INVEST SECURITIES

BIOTECH

NOXXON

BUY

TARGET PRICE : 4,9€ +358%

INITIATION OF COVERAGE

REFLECTING UPON TUMOR MICROENVIROMENT

We reinitiate the coverage of NOXXON PHARMA with BUY recommendation and a target price of €4.9. The recent topline results from the Phase 1/2 study of the company's most advanced asset, NOX-A12, in combination with Keytruda showed positive clinical activity in the heavily pretreated patients with pancreatic and colorectal cancer. Considering the observed therapeutic benefits in patients with the microsatellite stable tumors, we believe that NOX-A12 is a promising combination partner and an attractive in-licensing asset. In our view, NOXXON PHARMA, with the promising mid-stage clinical program, is an attractive option for the investors interested in oncology space.

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Novel approach to target tumor microenvironment

NOXXON PHARMA is developing novel therapies in oncology through targeting tumor microenvironment (TME). TME generates tumor-supporting conditions, leading to survival and propagation of cancer cells. NOXXON's most advanced asset, NOX-A12, is based on the company's proprietary Spielgemer technology and tackles TME through inhibition of CXCL12 chemokine. By inhibiting CXCL12 activity, NOX-A12 could eliminate the defensive mechanisms of TME, making cancer more susceptible to the cytotoxic and immuno-therapies. NOX-A12 in combination with Keytruda (anti-PD-1 from MERCK & CO) has already shown promising clinical activity in the Phase 1/2 study in heavily pre-treated patients with microsatellite-stable pancreatic (PaC) and colorectal (CRC) cancer. In our view, NOXXON, with the promising combination asset in the fast growing immunotherapy market, is undervalued at the current market valuation of €11m.

NOX-A12 showed encouraging clinical activity in combination with Keytruda

The top-line results form the Phase 1/2 study, presented at the ESMO Conference in December 2018, showed that NOX-A12 in combination with Keytruda achieved disease stabilization in 22% of PaC patients (n=9) and 27% of CRC patients (n=11). Importantly, the combination was able to significantly extend the patients' time on therapy compared to the prior line of treatment. Considering the extremely poor survival prognosis and resistance to the anti-PD-1 therapies of the treated patient population, we believe that these results signal the promising clinical activity of NOX-A12. According to management, the company is planning to release the progression-free survival (PFS) and overall survival (OS) data in 2Q19, which, we believe could be the next catalyst for the stock. We currently project the combination of NOX-A12 plus Keytruda to reach the PaC and CRC markets in the US and the EU in 2023, generating €24m in risk-adjusted revenues and growing to €235m by 2029.

Valuation

We rate shares of NOXXON PHARMA BUY with a price target of €4.9 per share. We derive our price target based on rNPV analysis of projected NOX-A12 sales in PaC and CRC through 2032, presuming a partnership agreement for both indications. Assuming a 15% discount rate, we derive rNPV of €56.1M for PaC and CRC. Upon inclusion of €7M *pro forma* cash and €5.4M of projected capital raise, we arrive at a target price of €4.9 per fully diluted share.

in € / share	2018e	2019e	2020e
Adjusted EPS	-0,30	-0,39	0,38
chg.	*n.s.*	*n.s.*	*n.s.*
estimates chg.	*-37,1%*	*-17,9%*	*-180,1%*

au 31/12	2018e	2019e	2020e
PE	n.s.	n.s.	2,8x
EV/Sales	69,93x	70,45x	0,36x
EV/EBITDA	n.s.	n.s.	0,7x
EV/EBITA	n.s.	n.s.	0,7x
FCF yield*	n.s.	n.s.	96,4%
Div. yield (%)	n.s.	n.s.	n.s.

* After tax op. FCF before WCR

key points	
Share price (€)	1,1
Number of Shares (m)	10,1
Market cap. (€m)	11
Free float (€m)	0
ISIN	NL0012044762
Ticker	ALNOX-FR
DJ Sector	Health Technology

	1m	3m	Ytd
Absolute perf.	-26,2%	-53,9%	-83,1%
Relative perf.	-20,8%	-43,1%	-78,5%

Source : Factset, Invest Securities estimates

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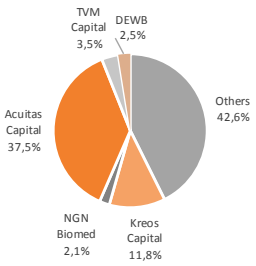
INVESTMENT CASE

We reinitiate the coverage of NOXXON PHARMA with BUY recommendation and a target price of €4.9. The recent topline results from the Phase 1/2 study of the company's most advanced asset, NOX-A12, in combination with Keytruda showed positive clinical activity in the heavily pretreated patients with pancreatic and colorectal cancer. Considering the observed therapeutic benefits in patients with the microsatellite stable tumors, we believe that NOX-A12 is a promising combination partner and an attractive in-lisencing asset. In our view, NOXXON PHARMA, with the promising mid-stage clinical program, is an attractive option for the investors interested in oncology space.

FINANCIALS

Shareholders



Share information	2015	2016	2017e	2018e	2019e	2020e	2021e	2022e
Published EPS (€)	-14,77	-6,71	-2,54	-0,30	-0,39	0,38	-0,49	0,59
Adjusted EPS (€)	**-14,77**	**-6,71**	**-2,54**	**-0,30**	**-0,39**	**0,38**	**-0,49**	**0,59**
Diff. I.S. vs Consensus	*n.s.*	*n.s.*	*n.s.*	*n.s.*	*n.s.*	*n.s.*	*n.s.*	*n.s.*
Dividend	0,00	0,00	0,00	0,00	0,00	0,00	0,00	0,00

Valuation ratios	2015	2016	2017e	2018e	2019e	2020e	2021e	2022e
P/E	n.s.	n.s.	n.s.	n.s.	n.s.	2,8x	n.s.	1,8x
EV/Sales	n.s.	88,18x	144,66x	69,93x	70,45x	0,36x	59,23x	-0,05x
VE/EBITDA	n.s.	n.s.	n.s.	n.s.	n.s.	0,7x	n.s.	-0,1x
VE/EBITA	n.s.	n.s.	n.s.	n.s.	n.s.	0,7x	n.s.	-0,1x
Op. FCF bef. WCR yield	n.s.	n.s.	n.s.	n.s.	n.s.	96,4%	n.s.	-954,7%
Op. FCF yield	n.s.	n.s.	n.s.	n.s.	n.s.	96,4%	n.s.	-954,7%
Div. yield (%)	n.s.	n.s.	n.s.	n.s.	n.s.	n.s.	n.s.	n.s.

NB : valuation based on annual average price for past exercise

Entreprise Value (€m)	2015	2016	2017e	2018e	2019e	2020e	2021e	2022e
Share price in €	*n.s.*	*22,0*	*15,6*	*1,1*	*1,1*	*1,1*	*1,1*	*1,1*
Market cap.	*n.s.*	45	36	11	11	11	11	11
Net Debt	*4,8*	*0,7*	*2,0*	*-0,1*	*0,0*	*-5,3*	*-1,7*	*-11,9*
Minorities	*0,0*	*0,0*	*0,0*	*0,0*	*0,0*	*0,0*	*0,0*	*0,0*
Provisions/ near-debt	*0,0*	*0,0*	*0,0*	*0,0*	*0,0*	*0,0*	*0,0*	*0,0*
+/- Adjustments	*0,0*	*0,0*	*0,0*	*0,0*	*0,0*	*0,0*	*0,0*	*0,0*
Entreprise Value (EV)	**n.s.**	**46**	**38**	**11**	**11**	**6**	**9**	**-1**

Income statement (€m)	2015	2016	2017e	2018e	2019e	2020e	2021e	2022e
Sales	0	1	0	0	0	15	0	20
chg.	*n.s.*	*n.s.*	*n.s.*	*n.s.*	*n.s.*	*n.s.*	*n.s.*	*n.s.*
EBITDA	-15	-9	-5	-4	-5	7	-9	10
EBITA	**-15**	**-9**	**-5**	**-4**	**-5**	**7**	**-9**	**10**
chg.	***n.s.***	***n.s.***	***n.s.***	***n.s.***	***n.s.***	***n.s.***	***n.s.***	***n.s.***
EBIT	-15	-9	-5	-4	-5	7	-9	10
Financial result	-1	-2	-1	0	0	0	0	0
Corp. tax	0	0	0	0	0	-2	0	0
Minorities+affiliates	0	0	0	0	0	0	0	0
Net attributable profit	-16	-11	-5	-4	-5	5	-9	10
Adjusted net att. profit	**-16**	**-11**	**-5**	**-4**	**-5**	**5**	**-9**	**10**
chg.	***n.s.***	***n.s.***	***n.s.***	***n.s.***	***n.s.***	***n.s.***	***n.s.***	***n.s.***

Cash flow statement (€m	2015	2016	2017e	2018e	2019e	2020e	2021e	2022e
EBITDA	-15	-9	-5	-4	-5	7	-9	10
Theoretical Tax / EBITA	0	0	0	0	0	-2	0	0
Capex	2	-2	0	0	0	0	0	0
Operating FCF bef. WCR	**-12**	**-11**	**-4**	**-4**	**-5**	**5**	**-9**	**10**
Change in WCR	-1	2	0	0	0	0	0	0
Operating FCF	**-13**	**-9**	**-4**	**-4**	**-5**	**5**	**-9**	**10**
Acquisitions/disposals	0	0	0	0	0	0	0	0
Capital increase/decrease	16	7	3	6	5	0	5	0
Dividends paid	0	0	0	0	0	0	0	0
Other adjustments	0	0	0	0	0	0	0	0
Published FreeCash Flow	**3**	**-2**	**-2**	**2**	**0**	**5**	**-4**	**10**

Balance Sheet (€m)	2015	2016	2017e	2018e	2019e	2020e	2021e	2022e
Assets	2	1	0	0	0	0	0	0
Intangible assets/GW	0	0	0	0	0	0	0	0
WCR	-4	-2	-2	-2	-2	-2	-2	-2
Group equity capital	-7	-2	-4	-2	-2	3	0	10
Minority shareholders	0	0	0	0	0	0	0	0
Provisions	0	0	0	0	0	0	0	0
Net financial debt	**5**	**1**	**2**	**0**	**0**	**-5**	**-2**	**-12**

Financial ratios	2015	2016	2017e	2018e	2019e	2020e	2021e	2022e
EBITDA margin	n.s.	n.s.	n.s.	n.s.	n.s.	49,1%	n.s.	50,6%
EBITA margin	n.s.	n.s.	n.s.	n.s.	n.s.	49,1%	n.s.	50,6%
Adjusted Net Profit/Sales	n.s.	n.s.	n.s.	n.s.	n.s.	35,1%	n.s.	50,6%
ROCE	n.s.	n.s.	n.s.	n.s.	n.s.	-377,1%	n.s.	-513,8%
ROE adjusted	n.s.	n.s.	n.s.	n.s.	n.s.	159,3%	n.s.	102,7%
Gearing	n.s.	n.s.	n.s.	n.s.	n.s.	n.s.	n.s.	n.s.
ND/EBITDA (in x)	n.s.	n.s.	n.s.	n.s.	n.s.	-0,7x	n.s.	-1,2x

Source : company, Invest Securities Estimates

Next event
Q1 19 : Initiation of phase I/II GBM

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Company Highlights

Noxxon Pharma is a biopharmaceutical company that is leveraging its proprietary Spiegelmer technology to target tumor microenviroment (TME) in order to develop novel oncologic therapies. TME generates tumor-supporting conditions, leading to survival and propagation of cancer cells. The company's most advanced asset, NOX-A12, is designed to target CXCL12 chemokine, a signalling molecule that plays a crucial role in TME and stimulates tumor proliferation, vasculogenesis and metastasis. By inhibiting CXCL12 activity, NOX-A12 could eliminate the defensive mechanisms of TME, making cancer more susceptible to the cytotoxic and immuno-therapies. NOX-A12 in combination with pembrolizumab (anti-PD-1, Keytruda form Merck & Co) has already shown promising clinical activity in the Phase 1/2 study in heavily pre-treated patients with microsatellite-stable pancreatic (PaC) and colorectal (CRC) cancer. Importantly, the top-line results showed that this combination was able to significantly extend the time on the therapy compared to the prior line of treatment. According to management, the company is planning to release the progression-free survival (PFS) and overall survival (OS) data in 2Q19, which, we believe could be the next catalyst for the stock (Exhibit 1). Going forward, we expect the company to pursue the earlier lines of treatment in Pac and CRC and, potentially, in a triple combination, with a cytotoxic therapy in addition to NOX-A12 plus Keytruda. We currently project the combination of NOX-A12 plus Keytruda to reach the PaC and CRC markets in the US and the EU in 2023, generating €24M in risk-adjusted revenues and growing to €235M by 2029 (Exhibit 2).

We believe that NOX-A12, a potentially novel therapy that has already shown the encouraging therapeutic benefits in the hard-to-treat oncologic indications, is an attractive in-licensing asset. With the recent surge among the established immuno-oncology players, such as Bristol-Myers Squibb and Merck & Co, to gain even broader market for their blockbuster anti-PD-1 therapies, we believe that NOXXON is well positioned to strike a partnership agreement by the end of 2020.

Due to the dilutive financing and the convertible debt obligations NOXXON's stock declined gradually over 2017 by -80% (April 30, 2017 – April 30, 2017), compared to a 9% increase by the broader market (CAC Mid & Small) over the same period. We note that the company eliminated all convertible debt obligations, clearing up the capital structure, and secured additional funding through the equity raise, providing the company finical visibility into 2H19. Thus, with the promising mid-stage clinical assets and significant upcoming catalysts in 2019, we believe that it is a good time to start building position in NOXXON's stock.

Exhibit 1: Upcoming catalysts (12-18 months)

Program	Upcoming catalyst	Timing	Impact
NOX-A12	Final Phase 1/2 results (PFS and OS)	2Q19	++
	Initation of Phase 1/2 study in GBM	1Q19	+
	Preliminary results from the Phase 1/2 GBM study	4Q19	+++
NOX-E36	Iniation of Phase 2 study in oncologic indication	2H19	+

Source : Invest Securities estimates

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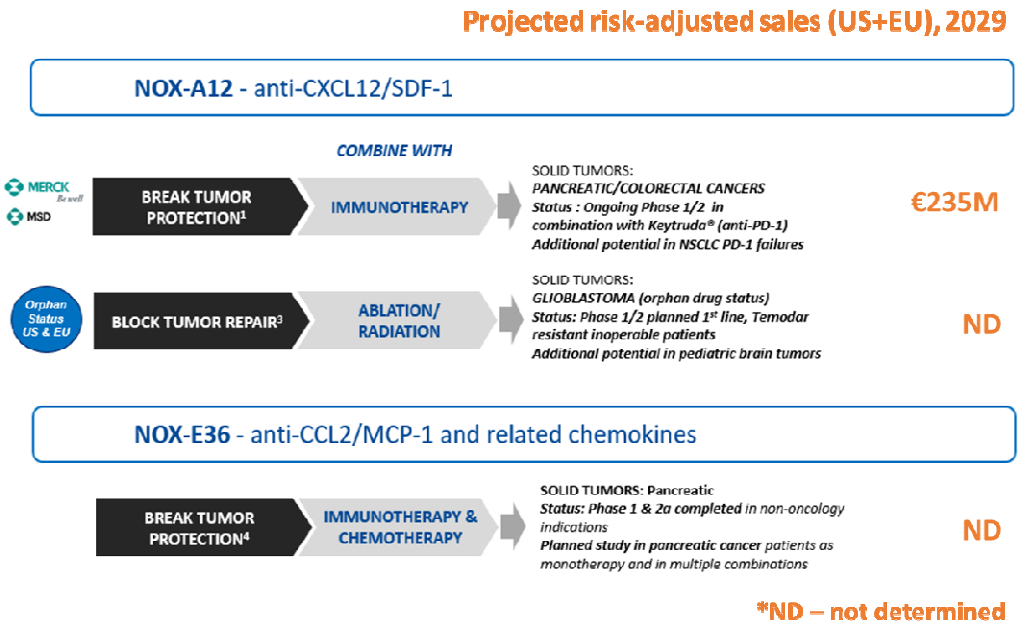
Company Highlights

Exhibit 2: Product pipeline and 2029 revenue projections



Source : NOXXON and Invest Securities estimates.

Source : Invest Securities, Quantum Genomics

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SUMMARY

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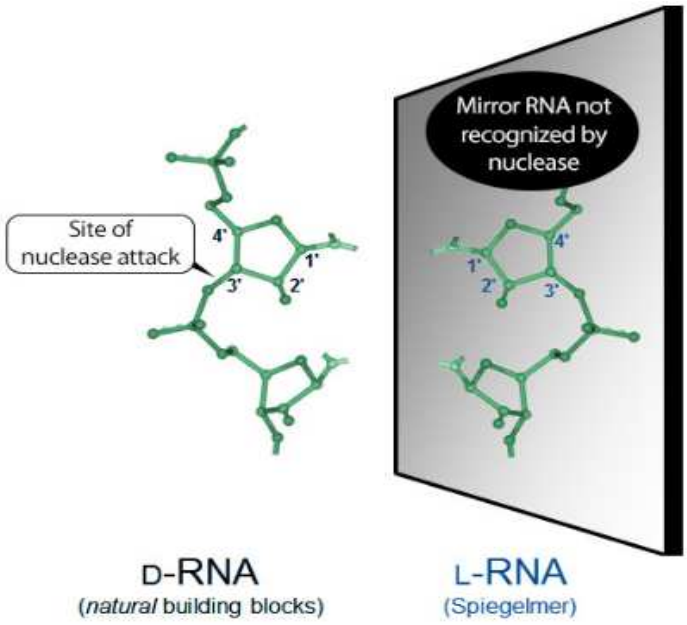
1 – Novel approach to target tumor microenvironment

1.1 Reflecting upon the power of Spiegelmers

NOXXON is leveraging its proprietary Spiegelmer technology to develop the new therapeutics for oncologic indication. Spiegelmers are synthetic single-stranded RNAs (ssRNA) that could be designed to selectively bind a specific target. Single-stranded nucleic acids (RNA and DNA) can form 3D structures based on their nucleotide sequence. Hence, ssRNAs could be designed to form a specific 3D structure that would bind to a selected target, making them an appealing tool for the drug-development. Albeit single-stranded nucleic acids are very unstable in the biological conditions, as they are quickly destroyed by nucleases, the cleaving enzymes that are abundant in the cells. To solve this problem, Spiegelmers technology uses non-natural levorotatory stereoisomers (L-form) of RNA, which are not recognized by nucleases. L-forms of nucleic acids are the mirror reflections of the naturally occurring D-forms (Exhibit 3). Since nucleases could recognize and cleave only D-forms, Spiegelmers are highly stable in biological conditions.



Source : Company's web site

Importantly, Spiegelmers can be directed against all types of targets, whether they are small molecules or macromolecules (such as large proteins). The binding of the Spiegelmers to a functional part of the target molecule could result in modification or disruption of the target's activity. Additionally, unlike biologics (such as antibodies), Spiegelmers are chemically synthesized and do not require complex biological production.

Thus, we believe that Spiegelmer technology provides an unique opportunity to design the drugs with the high specificity and well-controlled production process.

1.2 Chemokine-targeting Spielgemers could battle tumor microenviroment

Based on the Spiegelmer technology, NOXXON is currently developing two asset NOX-A12 and NOX-E36, both targeting the tumor microenvironment through the binding to the specific chemokines. Chemokines are signaling proteins secreted by the cells in order to communicate between each other. Communication through the signaling molecules is a fundamental feature that allows cells to perceive the surrounding environment and respond to it in a coordinated manner.

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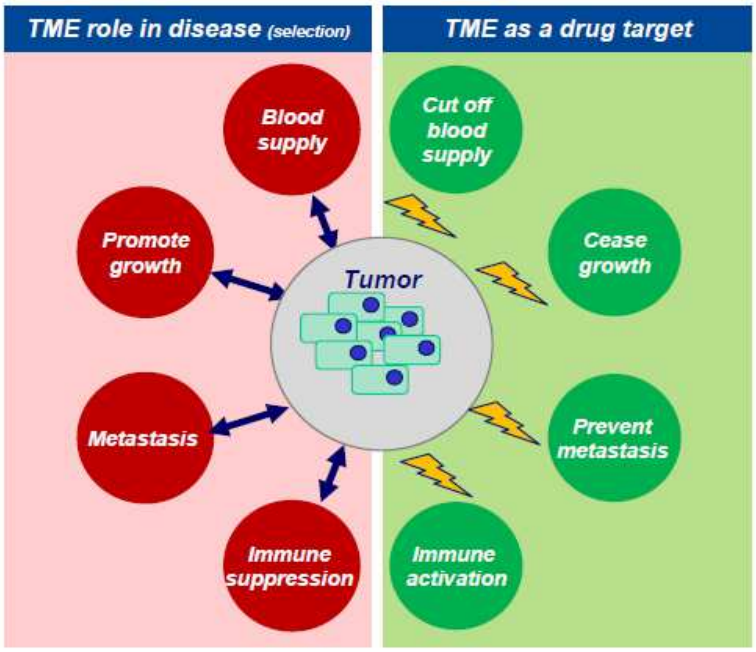
1 – Novel approach to target tumor microenvironment

While in the healthy tissues microenviroment is tightly regulated through cell signaling, the tumors hijacked this mechanism to create tumor microenvironment (TME) that promotes cancer growth and proliferation.

Tumors are not merely one type of cells, but rather a complex assembly of many different cell types that cohabit and communicate within the TME. TME plays a leading role in tumor growth, growth of the supporting blood vessel (angiogenesis and vasculogenesis) and spreading of the cancer cells to the different organs (metastasis) (Exhibit 4).

Exhibit 4: TME plays a crucial role in cancer development



Source : Company's web site

This cancer-nourishing microenviroment is created and maintained by the signaling molecules, such as chemokines. The term "chemokine" is derived from ability of these molecules to induce directed chemotaxis in the nearby responsive cells. In cancer, chemokines could alter the immune response by redirecting the trafficking of immune cells into the tumor microenvironment. Additionally, chemokines can directly promote cancer development through stimulation of tumor cells and the tumor-supporting cells in TME (Exhibit 4). To pass-on the "signal", chemokines bind to their corresponding receptors, unleashing the spectrum of down-stream reactions, leading to tumor growth, proliferation and metastasis. Thus, the ample role of the chemokines in the maintenance of TME makes them an attractive target in oncology. Based on scientific rational, we believe that chemokine-targeting approach has several advantages in the fight against cancer:

■ Cessation of cancer growth

Cancer cells can stimulate stromal cells to produce chemokines, establishing a tumor-stromal interaction that favors tumor growth. Additionally, cancer cells acquired the ability to produce chemokines themselves and to express chemokine receptors, increasing responsiveness to growth signals. Thus, inhibition of chemokine activity could potentially suppress these growth promoting mechanisms.

■ Cut off the blood supply

Since tumor is a rapidly dividing mass of cells, it requires constant oxygen supply, which is normally delivered through blood. Thus, growth of tumor requires formation of the new blood vessels (angiogenesis and vasculogenesis).

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1 – Novel approach to target tumor microenvironment

Chemokines can promote angiogenesis via binding to chemokine receptors found on the endothelial cells. Additionally, chemokines can attract monocytes and endothelial progenitor cells, that are important for vasculogenesis. Disruption of these chemokine-mediated process could lead to starvation of tumor due to the lack of blood supply.

- **Prevention of metastasis**

Metastasis usually develope at the specific anatomic sites, premetastatic niches, that promote the development of metastasis. Premetastatic niches are reach in the specific chemokines, which attract circulating cancer cells that express corresponding chemokine receptor. Chemokines are also responsible for the formation of the circulating tumor cells. Thus, interfering with the chemokine – receptor interaction could prevent the formation of metastasis.

- **Immune activation**

To eradicate a tumor, immune cells must migrate into the TE and the chemokines that attract immune cells play a crucial role in the immune response against tumor. Importantly, there are different types of immune cells: 1) the effector cells that kill the tumor (such as effector T cells); and 2) the immunosuppressive cells that promote tumor growth (such as tumor-associated macrophages, myeloid-derived suppressor cells and regulatory T cells). Specific chemokines have been shown to promote tumor progression by inducing the recruitment of immunosuppressive cells and repelling effector T cells.

Thus, we believe that suppression of the certain chemokine activity could be an effective therapeutic approach to target oncologic indication.

1.3 Chemokine-based approach could boost the efficacy of ICI

While we believe that the chemokine-targeting therapies could be a powerful tool in fight against cancer, in our view, combining chemokine-directed therapies with other oncologic drugs would be a rational developmental path. Speciffically, we believe that combination with immune checkpoint inhibitors (ICIs) and cytotoxic therapy (chemo- or radiation agents) holds a lot of promise. While cytotoxic therapies could help to debulk the tumor and release tumor antigens, ICIs prime the patient's own immune systmem to attack cancer cells.

ICI therapies have seen a lot of success in the recent years, as they release the brakes on immune system to unleash its natural power against cancer. The immune cells could naturally recognize and attack cancer cells. Albeit tumors learned how to avoid this recognition through overexpression of the immune checkpoints (such as PD-L1 receptor) that signal the immune system to hold back. ICIs disrupt this inhibitory signaling and prime immune cells to fight tumors. This approach has rapidly become the standard of care in numerous cancers. Among the ICIs, the anti-PD-1 therapies were most successful, with the estimated $7B sales and 83% annual growth rate at the end of 2018. While anti-PD-1 drugs triumphed in the oncology sector, these therapies are effective in roughly 20-40% of cancer patients.

The major strategic challenge for the immuno-oncology players became the development of the combination approach, based on the potential therapeutic synergies. The goal is to make the ICIs even more effective, and especially in certain cancers (including pancreatic, colorectal and brain cancers) that are currently not responsive to ICI therapies. Consequently, all the major players that are well-positioned in the immuno-oncology area (BMS, Merck & Co, Roche, AZN, Pfizer etc.) have entered into a race to find successful combinations, ultimately making the combination assets the attractive take-over targets.

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1 – Novel approach to target tumor microenvironment

Immune recognition of cancer cells can only occur in the presence of lymphocytes. Hence, a major obstacle to obtain the maximal therapeutic effect from immunotherapy is the lack of tumor-infiltrating effector T cells at the tumor site due to abnormal angiogenesis, hypoxic conditions and an overall suppressive tumor microenvironment (TME). TME also favors immunosuppressive cells, including tumor-associated macrophages, myeloid-derived suppressor cells and regulatory T cells. A high local concentrations of chemokines have been shown to repel cytotoxic effector T cells and promote immunosuppressive TME. Thus, we believe that there is a strong scientific rational to combine ICIs with the chemokine-targeting drugs in order to enhance the therapeutic effect.

Based on the scientific rational, we believe that combination of the NOXXON's chemokine-targeting programs with ICIs could result in the therapeutic synergy and provide an attractive out-licensing opportunity. Additionally, we believe that such combination could be reinforced with the cytotoxic therapy in the future clinical development.

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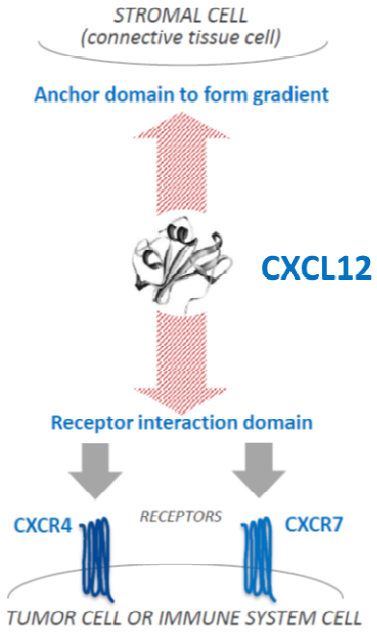
2 – NOX-A12 is a promising clinical asset

2.1 NOX-A12 could efficiently inhibit CXCL12

NOXXON's most advanced asset, NOX-A12, is a Spiegelmer that targets chemokine CXCL12. C-X-C motif ligand 12 (CXCL12), also known as stromal cell-derived factor 1 (SDF-1), plays an important role in the cell migration and the regulation of TME through binding to its receptors CXCR4 and CXCR7 (Exhibit 5). Upon binding to its receptors, CXCL12 promotes tumor proliferation, angiogenesis, vasculogenesis, and metastasis. Additionally, it suppresses programmed cell death (apoptosis), leading to survival of tumor cells. For instance, in pancreatic cancer, high expression of CXCR4 was associated with the advanced disease, and CXCR7 – with the development of metastasis. The overexpression of CXCR4 and CXCR7 renders the tumor cells responsive to CXCL12 ligand. Additionally, the high concentration of CXCL12 in the TME was shown to form a repulsive barrier that turns away cytotoxic lymphocytes, preventing the immune system to attack the cancer.

Exhibit 5: CXCL12 acts through its biding domains



Source : Company's presentation, 2017

Importantly, CXCL12 has two binding sites (receptor-binding and non-specific anchor), and NOX-A12 is designed to interfere with both CXCL12 binding sites in order to fully inhibit chemokine's activity (Exhibit 5). Both binding sites are important for the efficacy of CXCL12-targeting therapy:

- blockade of receptor-binding domain could disrupts down-stream reactions;
- blockade of anchor domain could neutralize CXCL12 concentration gradient.

While neutralization of CXCL12 concentration gradient is important to eliminate the chemo-repulsion of lymphocytes, the blockade of receptor binding domain is crucial for disruption of the direct CXCL12-mediated activity. By targeting chemokine itself and not the corresponding receptors, NOX-A12 is blocking the interaction of all CXC12 functions, suggesting the potentially higher therapeutic impact.

Hence, the disruption of CXCL12/CXCR4/CXCR7 axis represents and attractive therapeutic approach, as it could lead to the infiltration of lymphocytes into TME and the disruption of the tumor-supportive mechanisms.

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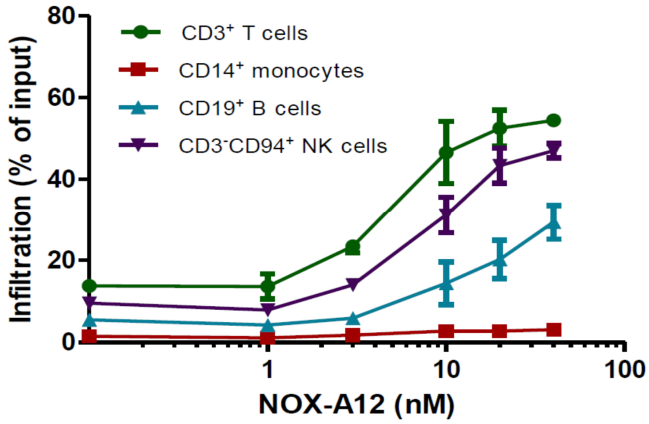
2 – NOX-A12 is a promising clinical asset

2.2 NOX-A12 could provide synergy with anti-PD-1 therapies

The lack of efficacy and low response rate to anti-PD-1 antibodies in certain cancers could stem from the fact that effector T cells, activated by these drugs, fail to permeate the immunosuppressive TME. Notably, by suppressing the immuno-repulsive activity of CXCL12, NOX-A12 could facilitate the infiltration of lymphocytes into tumors. In the preclinical models, NOX-A12 was able to significantly increase the amount of lymphocytes at the tumor site, including natural killer cells, B cells and T cells (Exhibit 6).

Exhibit 6: NOX-A12 attracts lymphocytes to tumor site



Source : Company's presentation, AACR, 2016

Since the infiltration of lymphocytes is crucial for the efficacy of immuno-oncology drugs, we believe that NOX-A12 could provide a significant therapeutic synergy as a combination partner. By interfering with the CXC12/CXCR4/CXCR7 axis, we believe that NOX-A12 could potentially lift the protection of the immunosuppressive TME, exposing cancer cells to the attack of immune system. **Based on this scientific rationale, in our view, combining NOX-A12 with an anti-PD-1 therapy could promote the infiltration of effector T cells (through NOX-A12) that were already activated to fight cancer (through anti-PD-1).**

2.3 NOX-A12 showed encouraging clinical activity in combination with Keytruda

NOX-A12 has already shown positive signs of clinical efficacy in combination with anti-PD-1 therapy, pembrolizumab (Keytruda from MERCK & CO), in patients with end-stage colorectal (CRC) and pancreatic (PaC) cancers. The combination is currently being evaluated in the Phase 1/2 study and the top-line results were presented at the ESMO Immuno-Oncology Conference in December, 2018. In-line with the previously reported results from NOX-A12 monotherapy, the combination of NOX-A12 plus Keytruda was well tolerated, with adverse event profile close to Keytruda alone: 47.3% grade 1; 36.5% grade 2; 15.5% grade 3; no grade 4 and 0.7% grade 5 (due to tumor progression). Importantly, the results from the Phase 2 part of the study showed that NOX-A12 plus Keytruda achieved the disease stabilization in 22% of PaC patients (n=9) and 27% of CRC patients (n=11) (Exhibit 7, violet bars).

We note that studied population had multiple prior lines of treatment (PaC – median 3, CRC – median 5) and very poor prognosis with an estimated survival less than 6 months (Exhibit 8). Notably, 35% of patients (n=20) were able to stay on the NOX-A12 plus Keytruda combination therapy considerably longer compared to the previous line of treatment (Exhibit 7). Considering that there is no available treatment for the end-stage PaC and CRC, presented results suggest encouraging clinical benefits of the NOX-A12 plus Keytruda combination in these hard-to-treat indications.

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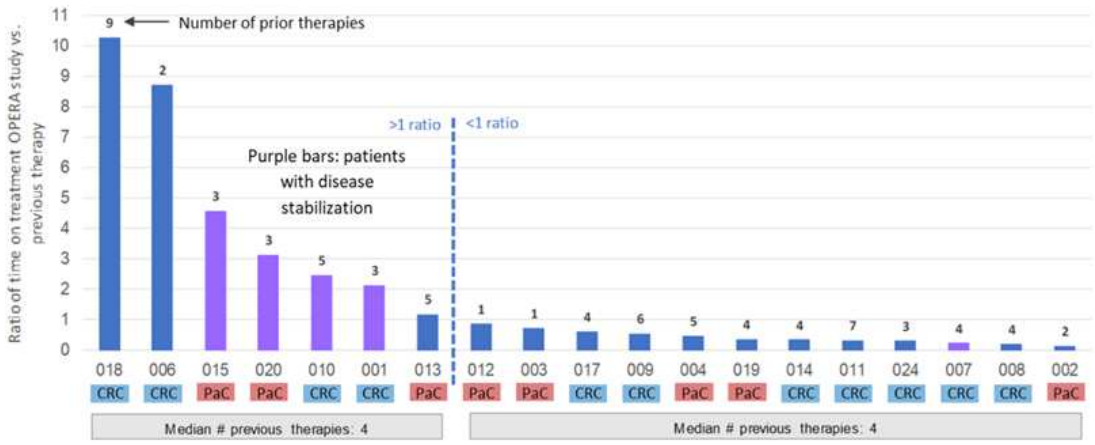
2 – NOX-A12 is a promising clinical asset

Exhibit 7: NOX-A12 plus Keytruda increased the time on treatment



Source : Company's presentation, ESMO, 2018

Exhibit 8: Patients demographics

	Colorectal Cancer	Pancreatic Cancer
N	11	9
Male/Female	7 / 4	8 / 1
Age, mean (range)	63 (55 – 73)	67 (48 - 82)
Stage at study entry	100% stage IV (metastatic)	
Microsatellite status at study entry	All patients MSS	
Prior lines of systemic treatment, mean (range)*	5 (2 – 9)	3 (1 – 5)
Patients with prior surgery (# of surgeries)	7 (1 – 4)	3 (1 – 2)
Best response last treatment	PD (10), SD (1)	PD (9)
Time since last systemic prior treatment (mean)	2.0 months	1.5 months

* excluding surgery

Source : Company's presentation, ESMO, 2018

Importantly, all patients in the study had microsatellite-stable (MSS) tumors, a subpopulation that does not respond to anti-PD-1 therapy. For instance, while Keytruda achieved objective response rate (ORR) of 36% in CRC with high microsatellite-instability (MSI-H) (n=90), the best response that was seen in MSS CRC was 11% SD (n=18). In PaC patients, Keytruda showed 83% ORR (n=6) in MSI-H disease and no reported responses in MSS type. These historical data suggest that the observed benefits could be attributed to the combination regimen rather than Keytruda alone.

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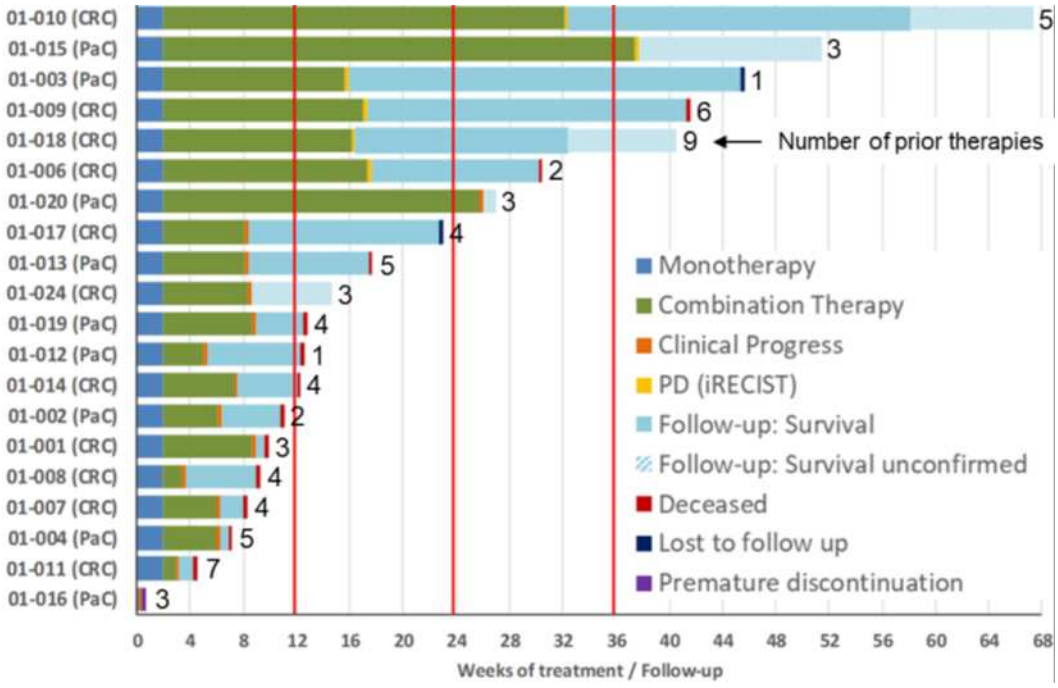
2 – NOX-A12 is a promising clinical asset

Exhibit 9: Patients time on study



Source : Company's presentation, ESMO, 2018

Additionally, while progression-free survival (PFS) and overall survival (OS) results have yet to mature, we note that 35% of patients survived at 6-months (7 out of 20) and 71% of them (5 out of 7) stayed on NOX-A12 plus pembro combination longer than on the previous treatment regimen (Exhibit 9 and 6). In our view, the observed survival benefits suggest that NOX-A12 plus pembro combination could out-perform the earlier lines of therapy and could show efficacy in the earlier treatment lines, with potentially broader market. According to management, the company is planning to release the PFS and OS data in 2Q19, which, we believe could further strengthen the reported top-line results.

Going forward, we expect the company to go after earlier lines of treatment in Pac and CRC and, potentially, with a tipple combination, adding cytotoxic therapy to NOX-A12 plus pembro combination. **We currently project combination of NOX-A12 plus Keytruda to reach the PaC and CRC markets in the US and the EU in 2023, generating €24M in risk-adjusted revenues and growing to €235M by 2029.**

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2 – NOX-A12 is a promising clinical asset

2.4 NOX-A12 stand out among the competition

Owning to its ample role in cell signaling, CXCL12/CXCR4 axis attracted a lot of interest in oncology, including large pharma players. Notably, due to the specific role of CXCL12/CXCR4 in hematopoietic stem cell (HSC) mobilization, the early studies were focused on targeting this pathway to mobilize HSCs from the bone marrow and release them into the blood. The release of HSCs into the blood stream helps to harvest these cells from the donor for the subsequent transplantation to the recipient with advanced hematological cancer. We note that CXCR4 inhibitor, plerixafor (Mobozil from Sanofi), was approved for HSCs mobilization for the transplantation back in 2008. Since then, many other avenues of CXCR4-targeting therapies were explored, with a lot of focus on the treatment of hematologic and solid tumor, albeit with the limited clinical success.

Discontinuation of the several clinical programs resulted in a setback in the field:

- Bristol-Myers Squibb discontinued its combination study of ulocuplumab (and anti-CXCR4) plus nivolumab (anti-PD-1, Opdivo from Bristol-Myers Squibb) in non-small cell lung cancer and pancreatic cancer.
- Eli Lilly terminated the study of its LY2510924 (CXCR4 inhibitor) with durvalumab (anti PD-L1) in XXX.
- Pfizer has discontinued its PF-06747143 (CXCR4 inhibitor)

In our view, the lack of efficacy that prompted the discontinuation of the early studies could be partly associated with CXCR4-centric approach and poor understanding of the biological mechanisms behind it. Unlike the CXCR4-focused approach, NOX-A12 targets CXCL12 chemokine, therefore leading to the blockage of both CXCL12/CXCR4 and CXCL12/CXCR7 pathways. Targeting both CXCR4 and CXCR7 simultaneously, has long been considered a more efficient pharmacological approach, since both receptors are involved in the cancer development. Additionally, NOX-A12 was shown to have a significantly prolonged duration of molecular response, as seen by the mobilization of the hematopoietic stem cells in the blood when compared to historical Mobozil results. As such, NOX-A12 was able to mobilize myeloma cell for up to 3 days compared to 1 day mobilization of HSC with plerixafor. Thus, we believe that, in addition to differentiated mechanism of action, NOX-A12 could also exhibit superior pharmacokinetic profile.

Despite some of the earlier setbacks, there has been a recent surge in interest in the industry to develop CXCR4-targeting therapies. Currently, the number of biotechnology companies pursuing the CXCR4-based approach has been on the rise (Exhibit 11). We note that most of the CXCR4-targeting assets are being developed in combination with cytotoxic chemotherapy or immunotherapy. Due to the excellent safety profile of NOX-A12, we believe that the triple combination (NOX-A12 plus chemo plus ICI) could carry the highest value.

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2 – NOX-A12 is a promising clinical asset

Exhibit 11: Selected CXCR4-targeting companies

Drug	Company	Phase	Indication	Combination regimen
Plerixafor	Sanofi	Marketed	Stem cell mobilisation	G-CSF
Burixafor	TaiGene	II	Stem cell mobilisation	G-CSF
BL-8040	BioLineRx	II	Pancreatic cancer	Keytruda and Chemo, 2L
LY2510924	Eli Lilly	I	Acute myeloid leukemia	Chemo
X4P-001	X4 Pharmaceuticals	II	Renal cell carcinoma	Inlyta (tyrosine kinase inhibitor)
		II	WHIM Syndrome	N/A
USL-311	Proximagen	I/II	Glioblastoma	Chemo
POL6326	Polyphor Ltd	III	Breast cancer	Chemo
Ulocuplumab	BMS	I/II	Acute myeloid leukemia	Chemo
		I/II	Waldenstrom's Macroglobulinemia	Ibrutinib (Bruton's tyrosine kinase inhibitor)

Source : Invest Securities

At this time, we believe that BL-8040 from BioLineRx represents direct competition to NOX-A12. BL-8040 is a short peptide that functions as an antagonist for CXCR4. BioLineRx is evaluating BL-8040 in combination with Keytruda in the Phase 2 COMBAT/Keynote-202 study in recurrent PaC patients. COMBAT results showed that BL-8040 plus Keytruda achieved SD in 32% of PaC patients and 6-months survival rate of 34%. We note that 58% of patients in the BioLineRx study had just 1 prior line of therapy, suggesting significantly more favorable diseases profile of evaluated patients compared to NOXXON's study. Nevertheless, the 6-months survival rate was still comparable (35% for NOX-A12 combo versus 34% for BL-8040 combo), signifying potentially stronger therapeutic activity of NOX-A12. Currently, BIOLINERX amended the study to add an extra arm with the chemotherapy on top of BL-8040 plus Keytruda combination. While in our view, NOXXON's results compare favorably to BIOLINERX, we believe that NOXXON could also pursue the earlier lines of treatment and in triple combination with additional cytotoxic agent.

Thus, in our view, the unique mechanism of action of NOX-A12 provides a significant competitive advantage compared to CXCR4-targeting therapies. With the differentiated mid-stage clinical program that has already shown the encouraging therapeutic benefits in the hard-to-treat oncologic indications, we believe that NOXXON is well positioned against the competition.

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3 – Potential upsides

3.1 Glioblastoma as a potential expansion indication for NOX-A12

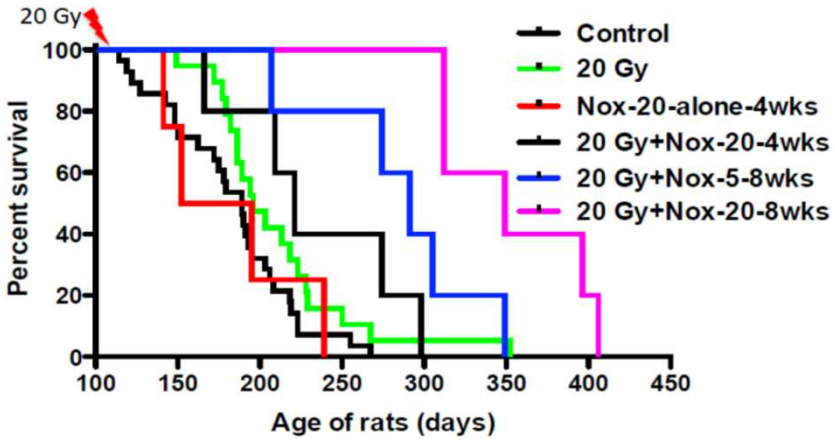
NOXXON is also planning a Phase 1/2 study of NOX-A12 in combination with radiotherapy in glioblastoma (GBM) patients with unmethylated MGMT. GBM remains the most frequent primary brain tumor, with a high recurrence rate and very poor survival prognosis of 3-20 months. Currently, the standard first-line therapy for GBM is a maximal resection, followed by radiotherapy (RT) and concurrent and maintenance treatment with temozolomide (TMZ), a chemotherapy agent. Resistance to TMZ could be provoked by O6-methylguanine-DNA methyltransferase (MGMT), which repairs TMZ-induced damages in cancer cells. Methylation of MGMT promoter halts the production of this enzyme. Consequently, GBM patients with unmethylated MGMT signature are unlikely to respond to TMZ and represent a population with high unmet medical need.

It is thought that GBM has a high rate of recurrence due to radioresistant tumor cell clones that stem from the hypoxic regions of the tumor with low drug-permeability. In the tumor, hypoxic regions are developed due to the accelerated growth and the inappropriate vasculature. Hypoxia activates the defensive mechanisms of the cells, including the resistance to cytotoxic insults (RT) and chemokine-mediated recovery. CXCL12, upregulated by the hypoxia-induced factor 1 (HIF-1), was shown to promote the recovery of tumor after the cytotoxic insults by attracting the growth-supporting cells and reestablishing tumor vasculature. Interestingly, specifically vasculogenesis (orchestrated by bone marrow-derived cells) and not angiogenesis (supported by endothelial cells) was shown to be important for GBM recurrence. Therefore, inhibition of the CXCL12 activity could lead to suppression of these recovery mechanisms, including vasculogenesis. Therefore, in the light of this scientific rationale, we believe that combining RT with NOX-A12 could be particularly promising therapeutic approach.

We note that the importance of CXCL12/CXCR4 blockade for GBM recurrence was shown by the academic group from Stanford, US, using plerixafor. The results form the Phase 1/2 study of plerixafor plus RT in newly diagnosed GBM patients showed that the combination therapy reduced the cerebral blood volume and in-filed recurrence rate. These parameters suggest distinct clinical activity of CXCR4 blocker plus RT in GBM, which could overcome the resistance to RT in this tumor type.

Having a broader mechanism of action and superior pharmacokinetics profile, we believe that NOX-A12 could potentially provide significant clinical benefit in combination with RT to GBM patients. In the preclinical models of GBM, the combination of NOX-A12 plus RT was able to substantially increased the survival of tumor-bearing rats (Exhibit 12).

Exhibit 12: NOX-A12 in combination with RT prolonged the survival in GBM model



Source : Company's presentation

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3 – Potential upsides

We also note that due to the high rate of clinical failures, the standard of treatment in GBM has not substantially changed since 2005. In 2017, bevacizumab (Avastin from Roche), an anti-VEGF antibody, was approved for second-line GBM in combination with lomustine, a chemotherapy agent. Remarkably, bevacizumab plus lomustine did not extend OS, but rather PFS compared to lomustine alone (4.2 months vs 1.5 months, respectively). In our view, this indicate an extremely high interest among regulatory authorities in the novel therapies against GBM, as well as low expectations on the efficacy front to secure regulatory approval.

Thus, we believe that the prospective Phase 1/2 study in GBM could provide a new therapeutic avenue for the development of NOX-A12. While we are encouraged by the prospects of NOX-A12 in this oncologic indication, at this time, we do not include it in our financial valuation due to unfavorable risk profile of the clinical studies in GBM and high competitive pressure. We currently expect the preliminary readout from the prospective Phase 1/2 study in GBM in 4Q19. If successful, it would provide an additional upside to our current estimates.

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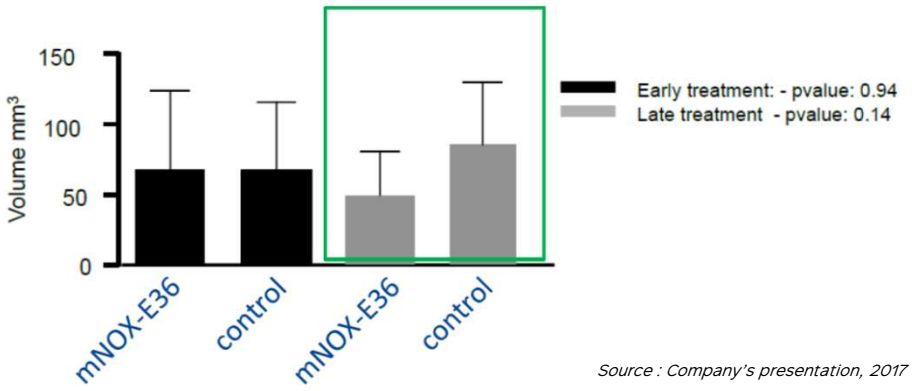
3 – Potential upsides

3.2 NOX-E36 is an attractive asset in oncology

NOXXON has a second clinical program, NOX-E36, that was designed to inhibit the activity of C-C motif ligand 2 (CCL2) chemokine, also known as monocyte chemoattractant protein 1 (MCP-1), as well as several closely related chemokines (CCL8, CCL11 and CCL13). Elevated CCL2 levels in the TME, as well as high circulating concentrations of CCL2, is associated with poor prognosis in oncologic patients. Within the TME, CCL2 can recruit the immunosuppressive cells, such as tumor-associated macrophages (TAM). Accumulation of TAMs in TME could drive tumor cell proliferation, angiogenesis, metastasis, immunosuppression, and drug resistance. Thus, the high level of TAMs is linked to a poor prognosis in some types of cancer, including pancreatic, breast, ovarian and lung. NOX-E36 neutralizes CCL2, which could potentially eliminate the recruitment of TAMs to the TME. In the preclinical model of pancreatic cancer, NOX-E36 was able to prevent the recruitment of TAMs, increase the infiltration of cytotoxic T cells and reduce the tumor size.

Exhibit 13: NOX-E36 reduced the tumor volume in the preclinical model



Source : Company's presentation, 2017

Additionally, NOX-E36 was previously evaluated in the Phase 1/2 study in diabetic nephropathy. Due to the pro-inflammatory functions of CCL2, NOX-E36 was initially developed for this inflammatory kidney damaging disease. The Phase 1/2 study of NOX-E36 showed that the drug was well tolerated and decreased the number of monocytes in the peripheral blood, as well as the expression of CCR2 by the monocytes. These results suggest that NOX-A12 could potentially block CCL2/CCR2 axis.

We note that Pfizer was also developing the blocker of CCL2/CCR2 pathway through targeting CCL2's receptor, CCR2. The Phase 1b study of Pfizer's CCR2 inhibitor (PF-04136309) in combination with chemotherapy regimen (FLORINOX) in patients with advanced pancreatic cancer showed 49% ORR (16 out 33 patients) compared to 0% ORR (0 out of 5) for FOLFIRINOX alone. Albeit Pfizer has discontinued PF-04136309 program, we believe that these results could support the potential of NOX-E36 in oncologic indications.

Moreover, NOXXON's unique approach to target CCL2 chemokine itself (as well as CCL8, CCL11 and CCL13) rather than the individual receptors could potentially result in a broader mechanism of action and a superior efficacy. CCL2, CCL7, CCL8 and CCL13 chemokines were shown to be involved in the innate anti-PD-1 resistance. Thus, the neutralization of CCL2 and the related chemokines (CCL8, CCL11 and CCL13) by NOX-E36 could potentially have more extensive therapeutic action than the CCL2/CCR2 blocker.

Considering the clean safety profile, previous results on CCR2-monocytes and the preclinical data in oncologic models, we believe that NOX-E36 represents an attractive combination asset in oncology. While we are encouraged by the NOX-E36 prospects, we do not include it in our financial valuation due to the early stage of this program in oncologic indication at this time. We expect the company to launch the proof of concept study with NOX-E36 in 2H19.

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4- Valuation

We value the shares of NOXXON Pharma using risk-adjusted net present value (rNPV) analysis of NOX-A12 in treatment of pancreatic and colorectal cancer. To account for uncertainties in the drugs development, we assume a 15% discount rate in our analysis, which we derived using WACC based on (i) a risk-free rate of 0.77%, (ii) an equity risk premium of 5.76% (source: Factset) and (iii) a beta of 2.5, in-line with beta that we use for the similar development-stage companies.

4.1 IP position

The company own a family of a patents that cover NOX-A12 composition of matter. This family of patents begins to expire in 2027. Considering the timing of potential marketing approval, we expect the company to receive a standard 5-year extension post marketing approval. Thus, we project potential revenue stream from NOX-A12 program until 2032.

4.2 Key model assumptions

In our financial model, we assume that NOXXON would seek a commercial partner to develop and NOX-A12 in the US and EU in treatment of pancreatic and colorectal cancer. We also assume that the potential partner will be responsible for all costs, associated with the Phase 3 clinical studies and the filling with the regulatory agencies, as well as all manufacturing, sales and marketing costs. Therefore, we project the company to receive milestone payments and 15% royalty revenues on NOX-A12's sales in these indications. To be conservative in our estimates, we are risk-adjusting the expected milestone and royalty revenue stream based on the respective development phase and the typical probability of reaching the market (13% for the Phase 2 studies in oncologic indications, Exhibit 14).

Exhibit 14: Probability of Market Launch

Oncologic indications	
Phase I	11%
Phase II	13%
Phase III	49%
Filed	85%
Marketed	100%

Source : Chi Heemwong and Kien Wei Siah, Biostat., 2018

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4- Valuation

We project NOXXON's lead program, NOX-A12, to generate risk-adjusted revenues of approximately €24M in 2023, growing to €235M in 2029. We expect NOX-A12 to reach the market for pancreatic cancer in the US and the EU in 2023 and generate risk-adjusted revenues of €118M by 2029. Additionally, we project launch of NOX-A12 in colorectal cancer in 2H23, generating risk-adjusted revenues of €118M by 2029. Potential upside to our estimates include: 1) earlier-than-expected market approvals; 2) additional indications for NOX-A12; 3) market approvals in the territories outside of the US and the EU; 4) higher-than-expected product price or market uptake; and 5) launch of additional future products which we have not yet included in our projections.

Exhibit 15: Risk-Adjusted Pipeline Product Revenues, 2023e-2032e

PaC (EU)	2023	2024	2025	2026	2027	2028	2029	2030	2031	2032
Incidence of end-stage PaC ('000)	70	70	70	70	70	70	70	70	70	70
NOX-A12 market share	1%	5%	9%	12%	15%	15%	15%	14%	13%	12%
Price, €	50,000	50,000	50,000	50,000	50,000	50,000	50,000	50,000	50,000	50,000
Sales EU (€M)	35	174	313	418	523	524	524	490	455	420

PaC (US)	2023	2024	2025	2026	2027	2028	2029	2030	2031	2032
Incidence of end-stage PaC ('000)	45	45	45	45	45	45	45	45	45	45
NOX-A12 market share	3%	7%	9%	12%	15%	15%	15%	13%	12%	10%
Price, $	65,000	65,000	65,000	65,000	65,000	65,000	65,000	65,000	65,000	65,000
Sales US (€M)	**76**	**178**	**229**	**305**	**382**	**382**	**382**	**332**	**306**	**256**
TOTAL PaC (€M)	**111**	**352**	**542**	**724**	**905**	**906**	**907**	**821**	**761**	**676**
Risk-adjusted Sales in PaC (€M)	14	46	70	94	118	118	118	107	99	88

CRC (EU)	2023	2024	2025	2026	2027	2028	2029	2030	2031	2032
Incidence of end-stage CRC ('000)	82	83	84	84	85	86	86	87	88	88
NOX-A12 market share	1%	3%	5%	7%	10%	10%	10%	9%	8%	7%
Price, €	50,000	50,000	50,000	50,000	50,000	50,000	50,000	50,000	50,000	50,000
Sales EU (€M)	**41**	**124**	**209**	**295**	**425**	**428**	**432**	**392**	**351**	**310**

CRC (US)	2023	2024	2025	2026	2027	2028	2029	2030	2031	2032
Incidence of end-stage CRC ('000)	53	53	54	54	54	55	55	56	56	57
NOX-A12 market share	1%	5%	9%	13%	15%	15%	15%	13%	11%	10%
Price, $	65,000	65,000	65,000	65,000	65,000	65,000	65,000	65,000	65,000	65,000
Sales US (€M)	**30**	**151**	**275**	**400**	**465**	**469**	**473**	**413**	**352**	**323**
TOTAL CRC (€M)	**71**	**276**	**484**	**695**	**890**	**897**	**904**	**805**	**703**	**632**
Risk-adjusted Sales in CRC (€M)	9	36	63	90	116	117	118	105	91	82

Source : Invest Securities estimates

Source: Invest Securities estimates

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4- Valuation

4.3 rNPV analysis

Considering the development stage of the NOX-A12 program and the achieved clinical results, we expect the company to sign an agreement with the prospective commercial partner in 2020, leading to a potential upfront payment of €15M, followed by regulatory and milestone payments of up to €181M (Exhibit 13). In our rNPV model, we include the general and administrative costs attributed to the support of NOX-A12 programs and R&D costs attributed to the development of NOX-A12 until the Phase 3. Assuming a 15.0% discount rate, we arrive at an rNPV of €106M for NOXXON's NOX-A12. To this we add the €12.4M in cash and cash equivalents, which we derived from the cash and cash equivalents held by NOXXON at the end of 1H18 of €0.8M, the subsequent equity raise €6.2M and the potential exercise of the outstanding warrants at the strike price of €1.4 per share, resulting in additional €5.4M. As a result, we arrive at price target of €4.9 per share (Exhibit 16).

Exhibit 16: Risk-Adjusted NPV Analysis

In €M	2H18	2019	2020	2021	2022	2023	2024	2025	2026	2027	2028	2029	2030	2031	2032
Upfront / Milestone Payments		**0**	**15**		**20**	**50**		**96**							
Royalties				**0**	**0**	**27**	**94**	**154**	**213**	**269**	**270**	**272**	**244**	**220**	**196**
Royalties for Spiegelmer technology				*0*	*0*	*-1*	*-2*	*-3*	*-4*	*-5*	*-5*	*-5*	*-5*	*-4*	*-4*
R&D	-1.2	-3.3	-5.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
SG&A	-1	-2	-3	-3	-3	-3	-3	-3	-3	-3	-3	-3	-3	-3	-3
EBIT	**-2**	**-6**	**7**	**-3**	**17**	**74**	**90**	**244**	**206**	**261**	**262**	**263**	**236**	**213**	**190**
Tax	0	0	-1	0	-2	-10	-13	-34	-29	-37	-37	-37	-33	-30	-27
Net	**-2**	**-6**	**6**	**-3**	**15**	**64**	**77**	**210**	**177**	**225**	**226**	**227**	**203**	**183**	**163**
Discount rate	0.93	0.81	0.70	0.61	0.53	0.46	0.40	0.35	0.30	0.26	0.23	0.20	0.17	0.15	0.13
Discounted FCF	**-2**	**-5**	**4**	**-2**	**8**	**29**	**31**	**73**	**53**	**59**	**51**	**45**	**35**	**27**	**21**

WACC	15%
POS	13%
Terminal value	34
Discounted terminal value	4
Sum of DCF	56.1
Net debt (*pro forma* cash + projected raise)	12.4
Fully diluted number of shares	13.9
Price per share	4.9

Source: Invest Securities estimates

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4- Valuation

4.4 Financials

Revenues. NOXXON reported 1H18 financial results on October 11, 2018, and reported no revenues and operating income of €0.08M for the period due the sales of assets held for sale. According to our projections, we expect the company to report no revenues in 2H18. We currently project NOXXON to receive a potential upfront payment of €15M in 2020, when we expect the company to sign a partnership agreement. Overall, we project the company's royalty revenues to grow to €272M in by 2029.

Cash. NOXXON reported cash and cash equivalents of €0.8M and financial liabilities of €2.8M the end of 1H18. In November, the company secured additional €6.2M through the equity capital raise, and we believe that the resulting *pro forma* cash and cash equivalents of €7M is sufficient to maintain company's operations until 4Q19. We also note that all remaining convertible debt obligations were paid off in the form of NOXXON's shares during 2H18.

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SWOT ANALYSIS

STRENGTHS

- First-in-class mechanism of action
- Mid-stage clinical programs
- Secured financing through equity line

WEAKNESSES

- Competitve market
- More advanced competitor
- Potential dilution

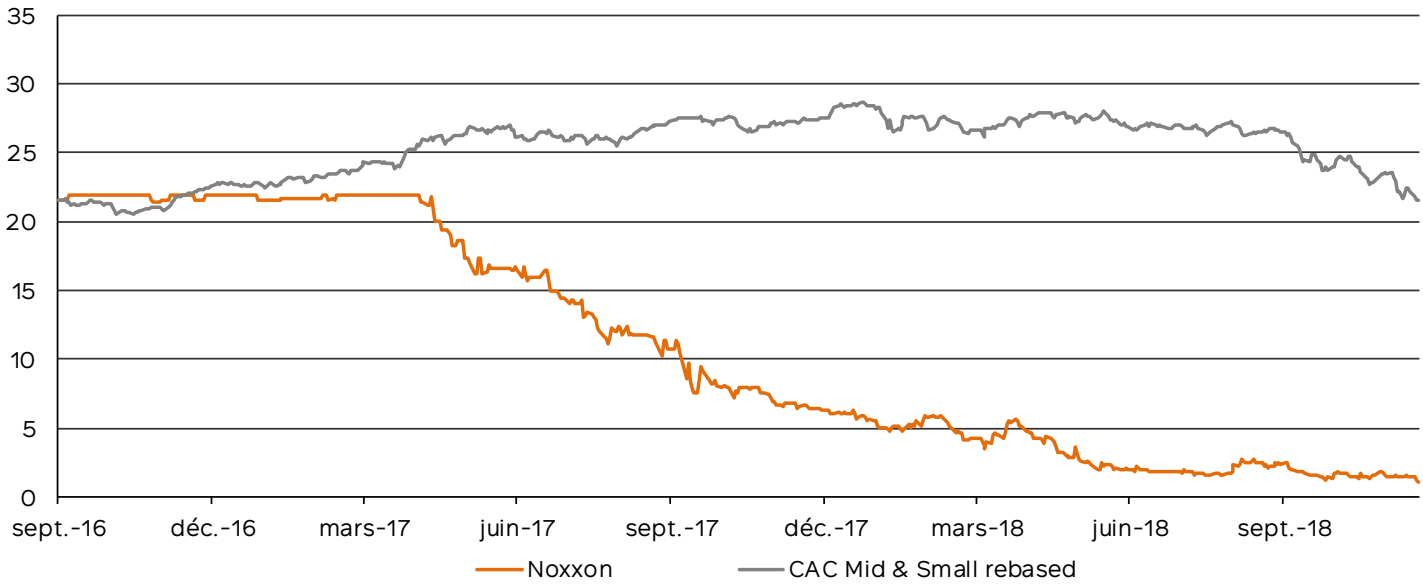
OPPORTUNITIES

- Commercialization agreement
- Earlier-than-expected market approvals
- Marketing in other territories

THREATS

- Clinical and regulatory risks
- Commercial risks
- Legal risks

SHARE PRICE CHANGE FOR 5 YEARS



DETECTION OF CONFLICTS OF INTEREST

	Corporate Finance	Treasury stocks holding	Prior communication to company	Analyst's personal interest	Liquidity contract	Listing Sponsor	Research Contract
Noxxon	No	No	Yes	No	Yes	No	Yes

DISCLAIMER

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