Biotechnology

MATN - OTCQX January 27, 2017

Closing Price 01/26/2017	**$0.39**
Rating:	Buy
12-Month Target Price:	$2.00
52-Week Range:	$0.27 - $1.02
Market Cap (M):	10
Shares O/S (M):	26.5
Float:	99.6%
Avg. Daily Volume (000):	163
Dividend:	$0.00
Dividend Yield:	0.00%
Risk Profile:	Speculative
Fiscal Year End:	December

Total Revenues ('000)

	2016E	2017E	2018E
1Q	0A	0	0
2Q	0A	0	0
3Q	0A	0	0
4Q	0	0	0
FY	0	0	0

Total Expenses ('000)

	2016E	2017E	2018E
1Q	3,352A	3,718	3,831
2Q	3,670A	3,880	3,997
3Q	3,262A	4,203	4,330
4Q	3,850	4,365	4,497
FY	14,134	16,165	16,655

GAAP EPS

	2016E	2017E	2018E
1Q	(0.13)A	(0.14)	(0.10)
2Q	(0.14)A	(0.11)	(0.10)
3Q	(0.12)A	(0.12)	(0.11)
4Q	(0.14)	(0.13)	(0.11)
FY	(0.53)	(0.50)	(0.42)

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Mateon Therapeutics inc

Buy

CA4P - Killing Tumors From The Inside Out; Initiating Coverage with a Buy Rating

Summary

- **Mateon's small-molecule combretastatin A-4 phosphate (CA4P) selectively induces the blockage of blood vessels that feed tumors, inducing tumor death from the inside out. Combine CA4P & Avastin, which blocks new blood vessel growth and Mateon may have a one-two punch that destroys solid tumors.**
- **What drives biotech is data; Mateon has conducted 15 early-stage clinical trials with CA4P in multiple indications, amassing many pieces of data but without definitive proof of concept as a result, valuation has been suppressed.**
- **However, the road forward has been paved as a result of a combination of having the right management in place, the knowledge from 15 prior studies, and positive data (P-value in PFS) from a recent P2 study evaluating CA4P in combination with Avastin in recurrent ovarian cancer (rOC). A P2/3 study (N=436) combining CA4P, Avastin, and SOC chemo is underway and if successful could translate into proof of concept (POC) and, as such, could be an inflection point for the stock (P2 data 1H17, top-line phase 3 data 2020).**
- **Conclusion. The anti-angiogenic (AA) market generates over $15B annually (Avastin=$6B). Recall that Avastin is approved not just for ovarian cancer, but also glioblastoma, lung cancer, and other indications. Thus, if a CA4P combination with Avastin is successful, even just slightly (beginning with OC), there is upside at the current valuation given the market size, in our view. Accordingly, we initiate coverage with a Buy rating and $2 price target.**

Details

Combretastatins: Cutting off the tumor lifeline. Solid tumors of any type need a blood supply to survive. Blockbuster drugs like Avastin and other billion dollar anti-angiogenic agents (AAs; Afinitor, Sutent, and Nexavar) "prevent" the tumor from inducing the formation of new blood vessels (angiogenesis) and thus only are effective at the tumor rim. AAs cannot destroy existing blood vessels that feed the tumor core. Combretastatins, including CA4P, bind to "pathogenic" tubulin in the endothelial cells that line the blood vessels supplying tumors (not healthy blood vessels) causing them to swell and block blood flow. As blood can no longer get through and feed the tumor, the tumor dies. Upon injection the blood vessel is cutoff within minutes, followed by the necrosis of the tumor core.

CA4P synergy with Avastin. Multiple studies have found that while CA4P kills the tumor core, it does not prevent the outer rim of the tumor from sending out signals to induce angiogenesis—but Avastin does. In a P2 study (N=107) in recurrent ovarian cancer (rOC), a combination of CA4P and Avastin induced a statistically significant increase in progression free survival (PFS), 7.3 months vs. 4.8 months for Avastin alone (P<0.05, HR=0.685), as well as a trend in overall survival improvement (25.2 months vs. 22 months). In patients with platinum resistant disease (prOC, N=27) the difference in PFS widened to 6.7 months vs. 3.4 months (P<0.01, HR= 0.57). In platinum sensitive patients only (N=80) the difference was clinically meaningful, though not statistically significant, 7.6 months, vs. 6.1 months. Combined, the greatest effect of CA4P combined with Avastin appears to be prOC, as well as in patients with more advanced (i.e. measurable) diseases. In addition, this is the first published study in rOC to show beneficial effects of an AA combination. A P2/3 study (N=436) in prOC is underway. Data from the first 80 patients (P2, 1H17) will be used for safety, a look at early efficacy and powering assumptions for the phase III (N=356, data 2020).

Valuation. We assume CA4P launches in the U.S. in 2021 and 2022 in Europe for ovarian cancer, followed by additional indications in 2023. A 30% discount rate is applied to the free cash flow, discounted EPS, and sum-of-the-parts models which are equally weighted to derive a $2 price target.

CORPORATE PROFILE

Mateon Therapeutics Inc.,
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Investment Risk:
Mateon is a development-stage company with no approved products.

Regulatory Risk:
The company's products may not be successful in current and future clinical trials.

Commercial Risk:
There can be no assurances that the company's products will achieve market share and generate significant revenues.

Financial Risk:
The company is not yet profitable and may need to raise additional capital to continue funding operations.

Ownership:
Institutional: 8.5%
Insiders: 1%

Balance Sheet Summary:
Cash: $16.3M (3Q16)
Debt: $0M

Analysts covering the company (other than Maxim): 1

Company Background. Mateon is a biopharmaceutical company focused on developing vascular disrupting agents (VDAs) for the treatment of cancer. Vascular disrupting agents selectively target the vasculature of tumors and obstruct the tumor blood supply without disrupting the blood supply to normal tissues. The obstruction of tumor blood supply with VDA's has been shown to induce significant central tumor necrosis. VDA's belong to a larger class of oncology drugs called vascular-targeted therapies (VTTS). VTTs include anti-angiogenic agents (AAs). AAs, including Avastin, generate over $15B in annual revenues. Mateon is leveraging a combination approach of VDAs and AAs that can eliminate tumor blood supply using a combination approach—indirect inhibition of angiogenesis by anti-angiogenic agents coupled with the direct disruption of established tumor blood supply with a VDA. Lead candidate CA4P is a reversible tubulin binding agent which selectively targets the endothelial cells that line the blood vessel walls in most solid tumors. CA4P can cause endothelial cells to swell and obstruct blood flow to the tumor, ultimately leading to tumor cell death.

Mateon is currently developing two VDAs; combretastatin A-4 phosphate (CA4P; also known as fosbretabulin tromethamine, fosbretabulin, and ZYBRESTAT) and combretastatin A-1 phosphate (OXi4503). A phase 2 study (GOG-0186I, N=107) sponsored by the Gynecologic Oncology Group (GOG), evaluating CA4P plus Avastin (bevacizumab) in recurrent ovarian cancer met its primary endpoint demonstrating a statistically significant increase in progression-free survival (PFS) vs. bevacizumab alone. In addition, the study showed a preliminary trend in over survival (24.6 months in the combination group vs. 22 months in the Avastin-only group). Based on the GOG study, the phase 2/3 FOCUS study (initiated 2Q16) of CA4P in combination with Avastin and chemotherapy (i.e. SOC) in platinum-resistant ovarian cancer is now underway. The phase 2 portion (n=80) aims to demonstrate early efficacy and determine powering assumptions for the phase 3 (additional N=356) which is expected to begin later in 2H17. Mateon is also developing CA4P for neuroendocrine tumors and positive phase 2a data was announced in January 2017. A combination (CA4P + Avastin and chemotherapy) phase 2/3 study in glioblastoma (GBM) is in planning. Mateon's second product, OXi4503, is being evaluated in a phase 1b study for the treatment of acute myeloid leukemia (AML) in combination with azacitidine. Mateon has advanced the study, added more study sites, and expanded to a combination approach with standard of care chemotherapy (cytarabine).

Senior Management:

William D. Schwieterman, M.D., President and Chief Executive Officer. Dr. Schwieterman has been an independent consultant to biotech and pharmaceutical companies, including to Mateon, specializing in clinical development since 2002. Dr. Schwieterman is a board-certified internist and a rheumatologist. Dr. Schwieterman was previously a part-time employee of Perceptive Advisors, LLC. From 2009 to 2014, Dr. Schwieterman was the Chief Medical Officer of Chelsea Therapeutics, Inc., where he led the Chelsea Therapeutics clinical development team toward the approval of droxidopa for the treatment of symptoms of Parkinson's disease and other neurogenerative diseases. Dr. Schwieterman was formerly Chief of the Medicine Branch and Chief of the Immunology and Infectious Disease Branch in the Division of Clinical Trials at the Food and Drug Administration (FDA). Dr. Schwieterman spent 10 years at the FDA in the Center for Biologics overseeing a wide range of clinical development plans for a large number of different types of molecules.

David J. Chaplin, Ph.D., Chief Scientific Officer. Dr. Chaplin is currently Chief Scientific Officer, to Mateon. He previously served as president and CEO, as well as CSO and head of research and development. He has been a leader in the field of vascular targeting and was the original discoverer of the vascular disrupting action of combretastatin (fosbretabulin). Dr. Chaplin has more than 18 years of experience in oncology research. Prior to his association with Mateon, Dr. Chaplin was Vice President, Oncology at Aventis Pharma in Paris. Before the merger of Rhone Poulenc Rorer (RPR) with Hoechst Marion Roussell, Dr. Chaplin was Senior Director of Oncology at RPR from 1998 to 1999. From 1992 to 1998, Dr. Chaplin led the Cancer Research Campaign's (CRC) Tumor Microcirculation Group, based at the Gray Laboratory Cancer Research Trust, Mount Vernon Hospital, London.

INVESTMENT SUMMARY

The bull case. Mateon's combretastatins cut off the blood supply feeding the insides of solid tumors. Avastin prevents the rim of the tumor from forming new blood supplies—together, the effects could be synergistic. The company's lead combretastatin, CA4P, has a long history of early-stage trials in multiple indications that have shown early signs of efficacy but without proof of concept data and a clear path forward. Thus, valuation has been penalized and remains suppressed at sub-$20M. The recently published phase 2 study of CA4P combined with Avastin in ovarian cancer demonstrated statistically significant improvement in the primary endpoint of progression-free survival as well as a positive trend in overall survival. Combined with the right management team, Mateon is now ready to drive CA4P to proof of concept, in our view. A phase 2/3 study (CA4P + Avastin vs. Avastin alone) in ovarian cancer is underway, with interim data (phase II part) expected in 1H17—if positive, this could represent an inflection point for the stock and set the stage for the phase 3 study (data 2020). Mateon is also planning a phase 2/3 study for CA4P in glioblastoma, another Avastin-approved indication. In addition, a second combretastatin, OXi4503, is expected to have phase 1b data in AML later in 2016 as well. As such, bulls see multiple short-term catalysts (data) that should drive valuation. From a value proposition point of view, the anti-angiogeneic market is valued at over $15B, with Avastin leading the way with $6B. If successful, and CA4P is approved for use with Avastin, any success (market penetration) with Avastin-like pricing ($100K+) would suggest upside to the current valuation, in our view.

The bear case. Tubulin disrupting compounds like CA4P have been abandoned in later stage studies by pharma for either having only modest activity, no meaningful benefit, or safety concerns—CA4P touches upon all three of these. CA4P has been through 15 early-stage clinical studies targeting multiple indications and has yet to achieve a clear path forward to more advanced stages of development. While there was a modest benefit in a small phase II study with Avastin in ovarian cancer, advancing to a phase 2/3 and introducing a third variable, chemotherapy, suggests again that there may be no clear path forward. The beneficial most effects of CA4P with Avastin were only in a small subset of patients with platinum resistant disease, which though statistically significant, may not be repeated in the larger study. The prOC market is niche, with only 4,000 patients in the U.S. and 6,000 in Europe, limiting commercial opportunity. In addition, key patents for "composition of matter" and "methods of use" are approaching expiration in 2021, leaving the company dependent on only orphan designation granting seven years of marketing exclusivity from the date of approval. Thus, a potential commercial opportunity, if CA4P were successful, could be significantly limited and would likely turn away potential pharma partners.

Our take. Solid tumors of any kind all have a common link: blood supply. Targeting blood supply with anti-angiogenic agents (AAs) like Avastin is a $15B market. However, AAs like Avastin can only prevent new blood vessel formation to the tumors and not destroy existing blood vessels—Mateon's combretastatins, like lead candidate CA4P, can. Thus, a combination approach with Avastin is an attractive one-two punch that could destroy solid tumors and, given the size of the market, could attract a big pharma partner, in our view. The question then is why is Mateon's valuation suppressed (sub-$20M). We believe part of the answer is a history of early-stage studies of combretastatins that have shown signs of efficacy in multiple indications but not definitive proof of concept data. In our opinion, Mateon now has the right management team in place with a strong strategic focus and a path forward, with the recently announced phase 2 data in ovarian cancer (CA4P + Avastin) demonstrating a statistically significant improvement in the primary endpoint of progression-free survival (PFS). In addition, there was a trend in overall survival benefit from the combination, a first for combinations of vascular-targeting agents in ovarian cancer. The company has now initiated a phase 2/3 study (N=436). Interim data from the phase II portion is expected by 2H17 and, if positive, could represent proof of concept, creating an inflection point in valuation and setting the stage for the phase 3 portion of the study (data 2020). Furthermore, Mateon presented positive phase 1b data at ASH 2016 for a second combretastatin candidate (OXi4503 in AML). The company also is planning for a phase 2/3 study in GBM (another Avastin-approved indication). With multiple data points coming, including potential POC data in ovarian cancer, we see upside to the current sub-$20M valuation.

Mateon financials. Mateon began 4Q16 with $16.3M in cash. With operating expenses over the next 12 months expected to be $16M-$19M, the company is funded into the 2H17. Multiple interim analyses expected over the next 12 months, including the phase II data from the FOCUS study, which, if positive, could represent POC and potentially attract a pharma partner (potential upfront payments and milestones), could extend the cash runway beyond 2017.

Exhibit 1. Mateon Product Pipeline

Product	Indication	Development	Pre-Clinical	Phase I	Phase II	Phase III	Marketed
Combretastatin A-4 Phosphate (CA4P)	Platinum resistant ovarian cancer (prOC)						
Combretastatin A-4 Phosphate (CA4P)	Recurrent ovarian cancer (rOC)						
Combretastatin A-4 Phosphate (CA4P)	Glioblastoma multiforme (GBM)						
Combretastatin A-4 Phosphate (CA4P)	Neuroendorine tumors (NETs)						
Combretastatin A-1 (OXi4503)	Acute Myeloid Leukemia (AML)						

Source: Company Reports and Maxim

Exhibit 2. Upcoming Catalysts

Product	Geography	Indication	Event	Timeline	Impact
Combretastatin A-4 Phosphate (CA4P)	U.S.	Recurrent ovarian cancer (rOC)	Gynecologic Oncology Group (GOG) phase II data publication	Complete	+
Combretastatin A-4 Phosphate (CA4P)	U.S.	Platinum-resistant ovarian cancer (prOC)	Phase II/III FOCUS study initiation; phase II portion N=80	Complete	+
Combretastatin A-4 Phosphate (CA4P)	U.S.	Recurrent ovarian cancer (rOC)	Inititate phase I/II study in combination with pazooanib	Complete	+
Combretastatin A-4 Phosphate (CA4P)	U.S.	Neuroendocrine tumors (NET)	Phase IIa data	Complete	+
Combretastatin A-4 Phosphate (CA4P)	U.S.	Platinum-resistant ovarian cancer (prOC)	Phase II/III FOCUS: interim phase II data	1H17	
Combretastatin A-1 (OXi4503)	U.S.	Acute myeloid leukemia (AML)	Phase Ib data	1H17	++
Combretastatin A-4 Phosphate (CA4P)	U.S.	Gliobastoma multiforme (GBM)	Initiate phase I/II study	1H17	+
Combretastatin A-4 Phosphate (CA4P)	U.S.	Platinum-resistant ovarian cancer (prOC)	Phase II/III FOCUS: Phase II data, expand to phase III N=356	2H17	++
Combretastatin A-1 (OXi4503)	U.S.	Acute myeloid leukemia (AML)	Initiate phase II/III study, combination study	2018	++
Combretastatin A-4 Phosphate (CA4P)	U.S.	Platinum-resistant ovarian cancer (prOC)	Phase III top-line data	2020	+++

Stock Significance Scale: + of moderate importance; ++ higher level; +++ very important

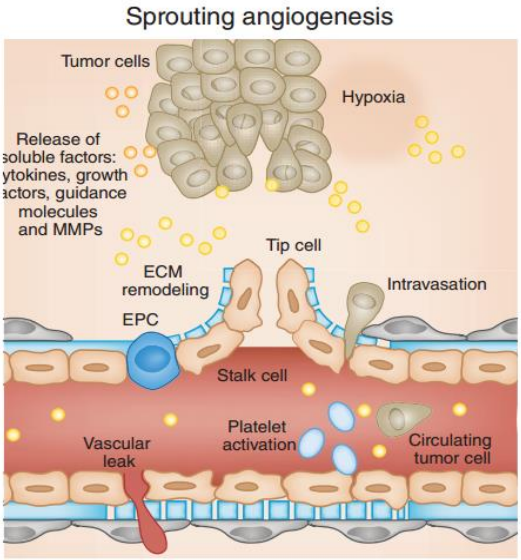
Source: Company reports and Maxim Forecasts

VASCULAR TARGETING THERAPY (VTT)

Tumor growth and blood supply. It has become generally well-accepted in the oncology space that most tumors lay dormant and fail to grow beyond just a millimeter or two in the absence of a tumor-specific blood supply. The process of new blood vessel formation, or angiogenesis, is essential for tumor growth and metastasis. While mutations and genomic aberrations accumulate inside tumor cells that drive uncontrollable cell growth and cell survival (i.e. mutations that prevent apoptosis, or programmed cell death), the continued growth of the tumor, no matter what mutations it has or doesn't have, is dependent on a nutrient supply from a network of microvessels. The vessels that "feed" a tumor may arise via angiogenesis (new blood vessels form from preexisting vessels), vasculogenesis (formation of new vessels from precursor cells called angioblasts), vessel intussusception (splitting of a blood vessel in two, aka "splitting angiogenesis"), and vascular mimicry (tumor cells line a channel mimicking a blood vessel), or any combination of these. Neovascularization, or the formation of new blood vessels, is atypical for healthy tissues. The tumor microvasculature is disorganized and chaotic, which the morphology of the vessels and their functionality distinct from normal blood vessels.

The tumor microenvironment is the driver of angiogenesis. Once a tumor grows beyond a few millimeters it becomes starved for nutrients to support its continued growth. In addition, the lack of blood supply induces hypoxia in the microenvironment and the buildup of free radicals. These changes in the tumor microenvironment trigger the flipping of multiple molecular switches. Tumors secrete growth factors and cytokines in a concentration gradient that triggers quiescent endothelial cells to start the angiogenesis process. The gradient of growth factors, cytokines, and chemokines causes the "sprouting" of new vessels to grow towards the tumor. For example, vascular endothelial growth factor (VEGF) stimulates endothelial cell growth, survival, and proliferation. VEGF and other growth factors regulate the growth and differentiation of multiple components of the vascular system, especially blood and lymph vessels. VEGF is overexpressed and contributes to the establishing of new vasculature (blood vessels) for tumors. VEGF also helps tumors to grow and survive, and is the target of Avastin (bevacizumab) which is used to "prevent" the formation of new blood vessels that supply the tumor. The tumor vasculature network is constantly changing and is poorly formed, thus it leaks. Leaking vessels trigger repair pathways and inflammatory responses to come fix the leaks. The tumor microvasculature is constantly being remodeled and changed as the tumor grows.

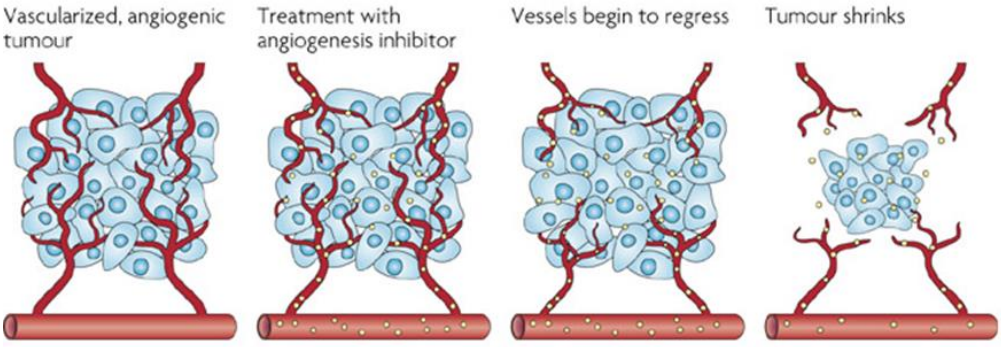
Exhibit 3. Angiogenesis. The tumor microenvironment is deprived of a blood supply, limiting both nutrients for tumor growth and oxygen (hypoxia). The tumor responds by secreting cytokines, growth factors, guidance molecules, and matrix metalloproteases (MMPs). An existing blood vessel then "sprouts" a new shoot that grows towards the tumor. MMPs act to degrade and remodel the basement membrane framework around the newly growing vessels.



Source: Weiss. SM and Cheresh DA., "Tumor Angiogenesis: Molecular Pathways and Therapeutic Targets." Nature Medicine. 2011.

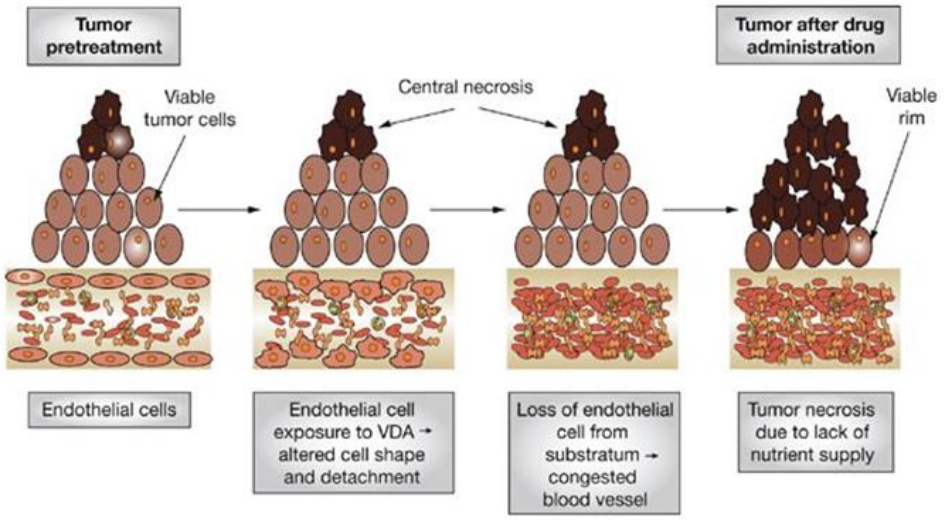
Vascular targeted therapies (VTTs). Targeting tumor blood supply is a rapidly expanding field in oncology. VTTs interfere with blood supply to the tumor, unlike targeted therapies or cytotoxic chemotherapies, that aim to destroy as much tumor as possible while limiting collateral damage to healthy tissue, making VTTs applicable to essentially any tumor. VTTs fall into two categories: anti-angiogeneic therapies (AAs [i.e. Avastin]) and vascular disrupting agents (VDAs). AAs target the factors and molecules that lead to the development of new blood vessels to supply the tumor. Perhaps the most well-known AA, is Avastin (bevacizumab), which targets Vascular Endothelial Growth Factor (VEGF). VDAs on the other hand directly impact and disrupt existing blood vessels, which results in the rapid and selective shutdown of tumor blood supply, leading to ischemic death of the tumor. VDAs fall into two groups, those that target specific ligands and those that take advantage of the distinct morphological differences between healthy vasculature and tumor-related vasculature. Mateon's combretastatins are VDAs that target the "pathogeneic" tubulin of endothelial cells lining the blood vessels of the tumor microvasculature.

Exhibit 4. Anti-angiogenic Agents (AAs) Prevent Blood Vessel Formation. Tumors secrete growth factors, cytokines, and other molecules to induce new blood vesssel formation. AAs disrupt this signaling, resulting in the regression of blood vessels supplying the tumor. Over time the tumor begins to shrink.



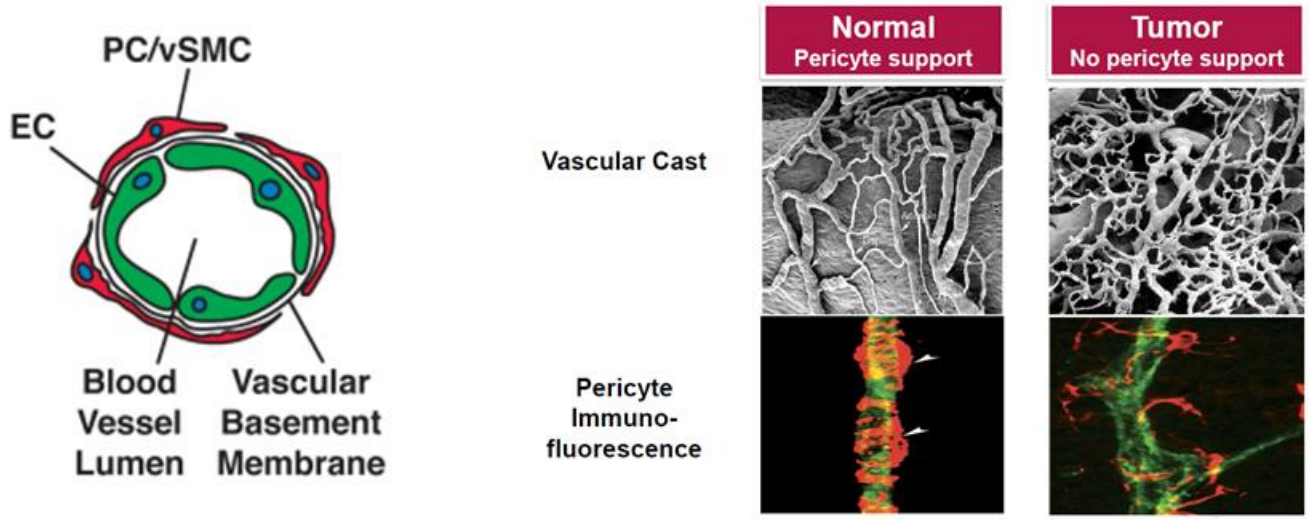
Source: Zetter BR. (2008) "Perspectives" Nature Reviews-Cancer. (8) 647-654.

Exhibit 5. Vascular Disrupting Agents (VDAs). VDAs, as opposed to anti-angiogenic agents, directly damage existing blood vessels that supply tumors. VDAs, like Mateon's combretastatins, can alter the endothelial cells lining the tumor blood vessels, causing them to block the blood vessel and cut off the blood flow. The tumor then dies essentially from the inside out. A viable tumor rim remains that can signal for new blood vessel formation, thus the rational for a combretastatin/Avastin combination (discussed below).



Source: Cooney M et al. (2006). "Drug Insight: Vascular disrupting agents and angiogenesis- Novel approaches for drug delivery" Nature Clinical Practice Oncology. (3) 682-692.

Exhibit 6. Pericytes: Structural Support of Blood Vessels is Absent in Tumor Blood Vessels. A hallmark of tumor vasculature is the lack of pericyte cell supports, which contributes in part to their "leakiness" and weakness. Pericytes are cells that wrap around the blood capillaries (*peri*=around, *cyte*=cell) and have long appendages that embrace and support the ablumenal (outside, versus luminal cells lining the blood vessel interior) endothelial cells. Pericytes not only provide scaffolding, but also signaling and communication with the endothelial cells. The communication is both chemical and physical, the latter where contractile forces from the pericyte influence the behavior of the endothelial cells around the blood vessels. VDAs disrupt the vascular architecture. **Left:** Pericytes exert contractile forces by wrapping themselves around the abluminal endothelial cells. **Right:** Normal blood vessels have the support of pericytes, giving them a very defined structure. Vessels associated with tumors do not have pericyte support.



Source: Mateon presentation and; Bergers G and Song S (2005) 'The role of pericytes in blood-vessel formation and maintenance" Neuro-Oncology (7) 452-464.

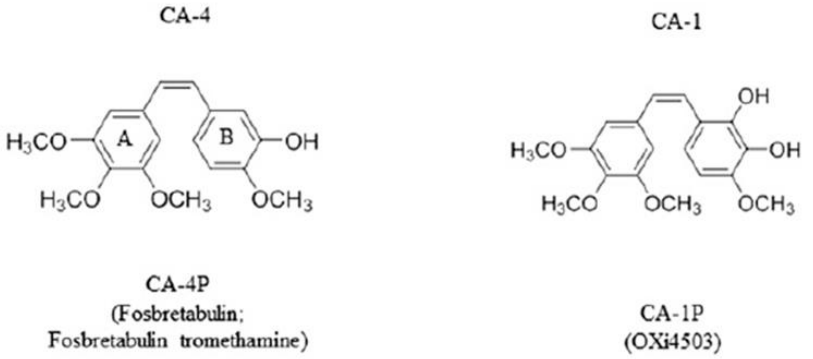
Combretastatins: CA4P, OXi4503

Combretastatins are natural compounds found in the Bushwillow tree (*Combretum caffrum*) which is indigenous to riverbanks of Eastern South Africa. The chemical structure of combretastatins consists of aromatic carbon rings linked by an alkene (double bonded carbon) bridge, allowing them to be easily altered chemically. These compounds are naturally antimitotic, meaning they interfere with cell division. Combretastatins are being developed as vascular disrupting agents (VDAs). VDAs target tumor blood vessels and within hours cut off the blood flow resulting in ischemic and/or hemorrhagic death of the tumor. Combretastatins target tubulin, which provides the structural support of tumor blood vessels.

This is in contrast to healthy, normal blood vessels, where additional support comes from intact basement membranes, cytoskeletal support, and pericytes. For these reasons, the combretastatins selectively target the "weaker" tumor blood vessels.

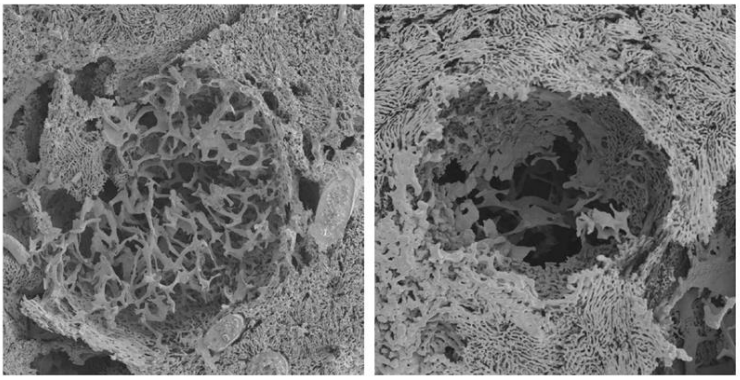
CA4P and OXi4503. Mateon is currently developing two combretastatins, combretastatin A-4 Phosphate (CA4P; fosbretabulin, fosbretabulin tromethamine, and Zybrestat) and combretastatin A-1 (OXi4503). Both compounds have been shown to rapidly induce tumor blood vessel membrane permeability ("leaky"). CA4P is Mateon's lead compound and is being developed for a number of indications including ovarian cancer, glioblastoma, lung cancer, and neuroendocrine tumors. CA4P was found to inhibit tumor blood flow at doses 10x less than the maximum tolerated dose, which prompted clinical development in the late 1990s by investigators, including Dr. David J Chaplin, Mateon's Chief Scientific Officer. OXi4503 is Mateon's second product in development. OXi4503 is being developed for the treatment of acute myeloid leukemia (AML) and has been shown to have a dual mechanism of action; reducing tumor blood flow and forming a potentially anti-proliferative metabolite.

Exhibit 7. CA4P and OXi4503 Chemical Structure. Combretastatins in general have a basic two ring chemical structure connected by an alkene bridge. This structure is easily manipulated chemically and has formed the basis of the development of synthetic combretastatins.



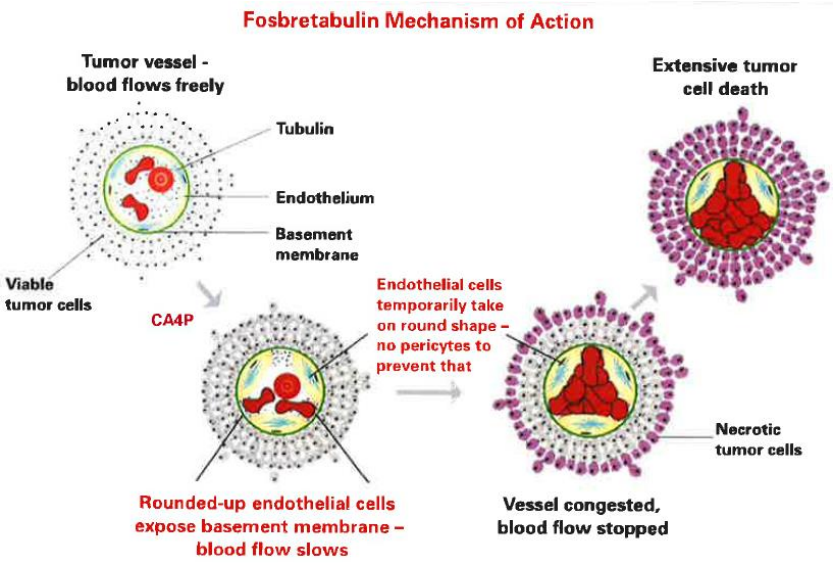
Source: Green LM, Meegan MJ, Zister DM., (2015) "Combretastatins: More than just vascular targeting agents?" The Journal of Pharmacology and Experimental Therapeutics. (355) 212-227.

Exhibit 8. CA4P Eliminates the Tumor Vasculature. CA4P monotherapy rapidly inhibits tubulin polymerization**.**



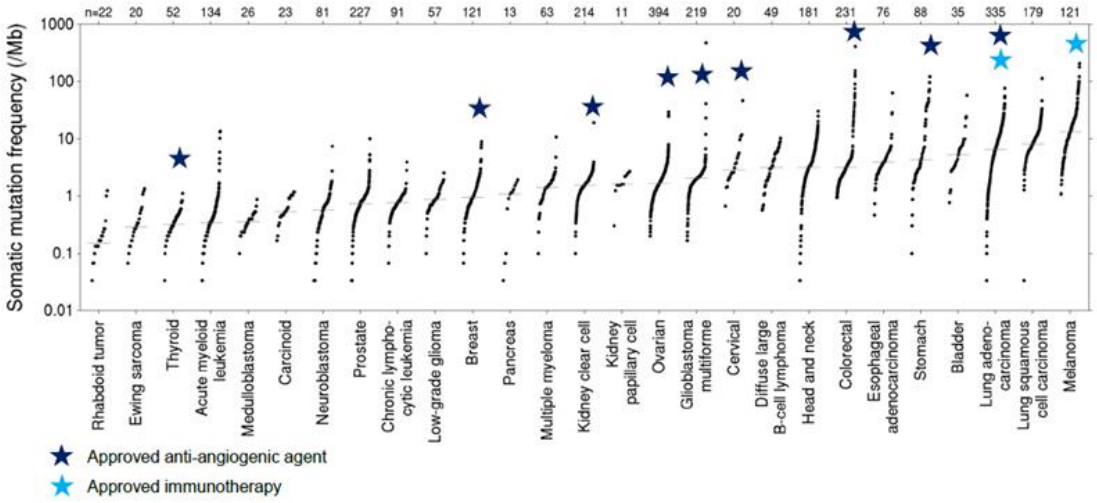
Source: Mateon presentation

Exhibit 9. CA4P (Fosbretabulin) Mechanism of Action



Source: Monk et al., "Randomized Phase II evaluation of bevacizumab versus bevacizumab plus fosbretabulin in recurrent ovarian cancer, tubal or peritoneal carcinoma: An NRG Oncology/Gynecologic Oncology Group Study". Journal of Clinical Oncology. (2016) (34) 1-8.

Exhibit 10. Targeting the Tumor Blood Supply is Applicable to a Broad Range of Tumor Types. Anti-angiogenic drugs have demonstrated efficacy and have been approved across a wide range of solid tumor types. This is in contrast to immune-based therapies (vaccines, CAR-T, MAbs) that are limited to a narrow range of tumors (often just one) as they are dependent on specific mutations and mutation frequency, both of which vary widely between tumor types and even within the same tumor. Targeting the blood supply is independent of mutation frequency and thus applies to most tumor types. Shown below is the mutation frequency per tumor type and approved anti-angiogenics (dark blue stars) and approved immunotherapies (light blue stars).

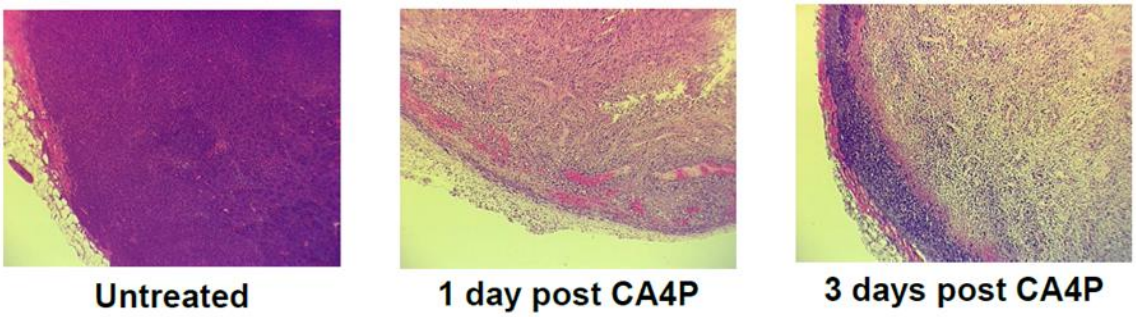


Source: Mateon presentation and Lawrence et al., "Mutational heterogeneity in cancer and the search for new cancer associated genes" Nature. 499 (7457): 214-8.

Rationale for combination therapy. There are significant advantages to targeting a tumors blood supply using vascular disrupting agents (VDAs). For one, there are thousands of tumor cells that may be dependent on one particular microvessel, thus occluding that vessel with CA4P or OXi4503 can effectively kills large quantities of cells. Targeting the blood supply is independent of the tumor type—therefore, any type of tumor can be targeted with a VDA. Also, as CA4P and OXi4503 target tubulin that is itself not mutated, there is little risk for drug-resistance developing. CA4P, for example, has been the subject of multiple clinical studies, mainly as a monotherapy for the treatment of multiple tumor types. Investigators found that as a monotherapy, and this is true of VDAs in general, the killing effect is in the core of the tumor. Meaning, the rim of the tumor remains viable and can stimulate growth of new blood vessels, which would require an anti-angiogenic agent (AA). From an AA perspective, and considering Avastin as an example, AAs are limited to killing the tumor rim but can't get to the core of the tumor as AAs only

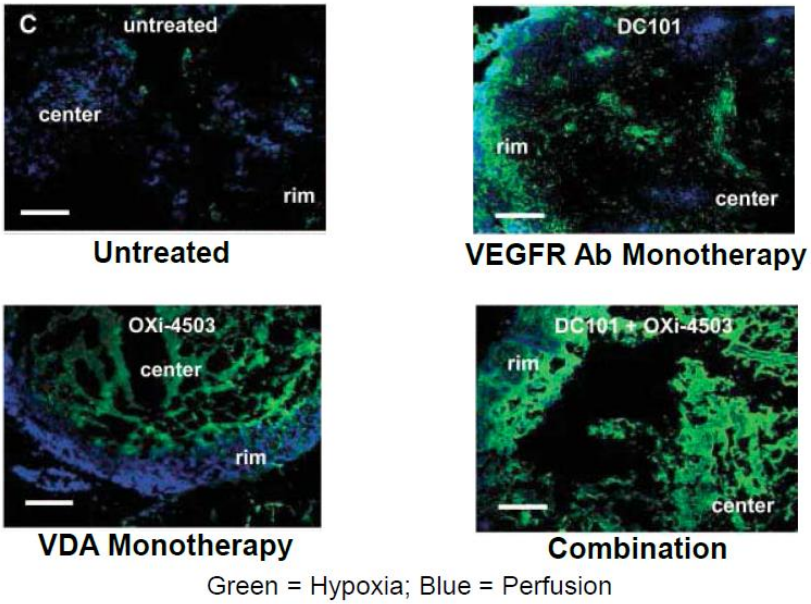
"prevent" new blood vessel formation and don't destroy existing vessels feeding the tumors. Combined, the rationale exists for combining an AA and a VDA. In addition, Mateon has shown CA4P combined with chemotherapy reduces tumor size in platinum resistant ovarian cancer. The phase 2/3 study will evaluate the triple combination of CA4P, Avastin, and chemotherapy vs. Avastin and Chemotherapy.

Exhibit 11. CA4P Monotherapy. CA4P treatment leads to significant central tumor necrosis, though the rim remains viable to stimulate new blood vessel growth.



Source: Mateon presentation

Exhibit 12. Combination Therapy of VDA with AA. The indirect anti-vascular mechanism of anti-angiogenic therapy complements/enhances the direct anti-vascular mechanism of VDAs resulting in a maximized therapy for the treatment of tumors. Hypoxia (green) and perfusion (blue) induced by each therapy, VDA and AA alone and in combination.



Source: Mateon presentation

Exhibit 13. Comparison of Mateon's Combretastatins (VDAs) and Anti-angiogenic Drugs.

	Anti-Angiogenic Drugs	CA4P (Ccombretastatin A-4 phosphate)	OXi4503 (combretastatin A-1 di-phosphate/CA1P
Molecular Characteristics	*Bevacizumab (Avastin), ranibizumab…Both are mAbs. *Sorafenib, sunitinib, pegaptanib, pazopanib, cediranib, axitinib, etc. are small molecule tyrosine kinase inhibitors (TKIs)	Small molecule reversible inhibitor of tubulin polymerization	Small molecule reversible inhibitor of tubulin polymerization. Also forms cytotoxic metabolite (orthoquinone) via oxidation
Target	Tumor rim	Tumor core	*Tumor core *Metabolite targets malignant cells of myeloid lineage
Mechanism	*MABs bind to VEGF, thereby rendering it inactive *TKIs inhibit the VEGF receptor, thereby inhibiting its activation	Rapid and selective binding to tubulin, which destabilizes microtubules, changes the shape of endothelial cells and disrupts the cell junctional protein VE-cadherin	Similar to CA4P, OXi4503 also produces an orthoquinone metabolite that has an anti-proliferative effect on leukemic cells
Biological Effect	Continuously inhibit pro-angiogenic growth factor signaling (e.g., VEGF) to prevent formation and growth of new blood vessels throughout the tumor rim	Occludes and collapses pre-existing abnormal tumor blood vessels that feed tumors	Similar to CA4P, OXi4503 also temporarily mobilizes hematopoietic and leukemic cells from the bone marrow
Rapidity of Effect	Weeks	Hours	Hours
Target Tissue	All angiogenesis	Selective for abnormal vasculature characteristically seen in tumor blood vessels	Similar to CA4P, OXi4503 also makes leukemic cells mobilized from the bone marrow vulnerable to the effects of its orthoquinone metabolite
Plasma Half-life	*MABs remain in circulation for days or weeks *TKI half-lives vary, average range is 4-12 hours	Approximately 4 hours	Approximately 2 hours and OXi4503 metabolite half-life is approximately 20 hours
Side Effects	Chronic hypertension with long-term use; Acute-impairment in wound healing; Hemorrhage, hemoptysis, gastrointestinal perforation, proteinuria, nephrotic syndrome, thromboembolic events, etc.	Transient blood pressure increases; Tumor pain, nausea, hematological adverse events; Overlapping with anti-angiogenics: no cumulative toxicities observed	Transient acute blood pressure increases; Tumor pain, nausea, vomiting, headache, fatigue; Effects on hematopoiesis and white blood cell counts; In AML — similar to solid tumors with more pronounced effects on coagulation and hematopoiesis

Source: Company reports and Maxim Group

CLINICAL DEVELOPMENT

CA4P, combretastatin A-4 phosphate. Lead combretastatin candidate, CA4P, has been the subject of 18 clinical studies and used to treat over 475 patients. A comprehensive list of the clinical studies is shown below. Each of the 18 studies was open label and evaluated CA4P as a monotherapy and in combination with chemotherapy, antibodies, and/or radiotherapy. The studies included patients across a wide range of tumor types and at varying stages of disease. Outcomes of the studies are summarized in the table shown below. When administered as a monotherapy, CA4P (or OXi4503) induces significant central ("core") tumor necrosis and patients experienced different levels of response (stable disease, partial response, etc.). However, further studies found that the while CA4P (or OXi4503, or VDA therapies in general) killed at the tumor core, they left the tumor rim cells intact that were able to induce revascularization of the necrotic tumor. Thus, the combination approach with an anti-angiogenic like Avastin or chemotherapy began to take shape. Mateon has collected data in several studies demonstrating potential synergies between CA4P and Avastin (Phase 2 GOG-0186I study, recurrent ovarian cancer), CA4P and chemotherapy (phase 1/2 UKCTC-207 in platinum resistant ovarian cancer, phase 2/3 FACT study in advanced thyroid cancer), and a triple combination of CA4P, Avastin and chemotherapy (phase 2 FALCON study in advanced lung cancer). A phase 2/3 study of CA4P + Avastin + Chemotherapy in platinum-resistant Ovarian Cancer is now underway with data from the phase 2 portion expected in mid-2017.

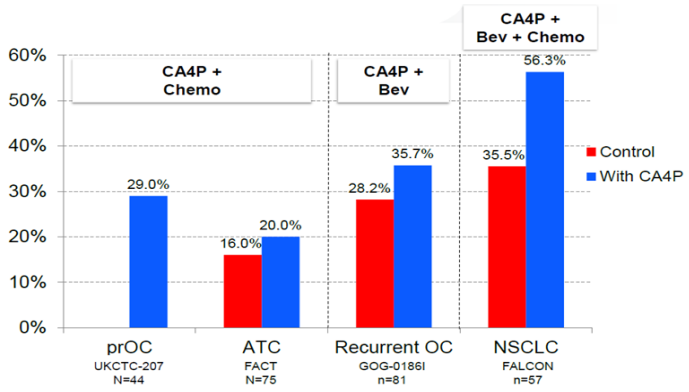
CA4P is also the subject of an ongoing phase 2a study (N=18) as a monotherapy for the treatment of neuroendocrine tumors demonstrated positive data in January 2017. A phase 2/3 study in glioblastoma is also in planning. OXi4503 is currently in an ongoing phase 1b study in combination with chemotherapy for the treatment of acute myeloid leukemia (positive data at ASH 2016 in first two cohorts) with more data expected in 2017. While multiple studies are ongoing or in the planning stages, the company's lead program and focus is prOC, stemming from data in the phase 2 GOG study in rOC.

Exhibit 14. CA4P Clinical Programs. With over 475 patients treated across 18 clinical studies, Mateon has amassed data demonstrating safety and efficacy, particularly in combination with anti-angiogenic agents, as well as chemotherapy.

Study #	N	Completed	Phase	Design	Indication	Results
CA4P-101	25	2001	1	OL, dose escalation	Advanced solid tumors	CR: n=1 (4%); SD: n=11 (44%)
CA4P-102	37	2001	1	OL, dose escalation	Advanced solid tumors	CR: n= 1 (3%); SD: n=16 (43%)
BMS-186527	9	2001	1	OL, dose ranging with carbo	Advanced solid tumors	Terminated
PH1/066	34	2001	1	OL, dose ranging	Advanced solid tumors	SD: n=4 (12%)
CA4P-103	16	2003	1	OL, dose escalation with carbo	Advanced solid tumors	SD: n=4 (25%)
UKR-104	23	2006	1	OL, dose escalation with radiotherapy	Advanced NSCLC, head & neck, or prostate	PR: n=6 (26%); SD: n=5 (22%)
UKCTC-207	46	2005	1b	OL, dose escalation with carbo/pacli	Advanced solid tumors	Overall Response Rate: 10 (22%)
UKCTC-207	44	2008	2	OL with carbo/pacli	prOC	Overall Response Rate: n=13 (29%)
ICC-2302	26	2007	2	OL	ATC	23% 1-year survival; SD: n= 7 (27%)
CA4P-212	13	2007	2	OL, dose ranging with carbo/pacli	Advanced, measurable dx	PR: n=1 (8.3%); SD: n=8 (66.7%)
PH1/092	12	2007	1/2	OL, dose escalating with anti-CEA antibody	Advanced GI carcinoma	SD: n=3 (25%)
CWRU-3302	4	2007	2	OL with doxorubicin, cisplatin, radiotherapy	ATC	Terminated
DKC-203	7	2007	1	OL, dose escalating with cisplatin	Advanced or recurrent cervical cancer	Not analyzed
OXC4P1-105	15	2008	1	OL, dose escalation with bev	Advanced solid tumors	SD: n=9/14 (60%) DCE-MRI positive results
FALCON	63	2010	2	OL, RCT with bev+carbo/pacli	Chemotherapy naive Stage IIIB/IV NSCLC	No difference in PFS (HR 1.04 [NS]) or OS (HR 1.06 [NS]). Improved ORR 56.3% vs 35.5%
FACT	75	2011	2/3	OL, RCT with carbo/pacli	ATC	Improved median OS from 4.0 to 5.2 months (NS)
GOG-0186I	107	2014	2	OL, RCT with bev	Recurrent OC	Improved median PFS from 4.8 to 7.3 months (p=0.049)

Source: Mateon presentation

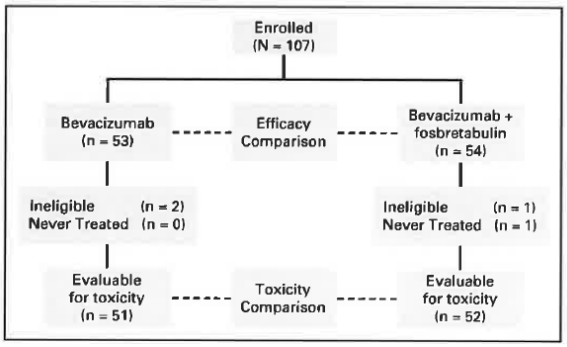
Exhibit 15. CA4B Combinations with Chemotherapy and/or Avastin ("bev," bevacizumab).



Source: Mateon presentation

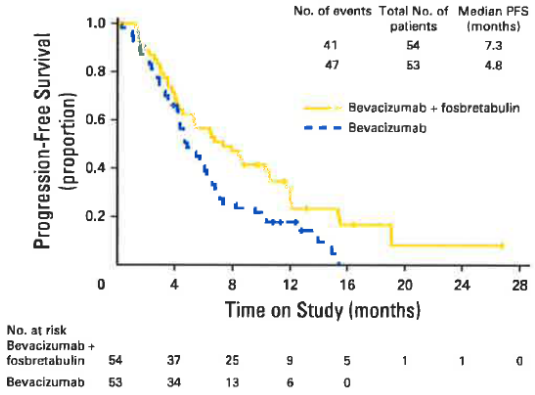
The GOG-0186I Study. The Gynecologic Oncology Group (GOG) of the National Cancer Institute evaluated CA4P combined with Avastin (bevacizumab) in women with second- and third-line recurrent ovarian cancer (rOC). The study (N=107) included patients with both platinum-sensitive (psOC) and platinum-resistant (prOC) disease. Patients were treated with either a combination of CA4P and Avastin (N=53) or Avastin alone (N=54). Avastin was administered at 15 mg/kg intravenously every three weeks. In the combination arm, patients also received CA4P intravenously at 60 mg/m^2 on the same schedule. The primary endpoint of the study was progression-free survival (PFS). The trial resulted in a statistically significant improvement in PFS, P=0.049). The hazard ratio was 0.685 with a 90% two-sided CI of 0.47. The median survival for the combination arm was 7.3 months vs. 4.8 months in the Avastin monotherapy arm. Sub-group analyses demonstrated a greater benefit in patients that had platinum-resistant disease (N=27) where PFS was 6.7 months in the combination arm vs. 3.4 months in the Avastin arm (P=0.01, HR=0.57)—this patient population is more difficult to treat. When the platinum-sensitive population was analyzed alone (N=80) the separation between the combination and Avastin-only arms was not significant, 7.6 months vs. 6.1 months. Overall, the preliminary median overall survival (OS) was longer for patients in the CA4P/Avastin arm versus Avastin alone (24.6 months vs. 22 months, HR=0.85). In addition, it was found that the beneficial effects of combination therapy were greater in patients that had measurable disease than it was for those that did not. Secondary endpoint of objective response rate (ORR) did not reach statistical significance where the ORR for the combination arm was 35.7% (N=42) vs. 28.2% in the Avastin-only arm (N=39). However, in the sub group analysis of 18 evaluable patients with platinum-resistant disease, the ORR in the combination arm (N=10) was 40% vs. 12.5% in the Avastin-only arm (N=8). Hypertension (grade 3), was the most notable adverse event. Long term OS will continue to be monitored.

Exhibit 16. Design of the Phase II Study (GOG-0186I)



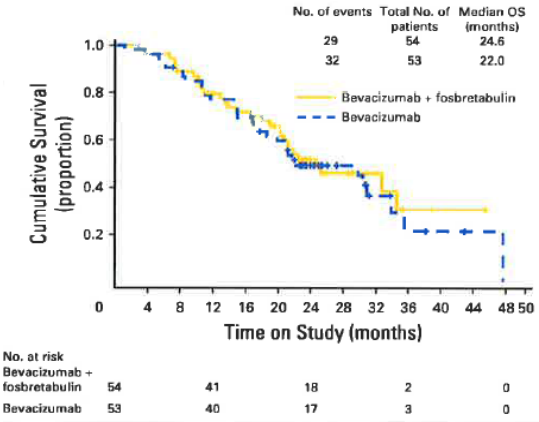
Source: Monk et al., "Randomized Phase II evaluation of bevacizumab versus bevacizumab plus fosbretabulin in recurrent ovarian cancer, tubal or peritoneal carcinoma: An NRG Oncology/Gynecologic Oncology Group Study". Journal of Clinical Oncology. (2016) (34) 1-8.

Exhibit 17. PFS Improvement in CA4P/Avastin vs. Avastin Arms. Patients receiving CA4P and Avastin had a median PFS of 7.3 months vs. 4.8 months in patients only receiving Avastin (P=0.49).



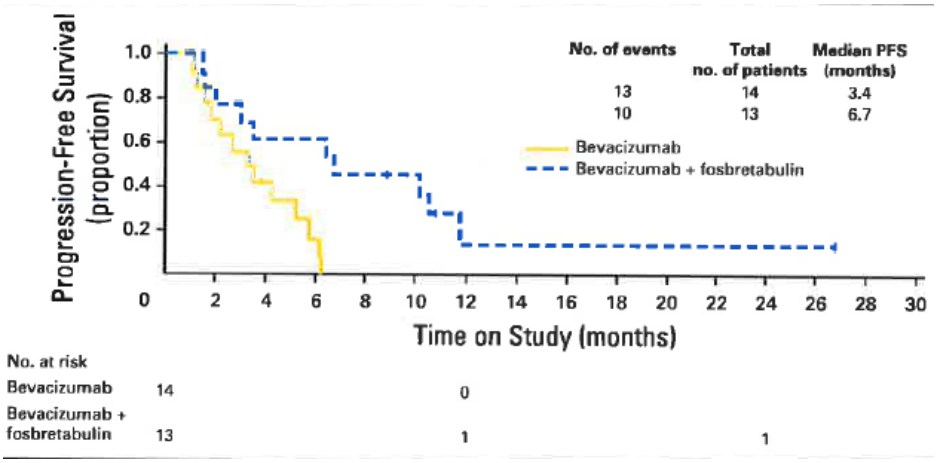
Source: Monk et al., "Randomized Phase II evaluation of bevacizumab versus bevacizumab plus fosbretabulin in recurrent ovarian cancer, tubal or peritoneal carcinoma: An NRG Oncology/Gynecologic Oncology Group Study". Journal of Clinical Oncology. (2016) (34) 1-8.

Exhibit 18. CA4P/Avastin Improvement in Overall Survival. At 61 events and stratifying by prior Avastin, measurable disease and PFI, the HR was 0.85 and the median overall survival in the combination group was 24.6 months vs. 22 months in the Avastin-only group.



Source: Monk et al., "Randomized phase II evaluation of bevacizumab versus bevacizumab plus fosbretabulin in recurrent ovarian cancer, tubal or peritoneal carcinoma: An NRG Oncology/Gynecologic Oncology Group Study." Journal of Clinical Oncology. (2016) (34) 1-8.

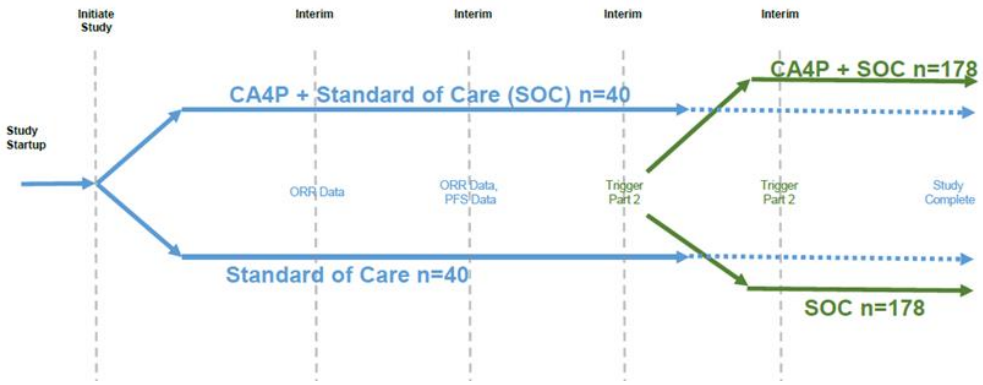
Exhibit 19. CA4P/Avastin is Most Effective in Difficult-to-Treat Patients with Platinum-resistant Disease. In a sub-group analysis of 27 patients, the improvement in PFS widened to 3.3 months between patients with platinum-resistant disease receiving the combination vs. Avastin alone.



Source: Mateon presentation

Phase 2/3 study in platinum-resistant ovarian cancer. Mateon is setting up to initiate a phase 2/3 study (FOCUS) in prOC evaluating the triple combination of CA4P, Avastin, and chemotherapy vs. the combination of Avastin and chemotherapy. The study is designed based on the data from the GOG-0186I in recurrent disease (rOC). Note that the current standard of care for prOC is Avastin and chemotherapy. The phase 2 stage of the study will enroll 80 patients, randomized 1:1, and conduct at least three interim analyses to assess safety, dosing, and efficacy to be used in the 356 patient (additional patients) phase 3 stage. The final interim analysis is expected to trigger the phase 3 stage. The primary endpoint of the study is PFS. Overall survival (OS) and overall response rate (ORR) will also be evaluated as secondary endpoints (and others). The trial initiated in 2Q16. Top-line data is expected in 2020 (phase 2, N=80 data 2H17). If positive, Mateon plans to file for approval and potentially launch CA4P in 2021.

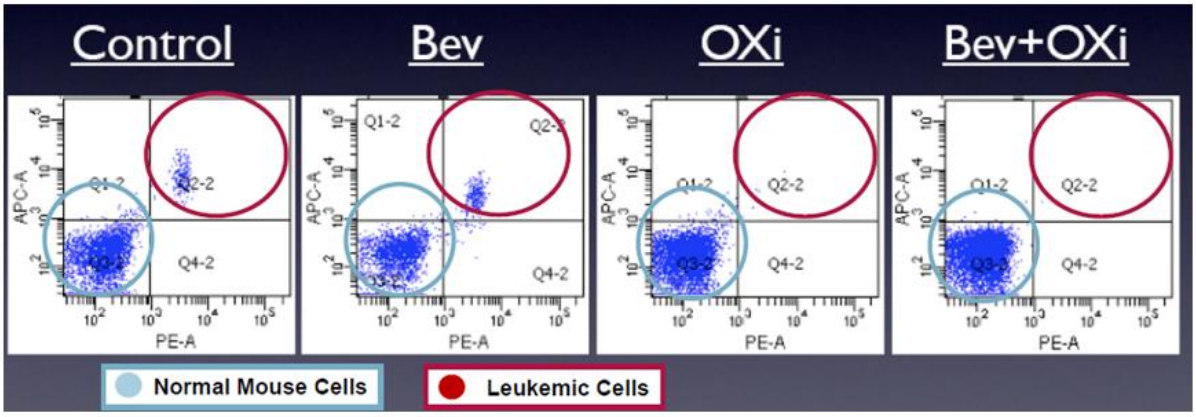
Exhibit 20. Phase II/III Trial Design in prOC



Source: Mateon presentation

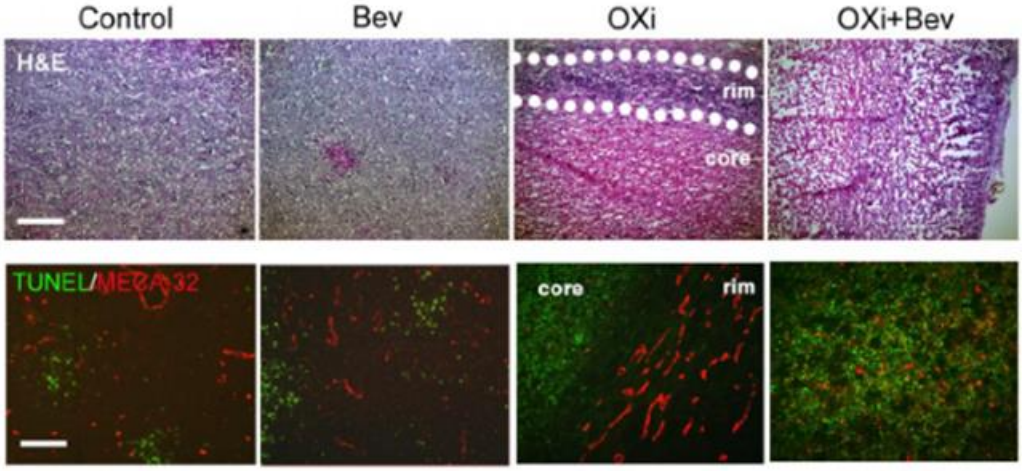
OXi4503, combretastatin A-1 diphosphate. Oxi4503 is the second combretastatin drug candidate. OXi4503 has been shown to not only reduce blood flow to a tumor but also generate anti-proliferative metabolites. The dual MOA makes OXi4503 ideal for specific types of cancer. Mateon has accumulated data in preclinical models demonstrating utility of OXi4503 in liver cancer, melanoma, and leukemia of the myeloid lineage (i.e. AML). OXi4503 has been the subject of an investigator-sponsored phase 1 study in patients with advance AML or MDS (myelodysplastic syndrome). The open-label study was intended to treat 36 patients; through the first 18 patients, as of October 2015, the MTD had not been reached. The trial was closed and taken over by Mateon in December of 2015. In the trial's second stage, OXi4503 was evaluated in combination with cytarbine, the SOC chemotherapy regimen for relapsed/refractory AML or MDS. As presented at ASH 2016, two of 10 patients achieved complete remission which continued at three and six months. In addition, partial responses were observed in two other patients. The third cohort is enrolling patients and more data is expected in 2017.

Exhibit 21. OXi4503 Eliminates Leukemic Cells. In preclinical models of leukemia, OXi4503 alone or in combination with Avastin results in tumor cell death. In leukemic chloromas, a solid tumor formed by leukemic cells, OXi4503 only destroys the tumor core, leaving a viable rim for which Avastin can be used to destroy.



Source: Mateon presentation and Madlambayn et al., (2016) "Leukemia regression by vascular disruption and antiangiogenic therapy." Blood. 116(9): 1539-47

Exhibit 22. OXi4503 + Avastin in Leukemia, Preclinical POC. Shown below are leukemic chloromas, solid tumors made up of leukemia cells. As was shown in other solid tumor types, using a vascular disrupting agent (VDA) kills the tumor core while leaving the rim of the tumor viable to re-vascularize the tumor. By adding Avastin with OXi4503, the entire tumor is destroyed. This is observed clearly in the second row of images. In the third image across, it's clear that OXi4503 results in death of the tumor core (in green) leaving the viable tumor rim intact (in red). In the fourth image, when Avastin and OXi4503 are combined the core and rim are both killed.



Source: Adapted from Madlambayn et al., (2016) "Leukemia regression by vascular disruption and antiangiogenic therapy". Blood. 116(9): 1539-47

MODELING ASSUMPTIONS

1. We assume that CA4P is approved and launched for prOC by 2021 in the U.S. and Europe in 2022.
2. CA4P will be used as a combination for lines of treatment in OC where Avastin is approved.
3. CA4P is approved for additional indications in glioblastoma and neuroendocrine tumors, as well as OXi4503 in AML in 2023.
4. We assume a high penetration rate in pcOC given the small size of the market and refractory nature of this stage of OC.
5. We assign risk rates to the therapeutic models based on stage of development.
6. Pricing in all indications is $100,000 per year based on the pricing of Avastin, $80,000 in Europe.
7. Pricing increased at 3% per year.
8. Mateon receives a royalty of 20% for each indication.

Exhibit 23. CA4P, Ovarian Cancer Market Model, U.S.

CA4P; Ovarain Cancer (U.S.)	2015E	2016E	2017E	2018E	2019E	2020E	2021E	2022E	2023E	2024E	2025E
Ovarian cancer incidence	22,280	22,503	22,728	22,955	23,185	23,417	23,651	23,887	24,126	24,367	24,611
Increase in incidence	1%	1%	1%	1%	1%	1%	1%	1%	1%	1%	1%
Stage III or Stage IV at diagnosis (60%)	13,368	13,502	13,637	13,773	13,911	14,050	14,190	14,332	14,476	14,620	14,767
Recurrent disease following front-line therapy, rOC (80%)	10,694	10,801	10,909	11,018	11,129	11,240	11,352	11,466	11,581	11,696	11,813
Platinum-resistant recurrent disease, prOC (50%)	5,347	5,401	5,455	5,509	5,564	5,620	5,676	5,733	5,790	5,848	5,907
Market penetration; prOC							10%	20%	25%	28%	30%
Market penetration rOC								5%	8%	10%	12%
Market penetration front-line									2%	5%	7%
Patients receiving CA4P						-	568	1,720	2,664	3,538	4,223
Cost of therapy							$ 100,000	$ 103,000	$ 106,090	$ 109,273	$ 112,551
Increase /decrease in price							3%	3%	3%	3%	3%
Revenue (000)							$ 56,762	$ 177,147	$ 282,572	$ 386,621	$ 475,330
Risk							30%	30%	30%	30%	30%
Total revenue (000)							$ 39,733	$ 124,003	$ 197,801	$ 270,635	$ 332,731
Royalty to Mateon (000) 20%							**$ 7,947**	**$ 24,801**	**$ 39,560**	**$ 54,127**	**$ 66,546**

Source: Maxim estimates

Exhibit 24. CA4P, Ovarian Cancer Market Model, Europe

CA4P; Ovarain Cancer (Europe)	2015E	2016E	2017E	2018E	2019E	2020E	2021E	2022E	2023E	2024E	2025E
Ovarian cancer incidence	45,550	46,006	46,466	46,930	47,400	47,874	48,352	48,836	49,324	49,817	50,316
Increase in incidence	1%	1%	1%	1%	1%	1%	1%	1%	1%	1%	1%
Stage III or Stage IV at diagnosis (60%)	27,330	27,603	27,879	28,158	28,440	28,724	29,011	29,301	29,594	29,890	30,189
Recurrent disease following front-line therapy, rOC (80%)	21,864	22,083	22,303	22,527	22,752	22,979	23,209	23,441	23,676	23,912	24,151
Platinum-resistant recurrent disease, prOC (40%)	8,746	8,833	8,921	9,011	9,101	9,192	9,284	9,376	9,470	9,565	9,661
Market penetration; prOC								5%	10%	20%	25%
Market penetration rOC									5%	6%	8%
Market penetration front-line									2%	4%	6%
Patients receiving CA4P								469	2,723	4,543	6,159
Cost of therapy								$ 80,000	$ 82,400	$ 84,872	$ 87,418
Increase /decrease in price								3%	3%	3%	3%
Revenue (000)								$ 37,506	$ 224,350	$ 385,603	$ 538,375
Risk								30%	30%	30%	30%
Total revenue (000)								$ 26,254	$ 157,045	$ 269,922	$ 376,863
Royalty to Mateon (000) 20%								**$ 5,251**	**$ 31,409**	**$ 53,984**	**$ 75,373**

Source: Maxim estimates

Exhibit 25. CA4P, Glioblastoma Market Model, U.S.

CA4P; Glioblastoma multiforme (GBM)	2015E	2016E	2017E	2018E	2019E	2020E	2021E	2022E	2023E	2024E	2025E
Glioblastoma incidence	10,500	10,605	10,711	10,818	10,926	11,036	11,146	11,257	11,370	11,484	11,599
Increase in incidence	1%	1%	1%	1%	1%	1%	1%	1%	1%	1%	1%
Market penetration									10%	15%	20%
Patients treated with CA4P									1,137	1,723	2,320
Cost of therapy									$ 100,000	$ 103,000	$ 106,090
Increase /decrease in price									3%	3%	3%
Revenue (000)									$ 113,700	$ 177,423	$ 246,098
Risk									75%	75%	75%
Total revenue (000)									$ 28,425	$ 44,356	$ 61,524
Royalty to Mateon (000) 20%									**$ 5,685**	**$ 8,871**	**$ 12,305**

Source: Maxim estimates

Exhibit 26. CA4P, Neuroendocrine Tumors Market Model, U.S.

CA4P; Neuroendocrine Tumors (NETs)	2015E	2016E	2017E	2018E	2019E	2020E	2021E	2022E	2023E	2024E	2025E
Neuroendocrine tumor incidence	16,200	16,362	16,526	16,691	16,858	17,026	17,197	17,369	17,542	17,718	17,895
Increase in incidence	1%	1%	1%	1%	1%	1%	1%	1%	1%	1%	1%
Market penetration									10%	15%	20%
Patients treated with CA4P									1,754	2,658	3,579
Cost of therapy									$ 100,000	$ 103,000	$ 106,090
Increase /decrease in price									3%	3%	3%
Revenue (000)									$ 175,423	$ 273,738	$ 379,694
Risk									75%	75%	75%
Total revenue (000)									$ 43,856	$ 68,435	$ 94,923
Royalty to Mateon (000) 20%									**$ 8,771**	**$ 13,687**	**$ 18,985**

Source: Maxim estimates

Exhibit 27. OXi4503, Acute Myeloid Leukemia Market Model, U.S.

OXi4503, Acute Myeloid Leukemia (AML)	2015E	2016E	2017E	2018E	2019E	2020E	2021E	2022E	2023E	2024E	2025E
Acute Myeloid Leukemia incidence	10,500	10,605	10,711	10,818	10,926	11,036	11,146	11,257	11,370	11,484	11,599
Increase in incidence	1%	1%	1%	1%	1%	1%	1%	1%	1%	1%	1%
Market penetration									10%	15%	20%
Patients treated with CA4P									1,137	1,723	2,320
Cost of therapy									$ 100,000	$ 103,000	$ 106,090
Increase /decrease in price									3%	3%	3%
Revenue (000)									$ 113,700	$ 177,423	$ 246,098
Risk									75%	75%	75%
Total revenue (000)									$ 28,425	$ 44,356	$ 61,524
Royalty to Mateon (000) 20%									**$ 5,685**	**$ 8,871**	**$ 12,305**

Source: Maxim estimates

VALUATION

Our therapeutic model assumes CA4P launches in the U.S. in 2021 and 2022 in Europe for ovarian cancer with a commercial partner, and Mateon receives a royalty stream. Additional indications in GBM and neuroendocrine tumors as well as OXi4503 for AML launch in 2023. We assign risk rates to our models based on the stage(s) of development. We then apply a 30% discount to our free cash flow, discounted EPS and sum-of-the-parts models which are equally weighted to derive a $2 price target.

Exhibit 28. Free Cash Flow Model

Average $	2

Price Target $	1
Year	2017

DCF Valuation Using FCF (mln):

units ('000)	2015E	2016E	2017E	2018E	2019E	2020E	2021E	2022E	2023E	2024E	2025E
EBIT	(13,654)	(14,052)	(16,165)	(16,655)	(17,164)	(17,336)	(9,562)	12,367	73,249	121,501	167,293
Tax Rate							0%	0%	0%	5%	8%
EBIT (1-t)	(13,654)	(14,052)	(16,165)	(16,655)	(17,164)	(17,336)	(9,562)	12,367	73,249	115,426	153,910
CapEx	(13)	-	-	-	-	-	-	-	-	-	-
Depreciation	20	20	-	-	-	-	-	-	-	-	-
Change in NWC											
FCF	(13,647)	(14,032)	(16,165)	(16,655)	(17,164)	(17,336)	(9,562)	12,367	73,249	115,426	153,910
PV of FCF	(23,063)	(18,242)	(16,165)	(12,812)	(10,156)	(7,891)	(3,348)	3,331	15,176	18,395	18,868
Discount Rate	30%										
Long Term Growth Rate	1%										
Terminal Cash Flow	536,031										
Terminal Value YE2025	65,712										
NPV	52,867										
NPV-Debt											
Shares out ('000)	50,634	2025E									
NPV Per Share	1										

Source: Maxim estimates

Exhibit 29. Discounted EPS Model

Current Year	2017
Year of EPS	2025
Earnings Multiple	10
Discount Factor	30%
Selected Year EPS	3
NPV	4

Source: Maxim estimates

	Discount Rate and Earnings Multiple Varies, Year is Constant						
	3.73	5%	10%	15%	20%	25%	30%
Earnings Multiple							
0	0	0	0	0	0	0	0
5	10.29	7.09	4.97	3.53	2.55	1.86	
10	20.57	14.18	9.94	7.07	5.10	3.73	
15	30.86	21.27	14.91	10.60	7.65	5.59	
20	41.15	28.36	19.87	14.14	10.20	7.45	
25	51.43	35.45	24.84	17.67	12.75	9.32	
30	61.72	42.54	29.81	21.21	15.30	11.18	
35	72.01	49.63	34.78	24.74	17.85	13.04	

Exhibit 30. Sum-of-the-Parts Model

Mateon Therapeutics, Inc.	LT Gr	Discount Rate	Yrs to Mkt	% Success	Peak Sales (MM's)	Term Val)
CA4P, Ovarian Cancer	1%	30%	4	80%	$142	$489
NPV						$1.62
CA4P, Glioblastoma	1%	30%	6	50%	$12	$42
NPV						$0.05
CA4P, Neuroendocrine tumors	1%	30%	6	50%	$19	$65
NPV						$0.08
OXi4503, AML	1%	30%	6	50%	$12	$42
NPV						$0.05
Net Margin						60%
MM Shrs OS (2024E)						51
Total						$2

Source: Maxim estimates

Mateon Therapeutics, Inc.: Income Statement ($000)															
: YE December 31	2015A	1Q16A	2Q16A	3Q16A	4Q16E	2016E	2017E	2018E	2019E	2020E	2021E	2022E	2023E	2024E	2025E
Revenue:															
Product sales															
Royalties															
CA4P Ovarian cancer (U.S.)										-	7,947	24,801	39,560	54,127	66,546
CA4P Ovarian cancer (EU)										-	-	5,251	31,409	53,984	75,373
CA4P, Glioblastoma multiforme												-	5,685	8,871	12,305
CA4P, Neuroendocrine tumors													8,771	13,687	18,985
OXi4503, (Acute Myeloid Leukemia)													5,685	8,871	12,305
Total Revenue	-	-	-	-	-	-	-	-	-	-	7,947	30,051	91,110	139,541	185,513
Expenses:															
Costs of Goods Sold										-	-	-	-	-	-
%COGS															
Research and Development	9,086	1,980	2,374	2,075	2,400	8,829	10,595	10,807	11,023	11,133	11,244	11,357	11,470	11,585	11,701
%R&D															
General and Administrative	4,596	1,372	1,296	1,187	1,450	5,305	5,570	5,849	6,141	6,203	6,265	6,327	6,391	6,454	6,519
%SG&A															
Total Expenses	13,682	3,352	3,670	3,262	3,850	14,134	16,165	16,655	17,164	17,336	17,509	17,684	17,861	18,040	18,220
Operating Income (Loss)	(13,682)	(3,352)	(3,670)	(3,262)	(3,850)	(14,134)	(16,165)	(16,655)	(17,164)	(17,336)	(9,562)	12,367	73,249	121,501	167,293
Investment income	27	28	29	26		83									
Other (expense) income, net	1	(1)				(1)									
Total Other Income	**28**	**27**	**29**	**26**	**-**	**82**	**-**	**-**	**-**	**-**	**-**	**-**	**-**	**-**	**-**
Pretax Income	(13,654)	(3,325)	(3,641)	(3,236)	(3,850)	(14,052)	(16,165)	(16,655)	(17,164)	(17,336)	(9,562)	12,367	73,249	121,501	167,293
Income Tax Benefit (Provision)	-	-	-	-	-	-	-	-	-	-	-	-	-	6,075	13,383
Tax Rate														5%	8%
GAAP Net Income (Loss)	(13,654)	(3,325)	(3,641)	(3,236)	(3,850)	(14,052)	(16,165)	(16,655)	(17,164)	(17,336)	(9,562)	12,367	73,249	115,426	153,910
GAAP-EPS	(0.54)	(0.13)	(0.14)	(0.12)	(0.14)	(0.53)	(0.50)	(0.42)	(0.39)	(0.36)	(0.19)	0.25	1.46	2.29	3.04
GAAP-EPS (Dil)	(0.54)	(0.13)	(0.14)	(0.12)	(0.14)	(0.53)	(0.50)	(0.42)	(0.39)	(0.36)	(0.19)	0.25	1.46	2.29	3.04
Wgtd Avg Shrs (Bas) - '000s	25,201	26,545	26,545	26,545	26,572	26,552	32,269	39,286	44,451	48,383	49,830	50,030	50,230	50,432	50,634
Wgtd Avg Shrs (Dil) - '000s	25,201	26,545	26,545	26,545	26,572	26,552	32,269	39,286	44,451	48,383	49,830	50,030	50,230	50,432	50,634

Source: Company reports and Maxim

DISCLOSURES

Mateon Therapeutics inc Rating History as of 01/26/2017

powered by: BlueMatrix

Maxim Group LLC Ratings Distribution			**As of: 01/26/17**
		% of Coverage Universe with Rating	**% of Rating for which Firm Provided Banking Services in the Last 12 months**
Buy	Fundamental metrics and/or identifiable catalysts exist such that we expect the stock to outperform its relevant index over the next 12 months.	**79%**	**29%**
Hold	Fundamental metrics are currently at, or approaching, industry averages. Therefore, we expect this stock to neither significantly outperform nor underperform its relevant index over the next 12 months.	**19%**	**11%**
Sell	Fundamental metrics and/or identifiable catalysts exist such that we expect the stock to underperform its relevant index over the next 12 months.	**3%**	**0%**
	**See valuation section for company specific relevant indices*		

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Maxim Group makes a market in Mateon Therapeutics inc

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MATN: For Mateon Therapeutics we use the BTK (Biotechnology Index) as the relevant index

Valuation Methods

MATN: Valuation. We assume CA4P launches in the U.S. in 2021 and 2022 in Europe for Ovarian Cancer, followed by additional indications in 2023. A 30% discount is applied to the FCF, Discounted EPS and SOP models which are equally weighted to derive a price target.

Price Target and Investment Risks

MATN: Aside from general market and other economic risks, risks particular to our price target and rating for MateonTherapeutics include: 1) No assurances of regulatory approval(s) and or timing of such; 2) No assurances of the company's ability to penetrate the market; 3) The company may need to raise additional capital to finance operations.

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