Current Medicinal Chemistry,10*(3), 257-266.

[7]Hang, Z., et al. (2016). Platinum-based anticancer drugs encapsulated liposome and polymeric micelle formulation in clinical trials. *Biochemical Compounds,4*(1), 1.

[8]Fuertes, M., et al. (2003).

[9]Babu, A., Amreddy, N., & Ramesh, R. (2015). Nanoparticle-based cisplatin therapy for cancer. *Therapeutic Delivery,6*(2), 115-119.

[10]Ivanov, A. I., et al. (1998). Cisplatin Binding Sites on Human Albumin. *Journal of Biological Chemistry,273*(24), 14721-14730.

[11]Wang, A., et al. (2013). Application of liposomal technologies for delivery of platinum analogs in oncology. *International Journal of Nanomedicine,*3309.

[12]Dragovich, T., et al. (2006). A Phase 2 trial of the liposomal DACH platinum L-NDDP in patients with therapy-refractory advanced colorectal cancer. *Cancer Chemotherapy and Pharmacology,58*(6), 759-764.

[13]Harrington, K. J., et al. (2001). Phase I-II study of pegylated liposomal cisplatin (SPI-077 TM) in patients with inoperable head and neck cancer*. *Annals of Oncology,12*(4), 493-496.

[14]Bulbake, U., et al. (2017). Liposomal Formulations in Clinical Use: An Updated Review. *Pharmaceutics,9*(4), 12.

clinically advanced liposome formulations are Lipoplatin and Nanoplatin. Regulon received EMA orphan drug designation for Lipoplatin for the treatment of metastatic pancreatic cancer and is evaluating the drug in a Phase II/III study. Regulon completed a double-arm Phase III study directly comparing toxicity and efficacy of Nanoplatin versus free cisplatin in combination with paclitaxel (an antineoplastic chemotherapy) in 202 patients with inoperable stage IIIB and IV non-squamous cell non-small cell lung cancer (NSCLC).[15]

Exhibit 2: Liposomal formulations of platinum drugs

Product (company)	Encapsulated drug	Indication	Clinical status
Lipoplatin/ Nanoplatin (Regulon)	Cisplatin	Metastatic pancreatic cancer/ NSCLC	Phase III
MBP-426* (Mebiopharm)	Oxaliplatin	Gastric and gastroesophageal cancer	Phase II
LiPlaCis (Oncology Venture)	Cisplatin	mBC	Phase II
Lipoxal (Regulon)	Oxaliplatin	Advanced gastrointestinal cancer	Phase I
Aroplatin (Agenus)	L-NDDP (cisplatin)	Mesothelioma and metastatic colorectal cancer	Discontinued
SPI-077** (N/A)	Cisplatin	Advanced head and neck cancer, NSCLC	Discontinued

Source: Multiple sources. Notes: MBP-426 is a transferrin (TF) PEGylated liposomal formulation of oxaliplatin; **SPI-077 is a PEGylated liposomal formulation of cisplatin originally developed by Sequus Pharmaceuticals and has been investigated by a number of academic institutions. L-NDDP=liposomal formulation of a third-generation platinum complex analogue of cisplatin.

OV in-licensed LiPlaCis in 2016 from LiPlasome Pharma with the goal of developing a LiPlaCis DRP to identify patients with advanced solid tumours highly likely to respond to the drug. LiPlaCis is a liposomal formulation of cisplatin that is designed to be degraded by secretory phospholipase A2(sPLA2), which is an enzyme expressed by cancerous cells. Increased expression of sPLA2s in tumours was found to be associated with the pathology of cancers of the colon, breast, stomach, oesophagus, ovaries and prostate.[16] In preclinical trials, the use of the sPLA2 enzyme effectively triggered targeted drug delivery.[17] LiPlasome Pharma discontinued the development of the asset due to severe renal toxicity and acute infusion reactions observed during an open-label dose escalating (10-120mg) Phase I clinical trial in 18 patients with advanced solid tumours.[18] Nephrotoxicity severity increased with dose and did not demonstrate any renal-sparing effect as the drug was designed to achieve. Additionally, there was no correlation (p=0.87) between the baseline levels of sPLA2 and the initial half-life (or time required for the concentration of the drug to decrease by half) of the liposome,[19] which therefore indicates that sPLA2 levels are not associated with the breakdown of LiPlaCis in vivo.

OV is investigating LiPlaCis for the treatment of mBC in a single-arm focused Phase II clinical trial treating only the top two-thirds of patients identified by the DRP. In total, 17 patients (out of the 20-patient recruitment goal) who have been exposed to a median of seven prior therapies (excluding all platinum-based therapy) have been enrolled to date. The Norwegian Research Council and Innovation Fund Denmark jointly granted OV and its co-development partner, Smerud, a contract research organisation (CRO), a total of SEK18m to further the development of the LiPlaCis programme. It is important to note that as Smerud is a CRO, it can access grant funds and will likely have a share of the project although this information has not been disclosed. In addition, OV has a collaboration agreement in place with Cadila Pharmaceuticals in which Cadila is coordinating a later Phase III trial in mBC and four Phase II trials in prostate, head and neck, oesophageal and

[15]Stathopoulos, G. P., et al. (2011). Comparison of liposomal cisplatin versus cisplatin in non-squamous cell non-small-cell lung cancer. *Cancer Chemotherapy and Pharmacology,68*(4), 945-950.

[16]Brglez, V., et al (2014). Secreted phospholipases A2 in cancer: Diverse mechanisms of action. *Biochimie,107*, 114-123.

[17]Jensen, S., et al (2004). Secretory phospholipase A2 as tumour specific trigger for targeted delivery of a novel class of liposomal prodrug anticancer etherlipids. *European Journal of Cancer Supplements,2*(8), 183.

[18]Jonge, M. J., et al. (2010). Early cessation of the clinical development of LiPlaCis, a liposomal cisplatin formulation. *European Journal of Cancer,46*(16), 3016-3021.

[19]Jonge, M. J., et al. (2010).

skin cancers. However, it is unclear the degree to which Cadila is participating in development at this point.

The results of the Phase Ib dose escalation portion of the trial revealed that the recommended dose of LiPlaCis is 75mg administered intravenously (IV) in three-week cycles on day one and on day eight. In January 2018, the company reported that seven out of 10 evaluable mBC patients demonstrated clinical benefit, which the company defines as stable disease and partial response, and of the top one-third of patients identified by the DRP to be most likely to respond, five out five demonstrated a median of 25 weeks of stable disease and partial remission. The company expects to complete enrolment in H118.

Market and competitive environment

According to the National Cancer Institute, approximately 252,000 patients in the US were diagnosed with BC in 2017, or 124.9 per 100,000 women on an age-adjusted basis, making it the most common cancer diagnosis in the country. The disease is less commonly diagnosed in the EU, at a rate of 80.3 per 100,000.[20] There were an estimated 40,610 deaths in the US from the disease during 2017, which although large on an absolute scale, makes BC one of the most treatable cancers. Due to screening efforts and other factors, the majority of BC is diagnosed during early stages whereas only 7% and 5% are initially diagnosed at stage III or IV respectively.

Chemotherapy can be given in the induction setting for advanced and metastatic tumours, although it is more common in the second line. In a retrospective study chemotherapy was used in 14% of patients in the first line and 31% in the second line (from a population of post-menopausal stage IV patents).[21] It is also significantly more common for these patients to receive follow up systemic chemotherapy. In addition to anthracyclines and taxanes, more aggressive chemotherapies such as cisplatin, gemcitabine and eribulin, (to name a few) are used in this setting. Recent studies suggest that platins can potentially be used in the treatment of triple negative BC (TNBC)[22] where the gold standard treatment is combination chemotherapy,[23] which typically includes alkylator and anthracycline chemotherapy followed by consecutive taxane treatment.[24] Two TNBC subgroups (basal-like 1 and 2) express high levels of DNA-damage response genes and may be particularly susceptible to the LiPlaCis mechanism of action previously described.[25]

Irofulven: Cytotoxic DNA-binding agent

The molecular pharmacology and precise mechanisms of action of irofulven are not well defined, however, preclinical models have demonstrated its ability to covalently bind to DNA and cellular proteins to inhibit DNA synthesis and induce apoptosis independently of p53 and p21/WAF1 gene expression, which regulate cell cycle arrest.[26] Irofulven's anticancer properties have been investigated in a number of clinical trials in solid cancers by its originator at the University of

[20]EUCAN

[21]Zanotti G, et al. (2017) Treatment patterns and real world clinical outcomes in ER+/HER2- post-menopausal metastatic breast cancer patients in the United States. *BMC Cancer* 17, 393.

[22]Eckstein, N. (2011).

[23]American Cancer Society.

[24]Mayer, E. L., & Burstein, H. J. (2016). Chemotherapy for Triple-Negative Breast Cancer: Is More Better? *Journal of Clinical Oncology,34*(28), 3369-3371.

[25]Lehmann, B. D., et al. (2011). Identification of human triple-negative breast cancer subtypes and preclinical models for selection of targeted therapies. *Journal of Clinical Investigation,121*(7), 2750-2767.

[26]Alexandre, J. (2004). Phase I and Pharmacokinetic Study of Irofulven Administered Weekly or Biweekly in Advanced Solid Tumor Patients. *Clinical Cancer Research,10*(10), 3377-3385.

California San Diego, as well as by MGI Pharma, a US biotechnology company, which acquired the asset in 1993.

Early trials verified the efficacy of irofulven against advanced solid cancers, and particularly in a population of patients with prostate cancer.[27] These findings guided MGI Pharma's investigation of the safety and efficacy of irofulven monotherapy in patients with metastatic hormone-refractory prostate cancer in a single-arm, open label Phase II trial. In total, 42 patients were administered a median of three courses of irofulven. Overall, 15% and 14% of participants experienced grade 3 or 4 thrombocytopenia and neutropenia, respectively, which are common haematological toxifies caused by chemotherapy drug used to treat hormone-refractory prostate cancer.[28] Four out of 32 evaluable patients experienced a partial response while 27 experienced stable disease. Median progression-free survival was 2.9 months. MGI was acquired by Eisai in 2007 for $3.9bn and irofulven development was ceased in 2009 when Eisai returned to its original developer. Lantern Pharma picked up the asset in 2015 and subsequently out-licensed it to OV soon after.

Together, Lantern Pharma and OV received a grant for $800,000 from the Life Sciences International Collaborative Industry Program to support the development of an irofulven DRP to identify patients with metastatic castration- and docetaxel-resistant prostate cancer (mCDRPC) most likely to respond to treatment. OV developed an irofulven DRP based on 205 mRNAs and began the screening portion of the clinical trial at two Danish University hospitals with the intent to screen 300 mCDRPC patients in August 2016. In August 2017, OV obtained a US patent for its irofulven DRP and obtained Danish Medicines Agency approval to initiate the Phase II portion of the trial in December of the same year. According to the company, interim data obtained from the first eight patients enrolled in the study (ie selected by the DRP algorithm to be sensitive to irofulven) will determine whether the company continues to develop this asset. If these select patients experience a particular response, the remainder of the Phase II trial will include 13-27 patients (out of the 300-patient screening) with the highest likelihood to respond to irofulven. The company expects to report interim data in Q318.

Market and competitive environment

The National Cancer Institute estimates that 161,360 patients in the US were diagnosed with prostate cancer in 2017, or 119.8 per 100,000 men on an age-adjusted basis, making it the second most common cancer among men in the US and fifth most common cancer worldwide.[29] There were an estimated 26,730 deaths from the disease in the US during 2017. The stage of prostate cancer at diagnosis is a significant contributor to survival as patients with early local disease have a five-year relative survival rate of almost 100%, while patients with advanced metastasis have a relative five-year survival of 28%.[30] Due to screening advances and more aggressive treatment, there has been an increase and decrease in incidence of localised disease and metastases, respectively.

Prostate cancer eventually progresses with androgen deprivation therapy (or hormone therapy), and this is termed castration-resistant prostate cancer. Newer medicines such as Xtandi (enzalutamide, Pfizer) and Zytiga (abiraterone acetate, Johnson & Johnson, J&J) have significantly improved patient outcomes. The rate of progression free survival at 12-months follow up in one

[27] Eckhardt, S. G., et al. (2000). Phase I and Pharmacokinetic Study of Irofulven, a Novel Mushroom-Derived Cytotoxin, Administered for Five Consecutive Days Every Four Weeks in Patients With Advanced Solid Malignancies. *Journal of Clinical Oncology,18*(24), 4086-4097.

[28] Senzer, N., et al. (2005). Irofulven Demonstrates Clinical Activity Against Metastatic Hormone-Refractory Prostate Cancer in a Phase 2 Single-Agent Trial. *American Journal of Clinical Oncology,28*(1), 36-42.

[29] Darves-Bornoz, A., Park, J., & Katz, A. (2014). Prostate Cancer Epidemiology. *Prostate Cancer,*1-15.

[30] Darves-Bornoz, A., Park, J., & Katz, A. (2014).

Xtandi trial was 68%.[31] Xtandi and Zytiga brought in approximately $2.5bn in sales each in 2017.[32] The gold standard treatment for the castration-resistant population includes docetaxel, which is a taxane chemotherapy, in combination with prednisone, a corticosteroid.[33] However, studies suggest that approximately 50% of these patients are either resistant or develop resistance to docetaxel and do not respond to treatment.[34] p53 protein overexpression is one of several known mechanisms of resistance to docetaxel in prostate cancer as it blocks apoptosis. Therefore, this sub-population of mCDRPC patients may be particularly susceptible to irofulven as it has been shown to inhibit DNA synthesis and induce apoptosis independently of p53 expression in preclinical development.[35]

APO010: Fas receptor agonist

Death receptors are members of the tumour necrosis factor family and are desirable targets for anticancer therapy. One such receptor of interest is the first apoptosis signal receptor (Fas, also known as apoptosis antigen 1 or cluster differentiation 95 [CD95]). The Fas receptor is a transmembrane protein and is predominantly expressed in activated T cells and natural killer cells.[36] The interaction between the natural FAS receptor and its ligand plays a critical role in the regulation of apoptosis and is associated in the pathogenesis of malignancies and immune system diseases.[37] In addition to triggering apoptosis, it has also been recognised that Fas induces cell proliferation in T cells, liver cells and neurons.[38] The Fas agonistic molecule has remained a questionable target as it has demonstrated either too strong haematological toxicities, or negligible activity as most cancer cells are resistant to Fas-mediated apoptosis.[39]

APO010 is a synthetic hexameric formulation of natural Fas ligands that targets Fas receptors on cancer cells to potentially induce caspase-dependent apoptosis and antineoplastic activity.[40] The recombinant molecule was originally developed by Apoxis, a private biopharmaceutical company based in Switzerland and was acquired by TopoTarget in 2007 (when Dr Buhl Jensen was the CEO of the company) from whom OV in-licensed it in 2012. The terms of this agreement have not been disclosed.

TopoTarget's in vitro preclinical models revealed that APO010 cytotoxicity is dependent upon CD95 expression in target cells, in this case human glioma cell lines, where low level CD95 expression is correlated with resistance to CD95-mediatated apoptosis. In in vivo glioma mouse models, the stereotactic injection of 40ng of APO010 on day seven and day 14 after tumour cell implantation demonstrated a survival rate of 80% at day 40 compared to 0% in the control arm. However,

[31]Beer, T. M., et al. (2014). Enzalutamide in Metastatic Prostate Cancer before Chemotherapy. *New England Journal of Medicine,371*(5), 424-433.

[32]Evaluate Pharma

[33]Hotte, S. J., &Saad, F. (2010). Current management of castrate-resistant prostate cancer. *Current Oncology,17*(0).

[34]Magadoux, L., et al. (2014). Emerging targets to monitor and overcome docetaxel resistance in castration resistant prostate cancer (Review). *International Journal of Oncology,45*(3), 919-928.

[35]Alexandre, J. (2004).

[36]Peter, ME., et al. (2015). The role of CD95 and CD95 ligand in cancer. *Cell Death and Differentiation. 22,* 549-559.

[37]Müller, M., et al. (1998). P53 Activates the CD95 (APO-1/Fas) Gene in Response to DNA Damage by Anticancer Drugs. *The Journal of Experimental Medicine,188*(11), 2033-2045.

[38]Peter, M. E., et al. (2015). The role of CD95 and CD95 ligand in cancer. *Cell Death & Differentiation,22*(4), 549-559.

[39]Eisele, G., et al. (2010). APO010, a synthetic hexameric CD95 ligand, induces human glioma cell death in vitro and in vivo. *Neuro-Oncology, 13(2),* 155-164.

[40]Villunger, A., et al. (1997). Constitutive Expression of Fas (Apo-1/CD95) Ligand on Multiple Myeloma Cells: A Potential Mechanism of Tumor-Induced Suppression of Immune Surveillance. *Blood, 90(1),* 12-20.

survival rate did not differ between the APO010 group and control group via systemic injections of 0.015mg/kg body weight three times per week. Histological studies determined that apoptotic tumour cells were only detected in the APO010 treatment group. Thus, the systemic application of APO010 to inhibit tumour growth was ineffective and the company concluded this failure was likely due to insufficient drug delivery to the target.[41]

TopoTarget (now Onexo after the 2014 merger with BioAlliance Pharma) led a Phase I pharmacokinetic, dose-escalating trial of the IV administration of APO010 in 25 patients with non-resectable solid tumours once per week. The results of this trial have not been disclosed, however, according to OV, the study serves as the basis for conducting a focused Phase I/bIIAPO010 trial using the DRP as a companion diagnostic. The Norwegian Research Council granted OV and Smerud approximately $1.64m to cover the costs for the APO010 clinical proof of concept trial. Based on the gene expression profiles of 3,200 human tumours, OV developed a specific DRP to predict APO010 responsiveness. The study revealed MM to be sensitive to APO010 in comparison to some solid tumours tested.[42] In May 2017, OV announced that the first patient was enrolled in the focused Phase Ib/II trial for the treatment of relapsed or refractory MM. OV is targeting enrolment of 15 patients most likely to respond to APO010 out of approximately 150 patient DRP screenings. The company first aims to demonstrate effective APO010 monotherapy and follow-up with combination trials with other agents such as PD-1 inhibitors.

Market and competitive environment

An estimated 30,280 patients in the US were diagnosed with MM in 2017, or 6.6 per 100,000 men and women on an age-adjusted basis, and approximately 12,590 deaths from the disease in the US during the same year.[43] The disease is less commonly diagnosed in the EU at a rate of 4.5 per 100,000.[44] According to the American Cancer Society stage I, II, and III MM are associated with median survival of 62, 44 and 29 months, respectively.

Front-line MM is commonly treated with autologous stem cell transplantation (ASCT). The combination of Velcade (bortezomib, J&J) with dexamethasone prior to ASCT.[45] J&J reported $1,042m in worldwide sales (ex US and Japan for MM and non-Hodgkin lymphoma) in 2017. Takeda distributes Velcade to the US and Japan and reported sales of $1,042m in 2017. Due to its November 2017 patent expiration, we expect sales to decrease over the next five years or so as generic competitors enter the market. For those who are ineligible for ASCT, MM is treated with the combination of bortezomib, melphalan and prednisone. According to one study, an estimated 61% and 38% of the patients with MM relapse and undergo second- and/or third-line treatment, respectively.[46] Relapsed or refractory MM is typically treated with Kyprolis (carfilzomib, Amgen) in combination with lenalidomide and dexamethasone. Amgen reported worldwide sales of $835m (ex Japan and India) for 2017. Because APO010 presumably targets Fas receptors and that preclinical studies have demonstrated the expression of functionally active FasL on B cell malignancies (including MM),[47] APO010 may be useful in the MM patient population.

[41] Eisele, G., et al. (2010).

[42] Vangsted, A., et al. (2016). APO010 sensitivity in relapsed multiple myeloma patients. *Annals of Oncology,27*(Suppl_6).

[43] NCI

[44] Moreau, P., et al. (2017). Multiple myeloma: ESMO Clinical Practice Guidelines for diagnosis, treatment and follow-up†. *Annals of Oncology,28*(Suppl_4), Iv52-Iv61.

[45] Moreau, P., et al. (2017).

[46] Yong, K., et al. (2016). Multiple myeloma: patient outcomes in real-world practice. *British Journal of Haematology,175*(2), 252-264.

[47] Villunger, A., et al. (1997).

It is likewise important to note that the drug development space for MM research is exceedingly competitive, although this is mitigated by the number of different therapies with which patients are treated. Nevertheless, the disruptive force lies in the development of CAR-T cells for the treatment of MM. CAR-T products in development target B cell maturation antigens on the surface of MM cells. The results of an early Chinese clinical trial in 35 patients with MM were presented at the 2017 American Society of Clinical Oncology meeting. Of 19 evaluable patients, 14 had a stringent complete response and five had a partial response. Last year, the FDA approved the first two CAR-T therapies, Kymriah (tisagenlecleucel, Novartis) for the treatment of relapsed acute lymphoblastic leukaemia (ALL), and Yescarta (axicabtageneciloleucel, Kite) for the treatment of relapsed/refractory large B cell lymphoma.

2X-121: Dual PARP/TNKS inhibitor

The most advanced development programme at 2X Oncology is 2X-121, an orally bioavailable small molecule and a dual PARP-1/2 and TNKS-1/2 inhibitor that was in-licensed from Eisai in July last year (previously named E7449). PARPs are a family of 17 enzymes that are involved in cellular metabolic regulation. PARP-1 is a critical anticancer target due to its role in DNA damage repair, maintenance of genomic stability, and functions in transcriptional regulation. More specifically, PARP-1 and -2 nuclear enzymes are responsible for majority of PARP activity in the cell where they are recruited to and triggered by sites of DNA damage.[48] PARP enzymes repair single-strand DNA breaks; as a result, PARP inhibition causes double strand breaks, which require BRCA1/2 for repair.[49] PARP inhibition is therefore particularly lethal in cancers containing BRCA1/2 mutations. TNKS enzymes also belong to the PARP family and are involved in Wnt/β-catenin signalling[50] that play a central role in cancer biology. Wnt overexpression contributes to tumour progression,[51] and consequently, TNKS inhibition interferes with Wnt signalling.

In early clinical trials, 2X-121 demonstrated antitumor activity in BRCA-deficient in vivo models and increased the effectiveness of radiotherapy and chemotherapy.[52] 2X-121 was well tolerated in a Phase I trial in 41 patients with solid tumours, and demonstrated 7.1% partial response. OV's 2X-121 DRP was tested in a small 13-patient blinded retrospective trial using biopsy materials provided by Eisai. The DRP predicted that seven patients would respond to 2X-121 treatment and that six would not respond. Overall survival after 400 days was five and one in the predicted responder and non-responder group, respectively (p=0.07) (Exhibit 3). It is important to note that this trial included cancers without regard to BRCA mutation status where PARP inhibitors are more active.

OV plans to use its unique 2X-121 DRP to select the top 10% patients with mBC and relapsed ovarian cancer highly likely to respond to the drug. OV is in possession of 13,000 capsules for initial studies. The laboratory in Europe is established with approximately 1,400 DRP screened patients with breast cancer while the US lab is undergoing Clinical Laboratory Improvement Amendments validation. The company expects to report interim data from the mBC trial in H218.

[48]Bai, P., & Cantó, C. (2012). The Role of PARP-1 and PARP-2 Enzymes in Metabolic Regulation and Disease. *Cell Metabolism,16*(3), 290-295.

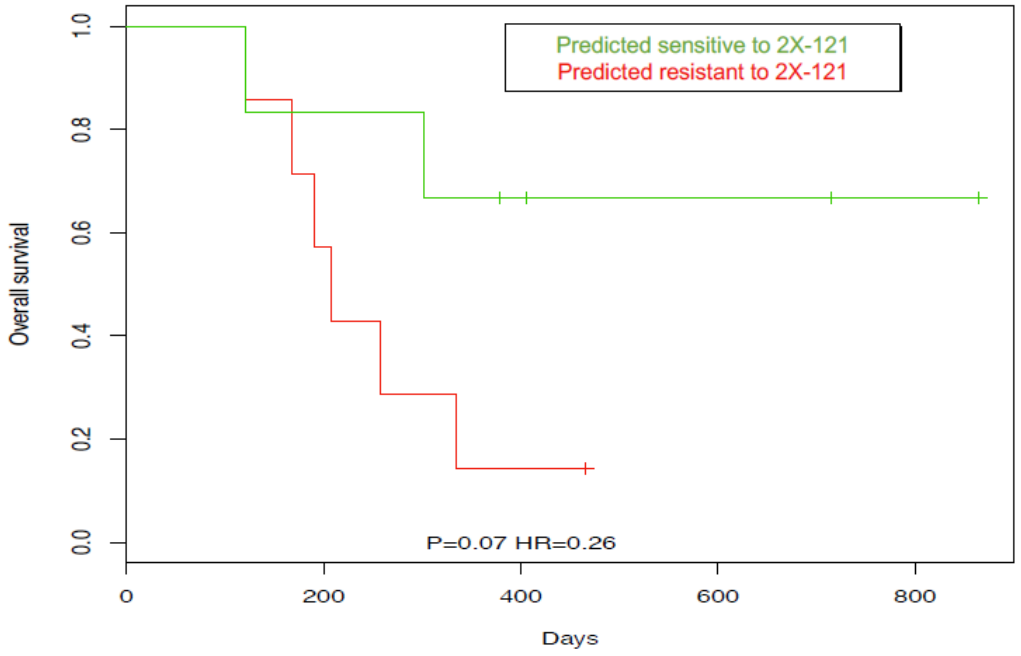
[49]Dziadkowiec, K.N. (2016). PARP inhibitors: review of mechanisms of action and BRCA1/2 mutation targeting. *PrzMenopauzalny 15(4)*, 215-219.

[50]Kamal, A., Riet al. (2014). Tankyrase Inhibitors as Therapeutic Targets for Cancer. *Current Topics in Medicinal Chemistry,14*(17), 1967-1976.

[51]Polakis, P. (2012). Wnt Signaling in Cancer. *Cold Spring Harbor Perspectives in Biology,4*(5).

[52]Mcgonigle, S., et al. (2015). E7449: A dual inhibitor of PARP1/2 and tankyrase1/2 inhibits growth of DNA repair deficient tumors and antagonizes Wnt signaling. *Oncotarget,6*(38).

Exhibit 3: Retrospective DRP validation of 2X-121



Source: Oncology Venture and 2X Oncology

Market and competitive environment

According to the National Cancer Institute, an estimated 22,440 and 252,710 women in the US were diagnosed with ovarian and breast cancers, respectively, in 2017 (11.7 and 124.9 per 100,000 women per year on an age-adjusted basis). Although diagnosed less frequently, ovarian cancer is associated with a relative five-year survival rate of only 46.5% while breast cancer has a relative five-year survival rate of 89.7%.[53] Moreover, of these diagnoses, approximately 15%[54] of all ovarian and 5-10%[55] of all breast cancers have a BRCA germline mutation and are therefore responsive to treatment with PARP inhibitors.

There are several PARP inhibitors on the market and in development (Exhibit 4). Lynparza (olaparib, AstraZeneca/Merck) is approved for the treatment of BRCA1/2 mutated breast and ovarian cancers and is distributed by both AstraZeneca and Merck such that both companies can potentially take advantage of the potential interaction between the PARP inhibitor and their respective immune-oncology drugs, Imfinzi (durvalumab, AstraZeneca) and Keytruda (pembrolizumab, Merck). The companies reported annual sales of $297m in 2017 (profit split 50/50).[56] Not only does 2X-121 inhibit PARP-1/2, but also it inhibits TNKS-1/2 and Wnt signalling, which differentiates the asset from the three PARPS on the market as well as those in development.

[53] NCI

[54] Neff, R. T., et al. (2017). BRCA mutation in ovarian cancer: Testing, implications and treatment considerations. *Therapeutic Advances in Medical Oncology,9*(8), 519-531.

[55] American Cancer Society

[56] Evaluate Pharma.

Exhibit 4: Select PARP inhibitors on the market and in development

Product	Status	Indication	Notes
Lynparza (Olaparib, AstraZeneca/Merck)	Market	Relapsed ovarian cancer, fallopian tube cancer, primary peritoneal cancer after response to platinum-based chemo. Advanced ovarian cancer with BRCA mutation and received three or more prior chemotherapy drugs. Metastatic HER2- breast cancer with BRCA mutation	Inhibitor of PARP1, PARP2 and PARP3
Rubraca (rucaparib, Clovis Oncology)	Market	advanced ovarian cancer with BRCA mutation and have received 2 or more prior chemotherapy drugs	Inhibitor of PARP1, PARP2 and PARP3
Zejula (niraparib, Tesaro)	Market	Maintenance of recurrent epithelial ovarian cancer, fallopian tube cancer, or primary peritoneal cancer in complete or partial response to platinum-based chemotherapy	Inhibitor of PARP1 and PARP2
Talazoparib (Pfizer)	Phase III	Locally advanced/mBC with BRCA mutation	Phase III trial demonstrated median PFS of 8.6 months in talazoparib treatment arm vs 5.6 months chemotherapy in patients with locally advanced/mBC with inherited BRCA mutation
Veliparib (AbbVie)	Phase III	NSCLC and TNBC	Two failed Phase III trials
Pamiparib (BeiGene)	Phase II	Advanced solid tumours	Inhibitor of PARP1 and PARP2
2X-121 (2X Oncology)	Phase Ib/II	DRP identified mBC and relapsed ovarian cancer	Inhibitor of PARP1, PARP2, TNKS1 and TNKS2

Source: Various sources.

2X-111: Penetrating the BBB with GSH

Through 2X Oncology, OV is also developing 2X-111, which is a glutathione (GSH) PEGylated liposomal formulation of doxorubicin (an anthracycline chemotherapy) for the treatment of brain metastases from breast cancer (BMBC) and glioblastoma (GBM). Similar to 2X-111, Doxil (doxorubicin, J&J) is a generic liposomal formulation of doxorubicin (without GSH) that was first approved in 1995 as a refractory chemotherapy. J&J reported worldwide sales of $181m in 2017.[57] 2X-111 was designed to enhance the delivery of doxorubicin to the brain and penetrate the BBB with 2-BBB Medicines G-technology, or GSH. The BBB is a natural barrier between the blood and the brain that maintains homeostasis in the brain extracellular fluid by selectively allowing compounds to penetrate the brain and limits the treatment of brain diseases.[58]

OV in-licensed the asset from 2-BBB Medicines who demonstrated that the blood-to-brain ratio of doxorubicin was 4.8 times greater after administration with 2X-111 (previously 2B3-101) in comparison to the generic liposomal formulation (p=0.0016) in a rat model measured by cerebral open-flow microperfusion.[59] A Phase I dose escalation trial in 28 patients with brain metastases from solid tumours and recurrent malignant gliomas demonstrated 2X-111 tolerability.[60] Pharmacokinetic analysis revealed that drug exposure did not increase linearly or build-up with dose escalation. In addition, the drug also demonstrated preliminary anti-tumour activity at doses greater than 40mg/m^2 whereas four brain metastasis patients, five patients with glioblastoma and three with grade III glioma demonstrated stable disease. The trial concluded that 2X-111 is safe and well tolerated up to 70mg/m^2 in both cancers and guided the Phase IIa expansion studies (Exhibit 5). It is important to note that the previous trials described did not make use of DRP technology.

[57] Evaluate Pharma.

[58] Maussang, D., et al. (2016). Glutathione conjugation dose-dependently increases brain-specific liposomal drug delivery in vitro and in vivo. *Drug Discovery Today: Technologies,20*, 59-69.

[59] Birngruber, T., et al. (2014). Enhanced Doxorubicin Delivery to the Brain Administered Through Glutathione PEGylated Liposomal Doxorubicin (2B3-101) as Compared with Generic Caelyx,®/Doxil®—A Cerebral Open Flow Microperfusion Pilot Study. *Journal of Pharmaceutical Sciences,103*(7), 1945-1948.

[60] Gaillard, P.J. (2014). Phase I dose escalating study of 2B3-101, glutathione PEGylated liposomal doxorubicin, in patients with solid tumours and brain metastases or recurrent malignant glioma. AACR annual meeting 2014; April 5-9, 2014; San Diego, CA.

Exhibit 5: Phase IIa 2X-111 established clinical history

Indication	No. of patients	Results
Brain metastases from breast cancer	17	PR: 12%; SD: 52%
GBM	20	PR: 5%; SD: 35%

Source: 2X Oncology. Notes: These trials did not use the 2X-111 DRP. PR= partial response; SD= stable disease.

OV obtained an IND for 2X-111 in June 2017 and plans to initiate the focused Phase II trials in BMBC and GBM in Q218 using the DRP technology. The company intends to select the top 40% and 10% of BMBC and GBM patients, respectively, most likely to respond to 2X-111 and enrol 20 patients in each trial. OV expects to report interim results in H119 and will determine whether it will consider discussing accelerated approvals with the FDA. For the GBM trial, if four or more patients demonstrate either partial response or stable disease after six months of 2X-111 treatment, the company will pursue accelerated approval, however, if only two to three patients respond, the company will enrol an additional 10 patients in the trial. For the BMBC trial, if 30% or more patients show partial response to 2X-111, the company will repeat the study and pursue accelerated approval with the FDA. However, if only four to five patients respond to treatment, the company will move to evaluate 2X-111 for the treatment of metastases other than the brain.

Market and competitive environment

Breast cancer is the second most common cause of brain metastases (after lung cancer) with rising incidence likely due to the fact that patients are living longer with systemic therapies to control the disease.[61] Of the estimated 124.9 per 100,000 diagnoses made per year in the US (on an age-adjusted basis)[62] a median of 21% (range of 15-35%) of breast cancer metastases to the brain and risk of developing brain metastasis is highly dependent upon primary tumour subtype (Exhibit 6). Despite local and systemic treatment of BMBC, such as whole-brain radiation therapy and stereotactic radiosurgery, only a few patients live longer than one year.[63] Also, cytotoxic therapies such as chemotherapy do not effectively cross the BBB.

Exhibit 6: Frequency of brain metastasis from breast cancer

	Frequency (%)
Median	21
Range	15 to 35
All subtypes	12 to 17
Luminal A	8 to 15
Luminal B	11
TNBC/basal	25 to 27
HER2+	11 to 20

Source: Adopted from Witzel, I., et al. (2016). Notes: TNBC= triple negative breast cancer; HER2+= human epidermal growth factor 2 positive.

GBM, commonly referred to as the 'terminator', is the most aggressive types of tumours of the central nervous system and affects less than 10 per 100,000 people in the US.[64] Despite treatment, (ie surgery to remove the tumour and adjuvant chemo- or radiation therapy) the disease is largely incurable whereas most patients with GBM have a median survival of about 14 to 15 months.[65] Although 2X-111 will not cure BMBC and GBM, the potential of GSH to facilitate the BBB crossing and deliver chemotherapy to the tumour site may effectively improve survival rates for these patient populations.

[61] Witzel, I., et al. (2016). Breast cancer brain metastases: Biology and new clinical perspectives. *Breast Cancer Research,18*(1).

[62] NCI

[63] Witzel, I., et al. (2016).

[64] Holland, E. C. (2000). Glioblastoma multiforme: The terminator. *Proceedings of the National Academy of Sciences,97*(12), 6242-6244.

[65] Hanif, F., et al. (2017). Glioblastoma Multiforme: A Review of its Epidemiology and Pathogenesis through Clinical Presentation and Treatment. *Asian Pacific Journal of Cancer Prevention : APJCP*, *18*(1), 3–9.

Dovitinib: TKI from Novartis

OV in-licensed dovitinib, an oral TKI that inhibits fibroblast growth factor (FGF), vascular endothelial growth factor (VEGF) and platelet-derived growth factor (PDGF) receptors from Novartis in January this year. Although one of the most recent additions to OV's pipeline, dovitinib is the most clinically advanced. OV intends to recondition dovitinib for the treatment of locally advanced or metastatic renal cell carcinoma (RCC) and liver cancer.

Signalling through the FGF pathway regulates cell proliferation and differentiation, angiogenesis, which is the development of new blood cells, as well as cell survival and would healing.[66] Abnormal FGF signalling plays a critical role in clinical tumour progression effecting cellular proliferation, resistance to cell death and chemotherapies, as well as increased angiogenesis and metastases. Similarly, VEGF also modulates angiogenesis in cancer and is stimulated by cancer-causing genes, or oncogenes.[67] Tumour vasculature promoted by VEGF is structurally and functionally irregular though it provides the tumour with nutrients and oxygen for growth. Correspondingly, hyper-active PDGF-receptor signalling via overexpression is associated with the development of malignant disease as well as benign diseases characterised by increased cell proliferation.[68] Therefore, dovitinib may effectively inhibit the growth of highly vascularised cancers that are dependent upon angiogenesis pathways such as RCC.

The safety of dovitinib was evaluated in a Phase I dose-escalating trial in heavily pre-treated (with VEGF and mTOR inhibitors) patients with advanced or metastatic RCC. The study showed that the maximum tolerated dose (MTD) was 500mg/day on a five days on, two days off schedule in 28-day cycles and was generally well tolerated in this cohort.[69] Two out of 15 patients demonstrated partial response, a median progression free survival of 8.1 months and overall survival of 13.3 months. This dovitinib MTD was later tested in a Phase III trial in contrast to Nexavar, an oral multi-kinase inhibitor that was approved in 2005 for the first-line treatment of advanced renal cell liver and thyroid cancer with an expected patent expiry in January 2020. Bayer reported worldwide sales of $850m ($312m in the US) for 2016.

In the randomised open-label Phase III trial, patients with metastatic RCC who previously received one VEGF-targeted therapy and one previous mTOR inhibitor were received either dovitinib (500mg orally, five days on, two days off schedule) or Nexavar (400mg orally 2x daily). Of the 284 patients who received dovitinib treatment and 280 patients received Nexavar. The median progression-free survival was 3.7 months in the dovitinib group compared to the 280 patients who received 3.6 months in the Nexavar group (p=0.063).[70] Adverse events were also similar in both treatment arms including fatigue and hypertension. Novartis ceased dovitinib development because it did not show efficacy or safety benefit over Nexavar. OV plans to develop the drug and its DRP to identify patients with metastatic renal cancer and liver cancer most likely to respond to treatment and plans to develop the asset to commercialisation via its Denmark-based subsidiary.

[66] Lieu, C., Heymach, J., Overman, M., Tran, H., & Kopetz, S. (2011). Beyond VEGF: Inhibition of the Fibroblast Growth Factor Pathway and Antiangiogenesis. *Clinical Cancer Research,17*(19), 6130-6139.

[67] Carmeliet, P. (2005). VEGF as a Key Mediator of Angiogenesis in Cancer. *Oncology,69*(3), 4-10.

[68] Heldin, C. (2013). Targeting the PDGF signaling pathway in tumor treatment. *Cell Communication and Signaling,11*(1), 97.

[69] Angevin, E., et al. (2013). Phase I Study of Dovitinib (TKI258), an Oral FGFR, VEGFR, and PDGFR Inhibitor, in Advanced or Metastatic Renal Cell Carcinoma. *Clinical Cancer Research,19*(5), 1257-1268.

[70] Motzer, R. J., et al. (2014). Dovitinib versus sorafenib for third-line targeted treatment of patients with metastatic renal cell carcinoma: an open-label, randomised phase 3 trial. *The Lancet Oncology,15*(3), 286-296.

Market and competitive environment

The National Cancer Institute estimates that 63,990 patients in the US were diagnosed with RCC in 2017, or 3.9 per 100,000 men and women on an age-adjusted basis. There were an estimated 14,400 deaths in the US from the disease during the same year. Moreover, the disease is associated with a relative five-year survival rate of 74.1%. Treatment for localised RCC includes either partial or radical removal of the kidney followed by adjuvant therapy, such as Sutent (sunitinib, Pfizer). Pfizer reported $1.1bn in sales of the drug for FY17. Management of advanced or metastatic RCC involves as many lines of targeted therapies that a patient may benefit from (Exhibit 7).[71] However, most patients develop resistance to TKIs via a number of mechanisms (ie genetic alterations, activation of other signalling pathways, or the increase in expression of a specific molecule in response to inhibition).[72]

Exhibit 7: RCC competitive landscape

Product	Mechanism	Indication	Notes
Nexavar (sorafenib, Bayer)	TKI of VEGF-1, -2 and -3, FLT3, KIT, and PDFGR-β as well as intracellular kinases	Advanced RCC	Median PFS: 5.6 months
Sutent (sunitinib, Pfizer)	TKI of VEGF-1 and -2, FLT3, KIT, and PDFGR-α and -β	Advanced RCC	Median PFS: 11.8 months (treatment-naïve patients)
Votrient (pazopanib, Novartis)	TKI of VEGF-1, -2, and -3, FGFR-1 and -3, KIT, and PDFGR-α and -β	Advanced RCC	Median PFS: 9.2 months
Inlyta (axitinib, Pfizer)	TKI of VEGF-1, -2, and -3	Advanced RCC after failure of systemic therapy	Median PFS: 6.7 months
Afinitor (everolimus, Novartis)	mTOR inhibitor	Advanced RCC following failure of one or more therapies (i.e. Nexavar, Sutent).	Median PFS: 4.9 months

Source: Multiple sources. Notes: TKI: tyrosine kinase inhibitor; PFS: progression free survival; VEGF= vascular endothelial growth factor; FLT3= Fms-like tyrosine kinase-3; FGF= fibroblast growth factor; KIT= stem cell factor receptor; mTOR= mammalian target of rapamycin.

Approximately 40,710 people in the US were diagnosed with liver cancer in 2017, or 8.6 per 100,000 patients per year. There were an estimated 28,920 deaths attributed to liver cancer in the same year and the disease is associated with a relative five-year survival rate of 17.6%. Liver cancer is often categorised as possibly resectable or transplantable, inoperable with local disease, and advanced, which determines treatment. Although rare, early-stage liver tumours may be resected if a portion of the liver is healthy. If the condition of the liver is inadequate but the cancer is still early stage a liver transplant may be a viable option. Unresectable liver cancers (ie tumours that are either too large to be removed, close to a blood vessel, or the cancer has spread) are treated with either ablation to destroy small tumours, or embolization, which reduces blood flow to the tumour. Targeted therapy, such as Nexavar, is generally used to treat both locally advanced and metastatic disease. In April 2018, Eli Lilly announced positive top-line data from its Phase III trial of Cyramza (ramucirumab, Eli Lilly), a VEGF antagonist, in patients with liver cancer who progressed on first-line Nexavar treatment. The company plans to present the safety, overall survival and PFS results at a future medical meeting and begin regulatory submissions mid-year. Cyramza is currently approved for the treatment of gastric cancer, metastatic colorectal cancer and platinum-resistant metastatic NSCLC.

Sensitivities

In the near term, the primary risks faced by OV are clinical given the stage of its development programmes and heavy reliance on the DRP platform to increase the probability of success of clinical trials by treating the patient population most likely to respond. All OV's drug programmes

[71] Ko, J. J., et al. (2014). First-, second-, third-line therapy for mRCC: Benchmarks for trial design from the IMDC. *British Journal of Cancer,110*(8), 1917-1922.

[72] Bielecka, Z., et al. (2014). Mechanisms of Acquired Resistance to Tyrosine Kinase Inhibitors in Clear - Cell Renal Cell Carcinoma (ccRCC). *Current Signal Transduction Therapy,8*(3), 219-228.

were previously abandoned at one point during clinical development due to poor pharmacokinetics, considerable toxicity profiles and minimal activity. Therefore, the success of these programmes is contingent on the ability of the DRP to mitigate these risks. There is independent evidence of the utility of the DRP, but it was insufficient to change clinical practice in the context of optimising existing care (from the MD Anderson validation study). However, the financial risks associated with these clinical programs are limited by the targeted clinical trial design, stating with small eight-patient pilot studies. The selection of a strict DRP threshold for inclusion significantly minimises market potential of each asset. Current studies will include only the top 10% of responders, but the total fraction of patients that will exhibit a clinical benefit could be significantly larger than this initial set. Finally, OV faces significant partnering risk: the company may successfully complete any number of focused Phase II trials demonstrating the use of drug-specific DRP's improves patient outcomes and not identify a partner to either out-license or exit to, which would negatively impact the company's financials.

Valuation

We arrive at an initial valuation of OV of SEK823.8m or SEK59.56 per share. Our valuation is derived from a risk-adjusted NPV analysis on the future earnings of six active clinical programmes, and as standard practice, this includes costs associated with each asset (Exhibit 8). Our earnings estimates are highly provisional, as the company has not disclosed details regarding patient sub-populations selected by the DRP for each asset. We construct a model market for each development programme based on the presumed mechanism of action of each asset, incidence rates of the indications being studied in the current focused Phase Ib/II clinical trials, and the presumed DRP threshold (Exhibit 9). As we have further efficacy data from ongoing trials, these estimates will be adjusted.

In all cases, our valuations of individual assets include associated costs (R&D, SG&A) in which we assume approximately $100,000 per patient per clinical trial including the costs of DRP development, validation and patient screening. We assume that each Phase IIa trial will include approximately 12-17 patients and that a Phase IIb trial will include 30-45 patients. We also assume a gross-to-net of 75% associated with discounts to payers. For all in-licensed assets we assume royalties of 5% to the respective rights holder as the terms of all the agreements have not been disclosed. The probability of success for each clinical programme is estimated based on an analysis of the underlying asset (Exhibit 9).

Exhibit 8: Key assumptions

Value driver	Indication	Addressable market (per 100,000 patient-years)	DRP threshold	Market penetration	Launch	Launch pricing per patient in the US (Europe)	US peak sales (Europe peak sales) (m)
LiPlaCis	Locally advanced or mBC	7	35%	30%	2023	$91,000 ($68,000)	$102 ($89)
Irofulven	mCDRPC patients alive one year after receiving alternative treatment	1	20%	45%	2023	$129,000 ($97,000)	$22 ($30)
APO010	Third-line relapsed/refractory MM	3	20%	20%	2023	$143,500 ($107,600)	$41 ($40)
2X-121	Locally advanced or mBC and recurrent ovarian cancer with BRCA gene mutations	3	20%	45%*	2023	$132,000 ($92,000)	$55 ($61)
2X-111	BMBC and GBM	6	20%	25%*	2024	$169,000 ($127,000)	$89 ($123)
Dovitinib	Metastatic liver cancer and third-line treatment for metastatic RCC	2	20%	50%	2024	$145,000 ($109,000)	$67 ($85)

Source: Edison Investment Research. Notes: Addressable market opportunity is age-adjusted in the US and in Europe; DRP threshold refers to cut off or the inclusion of patients as identified by DRP (our assumptions); *these are averages for the programme (ie programme is targeting more than one indication); mCDRPC= metastatic castration- and docetaxel-resistant prostate cancer; MM=multiple myeloma; BMBC=brain metastases from breast cancer; GBM=glioblastoma multiform; RCC=renal cell carcinoma.

For the LiPlaCis programme, we calculate an addressable market opportunity of seven per 100,000 woman-years in the US and in Europe (age adjusted), which is comprised of the patient with locally advanced or mBC having received prior chemotherapy. We assume the company will include the top 30% of patients most likely to respond to LiPlaCis treatment identified by the DRP, which corresponds to peak sales of $102m in the US ($89m in Europe). We assume a peak penetration of 30% into this market with a launch price of approximately $91,000 in 2023. In Europe we assume a 25% discount to this price ($68,000). This price is in line and adjusted for future price growth with Doxil, a liposomal formulation of doxorubicin. Our probability of success for LiPlaCis is 25%, which not only reflects the progress OV has made in Phase II development, but also previous failures of liposomal formulations of cisplatin.

Correspondingly, we calculate an approximate market opportunity of one per 100,000 man-years in the US and in Europe (age adjusted) for the irofulven programme. This comprises the patient population with mCDRPC also alive one year after alternative treatment (ie Xtandi or Zytiga). We assume the top 20% of this population identified via gene expression will be treated with irofulven which corresponds to peak sales of $22m in the US ($30m in Europe). We assume a peak penetration of 45% into this specific market with a launch price of roughly $129,000 in the US and $97,000 in Europe assuming a 25% discount, which is in line and adjusted for future price growth with Pfizer's Xtandi. We assume a 20% probability of approval considering the stage of development, limited in-human clinical data, and considering the irofulven DRP has not been proven and therefore may be ineffective.

For the APO010 programme, we estimate a target market of three patient-years in the US and in Europe (age adjusted) comprised of the MM patient population having already received third-line treatment. We assume a 20% DRP threshold for this group. We also assume a peak penetration of 35% with a 2023 launch price of approximately $143,500 in the US and $107,600 in Europe (25% discount), which corresponds to peak sales of $41m and $40m, respectively. This is price is in line (and adjusted for future growth) with Kyprolis. We similarly assign a probability of success of 20% to the APO010 programme, which is based on limited clinical data in humans, but a solid preclinical profile.

We calculate an addressable market opportunity for the 2X-121 programme of three per 100,000 woman-years in the US and in Europe. This estimation is composed of the patient population with locally advanced or mBC and recurrent ovarian cancer with the BRCA gene mutation. We assume a DRP cut off of 20% and an average penetration into these markets of 45%, which corresponds to peak sales of $55m in the US and $61m in Europe with a launch price of $132,000 in the US ($92,000 in Europe) in 2023. We have assigned a 25% probability of approval considering the demonstrated efficacy of PARP inhibitors in BRCA mutated cancers and the early stage of development.

Likewise, we calculate an average addressable market opportunity for the 2X-111 development program of six per 100,000 per patient-years in the US and in Europe, which includes the patient population with brain metastases from breast cancer (two per 100,000) and patients with glioblastoma (four per 100,000). We calculate a relatively small market opportunity for the brain metastases from breast cancer population given its relatively positive prognosis and that a median of 17%[73] of mBC cases metastases to the brain. We assume the top 20% of patients identified by the DRP will be treated with 2X-111 and an average peak penetration of 25% into this market, which corresponds to peak sales of $89m in the US ($123m in Europe) with a launch price of $169,000 in the US ($127,000 in Europe) in 2024. We have assigned a 25% probability of approval to this programme in which we consider the demonstrated the stage of development and ability of the DRP technology to, an anthracycline chemotherapy with a similar drug profile to doxorubicin.

[73]Witzel, I., et al (2016). Breast cancer brain metastases: biology and new clinical perspectives. *Breast Cancer Research : BCR*, *18*, 8.

Lastly, we estimate an addressable market opportunity for dovitinib of two per 100,000 patient-years in the US and in Europe, which includes the patient population having either metastatic liver cancer or renal carcinoma requiring third-line treatment. We assume that the top 20% of patients identified by the dovitinib DRP will be selected for treatment and that the drug will be able to achieve 50% penetration in this market, which corresponds to peak sales of $67m in the US and $85m in Europe. We also assume launch pricing of $145,000 in the US in 2024, which is in line (and adjusted for future growth) with Nexavar. We have assigned dovitinib with a 35% probability of approval, the highest for OV's pipeline at this time, which is based on the substantial amount of pre-existing clinical data that OV has inherited from Novartis. Given the targeted nature of this trial, there is a chance for a faster approval timeline.

We may increase our valuation if it is clear that the company will pursue the LiPlaCis programme for multiple indications as originally planned. Considering these trials have not been detailed we do not include these in the valuation at this time. We intend to update the valuation with the announcement of new deals (ie the in-licensing of new assets, or out-licensing Phase III ready programmes) and will adjust our valuation as we learn more about the details of the ongoing clinical programmes. Furthermore, we may increase our valuation with the potential validation of the DRP in clinical practice for a number of indications. Post-merger, the combined entity will be comprised of 50.3m shares and current OV shareholders will own 51% of the new company.

Exhibit 9: Valuation of OV

Development Program	Indication	Clinical stage	Prob. of success	Launch year	Launch pricing	Peak sales ($m)	rNPV (SEKm)	% owned by OV	OV rNPV (SEKm)
LiPlaCis	mBC	Phase II	25%	2023	$91,000	190.6	377.5	29%	109.5
Irofulven	Prostate cancer	Phase Ib/II	20%	2023	$129,000	52.6	50.9	100%	50.9
APO010	Multiple myeloma	Phase Ib/II	20%	2023	$143,000	80.9	79.3	100%	79.3
2X-121	mBC and ovarian cancer	Phase Ib/II	25%	2023	$132,000	116.4	140.5	92%	129.3
2X-111	Glioblastoma and brain metastases from breast cancer	Phase Ib/II	25%	2024	$169,000	212.6	264.4	92%	243.2
Dovitinib	Renal and liver cancer	Phase Ib/II	35%	2024	$145,000	152.0	387.4	40%	155.0
Total									767.2
Net cash and equivalents (as of December 2017 and January 2018 rights issue) (SEKm)									56.7
Total firm value (SEKm)									823.8
Total shares (m)									13.8
Value per basic share (SEK)									59.56

Source: Edison Investment Research

Financials

OV's FY17 post-tax loss was SEK57.8m (FY16: loss of SEK33.5m), which was primarily attributable to costs associated with the production of irofulven (SEK7m), screening, hospitalisation costs and clinical site costs (SEK6m) as well as the preparation for the Phase II clinical trials (SEK10m) as well as costs associated with 2X Oncology's development pipeline (SEK13m).

OV ended the year with SEK12m in cash and equivalents and raised SEK44.7m (2.7m shares at SEK16.30 per share) in January 2018. As a standalone company, our forecasts model a total of SEK610m (SEK60m in 2018, SEK300m in 2019, and SEK250m in 2020), which we record as illustrative debt, to bring all six of its anticancer programmes to Phase III out-licensing (Exhibit 10). However, following the merger, we expect MPI's cash (DKK3.3m at FY17) to partially offset this funding requirement. Such financial requirements may be offset further via the selling or out-licensing of Phase III ready drugs. These estimates are based on expected cost per patient (ie $100,000 per patient) and Phase II clinical trial size. We do not include the additional four (ie prostate, head and neck, oesophageal and skin cancers) LiPlaCis focused Phase II clinical trials at this time as future of the collaboration with Cadila Pharmaceuticals is uncertain. However, if the company pursues these indications further, we expect this funding requirement to increase.

Conversely, we expect the funding requirement to decrease significantly if the company discontinues any of the development programmes before completing the Phase II trials.

We forecast increases in combined R&D expenditure to SEK74m in 2018 and SEK194m in 2019 primarily associated with the advancement of the LiPlaCis programme into Phase IIb, ongoing irofulven and APO010 Phase IIa clinical trials, as well as 2X Oncology's 2X-121 development programme. We expect the company to become cash-flow positive by 2023. Due to the forthcoming merger between OV and MPI, we expect these financials to change to reflect the new entity.

Exhibit 10: Financial summary

SEK'000s	2016	2017	2018e	2019e
Year end 31 December	Swedish GAAP	Swedish GAAP	Swedish GAAP	Swedish GAAP
PROFIT & LOSS				
Revenue	1,305	2,091	1,727	1,708
Cost of Sales	0	0	0	0
Gross Profit	1,305	2,091	1,727	1,708
EBITDA	(43,408)	(81,001)	(141,440)	(263,526)
Operating Profit (before amort. and except.)	(40,874)	(67,462)	(127,901)	(249,987)
Intangible Amortisation	0	0	0	0
Exceptionals/Other	0	0	0	0
Operating Profit	(40,874)	(67,462)	(127,901)	(249,987)
Net Interest	346	2,588	0	0
Other (change in fair value of warrants)	0	0	0	0
Profit Before Tax (norm)	(40,528)	(64,874)	(127,901)	(249,987)
Profit Before Tax (IFRS)	(40,528)	(64,874)	(127,901)	(249,987)
Tax	6,985	7,114	0	0
Deferred tax	0	0	0	0
Profit After Tax (norm)	(33,543)	(57,760)	(127,901)	(249,987)
Profit After Tax (IFRS)	(33,543)	(57,760)	(127,901)	(249,987)
Average Number of Shares Outstanding (m)	10.1	10.9	14.5	15.3
EPS - normalised (SEK)	(3.33)	(5.28)	(8.81)	(16.39)
EPS - IFRS (SEK)	(3.33)	(5.28)	(8.81)	(16.39)
Dividend per share (SEK)	0.0	0.0	0.0	0.0
BALANCE SHEET				
Fixed Assets	19,767	45,384	31,845	18,306
Intangible Assets	18,885	44,633	31,094	17,555
Tangible Assets	624	485	485	485
Other	258	266	266	266
Current Assets	38,450	33,830	33,614	114,760
Stocks	316	9,149	9,149	9,149
Debtors	6,841	2,593	5,112	9,992
Cash	18,872	11,978	9,242	85,509
Other	12,421	10,110	10,110	10,110
Current Liabilities	(11,820)	(32,461)	(41,171)	(58,765)
Creditors	(11,820)	(32,461)	(41,171)	(58,765)
Short term borrowings	0	0	0	0
Long Term Liabilities	0	0	(60,000)	(360,000)
Long term borrowings	0	0	(60,000)	(360,000)
Other long term liabilities	0	0	0	0
Net Assets	46,397	46,753	(35,712)	(285,699)
CASH FLOW				
Operating Cash Flow	(36,066)	(48,216)	(108,172)	(223,734)
Net Interest	346	0	0	0
Tax	0	0	0	0
Capex	882	(8)	0	0
Acquisitions/disposals	(2,296)	(19,943)	0	0
Financing	39,523	60,702	45,436	0
Dividends	0	0	0	0
Other	0	0	0	0
Net Cash Flow	2,389	(7,465)	(62,736)	(223,734)
Opening net debt/(cash)	(16,786)	(18,872)	(11,978)	50,758
HP finance leases initiated	0	0	0	0
Exchange rate movements	(303)	571	0	0
Other	0	0	0	(0)
Closing net debt/(cash)	(18,872)	(11,978)	50,758	274,491

Source: Company reports, Edison Investment Research

Contact details	Revenue by geography
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Management team

CEO and co-founder: Peter Buhl Jensen	CSO and co-founder: Steen Knudsen
Dr Buhl Jensen is one of the co-founders of OV and has served as the CEO since its inception in 2012. He is also a member of the board at Medical Prognosis Institute where he has been the CEO since 2012. Since 2010, he has been acting CEO of LiPlasome Pharma from which OV has in-licensed the drug LiPlaCis. Formerly, Dr Buhl Jensen co-founded TopoTarget (now Onexo) where he was CEO from 2001 to 2010 and worked on the development of the oncology drug belinostat (Beleodaq, Spectrum Pharmaceuticals H117 net sales of $6.3m).	Dr Knudsen is the CSO and co-founder of OV. Dr Knudsen is also one of the founders of Medical Prognosis Institute where he has been the CSO since 2004. He is the inventor of the drug response predictor (DRO) technology, which OV licensed from Medical Prognosis Institute. Dr Knudsen has a PhD in microbiology.
COO and founder: Ulla Hald Buhl	**Chairman: Duncan Moore**
Ms Buhl is one of the co-founders of OV and has served as the COO since its inception in 2012. In addition, she has served as the COO of Medical Prognosis Institute since 2012. Since 2010, Ms Buhl has served as CCO of LiPlasome Pharma from which OV has in-licensed the drug LiPlaCis. Formerly, she led investor relations at TopoTarget from 2006 to 2010 and head of regulatory development from 2001 to 2006.	Dr Moore has served as the chairman of the board of OV since 2015. He is also a partner in the company East West Capital Partners (since 2007), serves as chairman of the board of Lamellar Biomedical (2013), deputy chairman at Braidlock (since 2015), and non-executive director at Forward Pharma (since 2016). Formerly, Dr Moore served as the global head of healthcare research at Morgan Stanley. Dr Moore has a PhD in biochemistry.

Principal shareholders	(%)
Sass & Larsen Aps	14.53
Buhl Krone Holding ApS	11.47
Medical Prognosis Institute A/S	10.64
DTU Symbion	7.81

Companies named in this report

AstraZeneca (AZN), Bayer (BAYN.DE), Eli Lilly (LLY), Eisai (ESALY), Johnson & Johnson (JNJ)LiPlasome Pharma, Lantern Pharma, Mebiopharm, Merck (MRK), Novartis (NVS), Onexo (ONEXO.EN), Pfizer (PFE), Regulon, 2-BBB Medicines

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