ROTH Capital Partners

**Mark Breidenbach, Ph.D.**, (646)-616-2786
mbreidenbach@roth.com

**Matthew Biegler**, (646) 616-2791
mbiegler@roth.com

**Sales** (800) 933-6830, **Trading** (800) 933-6820



## Healthcare: Biotechnology

# Mateon Therapeutics, Inc. | MATN - $0.77 - OTC | *Buy*

### Initiation of Coverage

| Stock Data | |
|---|---|
| 52-Week Low - High | $0.27 - $1.02 |
| Shares Out. (mil) | 26.55 |
| Mkt. Cap.(mil) | $20.5 |
| 3-Mo. Avg. Vol. | 81,391 |
| 12-Mo.Price Target | $2.00 |
| Cash (mil) | $12.0 |
| Tot. Debt (mil) | $0.0 |

**EPS $**

| Yr Dec | —2016— | —2017E— | —2018E— |
|---|---|---|---|
| | | Curr | Curr |
| 1Q | (0.13)A | (0.13)E | (0.10)E |
| 2Q | (0.14)A | (0.09)E | (0.08)E |
| 3Q | (0.12)A | (0.10)E | (0.08)E |
| 4Q | (0.13)A | (0.10)E | (0.09)E |
| YEAR | (0.51)A | (0.41)E | (0.35)E |
| P/E | NM | NM | NM |

**Revenue ($ millions)**

| Yr Dec | —2016— | —2017E— | —2018E— |
|---|---|---|---|
| | | Curr | Curr |
| 1Q | 0.0A | 0.0E | 0.0E |
| 2Q | 0.0A | 0.0E | 0.0E |
| 3Q | 0.0A | 0.0E | 0.0E |
| 4Q | 0.0A | 0.0E | 0.0E |
| YEAR | 0.0A | 0.0E | 0.0E |

## MATN: A Pathway to Registration Finally in FOCUS; Initiating with Buy

We are initiating coverage on Mateon Therapeutics with a Buy rating and $2 price target based on a discounted EPS and revenue multiple analysis. Following positive results from a randomized Phase 2 study conducted by the Gynecologic Oncology Group (GOG), Mateon is conducting a pivotal trial (FOCUS) of its lead drug, CA4P, in a group with high unmet need—patients with platinum-resistant ovarian cancer. We believe a series of interim clinical readouts in 2017 could drive shareholder value.

**Lead therapeutic candidate shows activity in the clinic.** CA4P is a reversible tubulin-binding small molecule that distorts the shape of vascular endothelial cells in disorganized blood vessels, obstructing blood flow to tumors. The drug has been tested in over 475 patients with a variety of different cancers. Of note, in a Phase 2 GOG-sponsored study comparing CA4P + bevacizumab to bevacizumab alone in ovarian cancer, the combination therapy showed a progression-free survival (PFS) advantage, with a median PFS of 7.3 versus 4.8 months for monotherapy (HR=0.68; p=0.049). We believe the results compare favorably to the current standard of care (bevacizumab + chemotherapy) set by the AURELIA trial in 2014 (n=360, mPFS 6.8 vs. 3.4 months, p<0.0001). The combination worked especially well in Pt-resistant patients, showing a median PFS of 6.7 months versus 3.4 months for the monotherapy (HR=0.57; p=0.01). Because Pt-resistant patients lack effective therapeutic options, CA4P received fast-track designation in this indication in 2016.

**Experienced leadership drives development strategy.** In 2015, Dr. Bill Schwieterman was appointed as CEO of Mateon. Of note, Dr. Schwieterman previously served with the FDA as Chief of Immunology and Infectious Diseases at the Center for Biologics Evaluation and Research (CBER). By focusing most of the company's resources on the registration of CA4P in Pt-resistant ovarian cancer, we believe the current management team is pursuing the best strategy to translate CA4P's known clinical activity into a clinically meaningful benefit. We believe Dr. Schwieterman has the correct pedigree to get CA4P across the finish line.

**VDAs are active in liquid tumors.** Mateon's second therapeutic candidate OXi4503 is being evaluated in AML, and the company is currently conducting a Phase 1b/2 clinical trial to evaluate a combination of OXi4503 and cytarabine. In liquid tumors, the company has presented evidence suggesting OXi4503 acts on endothelial cells in the bone marrow, forcing cancer cells into an active cell cycle, thereby sensitizing them to chemotherapy. Results from the first three dose-escalation cohorts have shown good safety coupled with signs of efficacy. We anticipate full Phase 1b results in 2H17.

## Investment Thesis

### Company Summary

Mateon Therapeutics (formerly OXiGENE) was founded and incorporated first in New York in 1988 and then Delaware in 1992, with an IPO in August 1993. Mateon's corporate headquarters are currently located in South San Francisco, CA. As of March 2017, Mateon had a total of 16 full-time employees. The company relies on outsourcing for much of its research, development, preclinical testing and clinical trial activity, but maintains managerial and quality control over its clinical trials. Mateon is focused on the development of vascular disrupting agents (VDAs) for the treatment of cancer. Mateon's clinical-stage pipeline includes two VDAs: CA4P, also known as fosbretabulin, and OXi4503. CA4P is currently being investigated in a Phase 2/3 pivotal trial (FOCUS) in platinum-resistant ovarian cancer, while OXi4503 is being investigated in a dose-escalation Phase 1/2 trial in acute myeloid leukemia (AML). Based on evidence from prior clinical trials of CA4P and OXi4503, it appears that VDAs have anti-tumor activity and synergize with chemotherapy and anti-angiogenic drugs.

### Investment highlights and key drivers

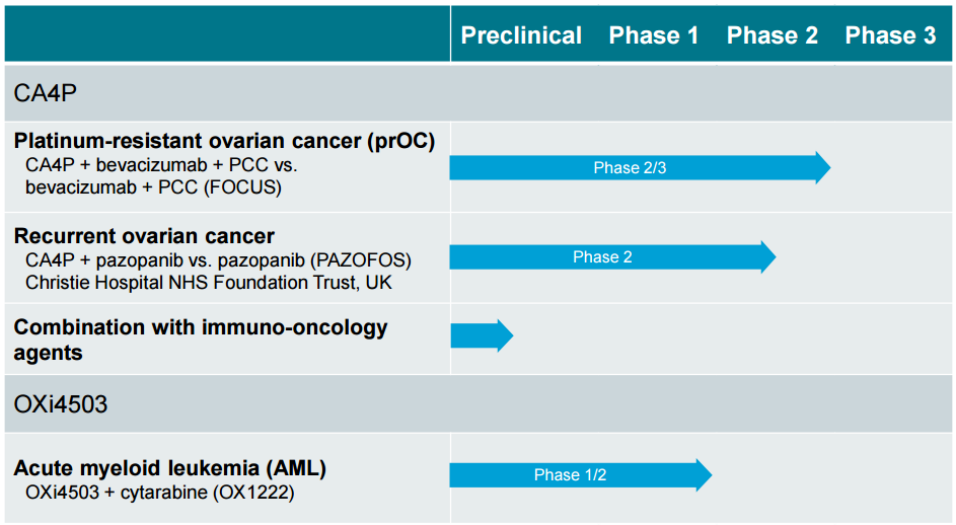
- **Under new leadership, CA4P has viable development strategy.** CA4P, also known as fosbretabulin, has been in clinical investigations for over a decade and has shown activity in a variety of different cancers. Based on its performance in three separate studies with control arms, we believe the molecule has undisputable evidence of clinical activity (in each trial, the active arm of treatment yielded higher response rates) paired with a manageable safety profile that allows combinational use with other agents. Under the leadership of a new CEO (appointed in 2015), Mateon has emerged with a streamlined clinical and regulatory strategy for CA4P, primarily aimed at platinum-resistant ovarian cancer.

- **Management knows what it takes to get FDA nod.** The current CEO of Mateon, Dr. Bill Schwieterman, served as the FDA's Chief of Immunology and Infectious Diseases in CBER. We believe Dr. Schwieterman has the necessary experience to get CA4P approved by the FDA, given his experience in drug development and clinical research. We believe Pt-resistant ovarian cancer is the best indication for the company to pursue. The company is currently enrolling patients in the Phase 2/3 FOCUS trial; while we believe the trial could take at least three years before delivering topline data, we believe that the multiple pre-planned efficacy analyses built into the protocol could drive shareholder value in the next 12 months. Specifically, the trial's Phase 2 portion includes three pre-planned efficacy analyses (ORR + PFS, see Expected Catalysts below), and the fourth pre-planned efficacy analysis after 80 patients have been enrolled could trigger the initiation of the Phase 3 portion of the study.

- **GOG study guides path for CA4P.** The GOG-0186I trial (NCT01305213) was a GOG-sponsored, randomized, open-label Phase 2 study in 107 patients with ovarian cancer that compared CA4P + bevacizumab to bevacizumab alone. The trial met its primary endpoint of improvement in progression-free survival (PFS), with a median PFS of 7.3 versus 4.8 months for monotherapy (HR=0.68; p=0.049). In our view, the results compare favorably to the current standard of care (bevacizumab + chemotherapy) set by the AURELIA trial in 2014 (n=360, mPFS 6.8 vs. 3.4 months, p<0.0001). To adapt to the new standard of care, Mateon's management team decided to focus CA4P development efforts in combination with bevacizumab and chemotherapy. Although the triplet had yet to be investigated in ovarian cancer, and we generally are cautious on jumping into large-scale studies without sufficient Phase 2 support, we believe that the company's Phase 2/3 FOCUS trial has been thoughtfully designed, with multiple pre-planned analyses of efficacy to ensure a prudent yet cost-effective approach to regulatory approval. The first interim analysis has indicated that the triplet has higher efficacy relative to the control arm (22% vs. 9% ORR) and is well tolerated.

- **AML could be a dark horse indication.** Mateon's second clinical-stage VDA, OXi4503, is being evaluated in AML. We believe the company has identified the main mechanisms of action of OXi4503 and is targeting an important aspect of AML tumor biology, chemoresistance and relapse. By disrupting the interactions between tumor cells and endothelial cells in the bone marrow niche, we believe OXi4503 sensitizes cancer cells to chemotherapy. In our view, this approach is potentially agnostic to patient-specific cytogenetic and molecular profiles, an important consideration in a heterogeneous disease like AML. Mateon is currently conducting a Phase 1b/2 clinical trial to evaluate a combination of OXi4503 and cytarabine, a strategy that we believe is supported by preclinical results. Data from the first three dose-escalation cohorts has shown encouraging signs of efficacy coupled with a good safety profile. We anticipate full Phase 1b results in 2H17, which could attract partnership interest and/or advancement into Phase 2.

## Mateon Therapeutics Pipeline




*Source: Mateon Company Presentation, March 2017*

## Expected Catalysts for the Next 12 Months

**CA4P (Platinum-Resistant Ovarian Cancer):**

- Potential to present Phase 2/3 FOCUS study 2nd interim analysis (n=40, ORR, PFS) (August 2017)
- Potential to present Phase 2/3 FOCUS study 3rd interim analysis (n=60, ORR, PFS) (2H17)
- Potential to present Phase 2/3 FOCUS study 4th interim analysis (n=80, ORR, PFS) (2H17)

**CA4P (Recurrent Ovarian Cancer):**

- Potential to complete Phase 2 PAZOFOS pazopanib combination study (n= ~116) (2018)

**CA4P (Glioblastoma):**

- Potential to initiate Phase 2/3 bevacizumab combination study (1H17)

**OXi4503/CA1P (AML):**

- Potential to complete Phase 1b OX1222 study 3rd and 4th cohorts (1H17)
- Potential to complete Phase 1b OX1222 study 5th cohort (study completion) and present data (2H17)

## Valuation Methodology

We value Mateon Therapeutics using a discounted earnings per share and revenue multiple analysis. Given the current stage of development of CA4P, we project a potential commercial launch in 2021. Based on the design of the ongoing FOCUS study, we believe CA4P could be approved as part of a second-line combination therapy (with bevacizumab and chemotherapy) in Pt-resistant ovarian cancer. The GOG has defined patients with documented recurrence within six months of completing initial therapy as Pt-resistant (*The Oncologist* 2000 vol. 5 no. 1 26-35). Approximately 25% of patients who relapse after front-line therapy fall into the Pt-resistant category (*Journal of Clinical Oncology* 32, no. 13 (May 2014) 1302-1308), translating to an addressable U.S. population of approximately 4,000 patients. Assuming an average treatment price of $60,000 for CA4P, we estimate a market size of $266M by 2025, the basis year of our valuation. Assuming moderate market penetration, we estimate a peak U.S. market of $150M, yielding probability adjusted (50%) revenues of $12M in 2021, increasing to $63.6M in 2025, as summarized in Exhibit 1.

**Exhibit 1: Market Model for CA4P**

| | 2017 | 2018 | 2019 | 2020 | 2021 | 2022 | 2023 | 2024 | 2025 |
|---|---|---|---|---|---|---|---|---|---|
| **CA4P-U.S. Market in Pt-resistant ovarian cancer** | | | | | | | | | |
| US Population ('000) | 327,756 | 330,444 | 333,127 | 335,820 | 340,888 | 347,760 | 354,771 | 361,924 | 369,220 |
| Deaths per year from ovarian cancer | 15,732 | 15,861 | 15,990 | 16,119 | 16,363 | 16,692 | 17,029 | 17,372 | 17,723 |
| Pt-resistant addressable population | 3,933 | 3,965 | 3,998 | 4,030 | 4,091 | 4,173 | 4,257 | 4,343 | 4,431 |
| Cost of treatment ($'s) | 60,000 | 60,000 | 60,000 | 60,000 | 60,000 | 60,000 | 60,000 | 60,000 | 60,000 |
| Market Size ($'s) | 235,984,030 | 237,919,580 | 239,851,468 | 241,790,400 | 245,439,083 | 250,387,292 | 255,435,260 | 260,584,999 | 265,838,560 |
| % Market penetration | | | | | 10% | 17% | 27% | 39% | 48% |
| CA4P Sales-U.S. ($'000) | | | | | | | | | |
| Sales ($ '000) - 2Q21 Launch | | | | | 24,000,000 | 43,500,000 | 69,900,000 | 100,800,000 | 127,200,000 |
| Sales Revenue ($'000) | | | | | 12,000 | 21,750 | 34,950 | 50,400 | 63,600 |

*Source: ROTH Capital Partners*

Profitable biotechnology companies have historically traded in a multiple range of 6-10x in revenues. For reference, an average of five profitable biotech companies with marketed products in oncology indications (including CELG-NC; AMGN-NC; SHPG-NC; NVS-NC; JNJ-NC) recently traded at an average of 5.4x sales revenue. We value Mateon Therapeutics using a revenue multiples analysis. Applying an 8x multiple to our probability-adjusted 2025 revenue estimate of $63.6M and discounting by 20% over eight periods, we obtain a $1.66 target price.

Profitable biotechnology companies have historically traded in a multiple range of 26-30x in EPS. For reference, an average of five profitable biotech companies with marketed products in oncology indications (including CELG; AMGN; SHPG; NVS; JNJ) recently traded at an average of 26.7x trailing EPS. We also value Mateon Therapeutics using a discounted earnings multiple analysis. Applying a 28x multiple to our probability adjusted 2025 EPS of $0.31 and discounting by 20% over eight periods, we obtain a $2.05 target price.

Averaging the results from these two methods and adding the projected cash per share in 12 months, we obtain a 12-month price target of $1.97 per share, which we round to $2.

**Exhibit 2: Valuation and Sensitivity Analysis**

| | | Discount Rate | | | | | |
|---|---|---|---|---|---|---|---|
| | | 10% | 15% | 20% | 25% | 30% | 35% |
| P/E Multiple | 24 | $3.52 | $2.46 | $1.75 | $1.26 | $0.92 | $0.68 |
| | 26 | $3.81 | $2.67 | $1.90 | $1.37 | $1.00 | $0.74 |
| | 28 | $4.10 | $2.88 | $2.05 | $1.48 | $1.08 | $0.80 |
| | 30 | $4.40 | $3.08 | $2.19 | $1.58 | $1.16 | $0.85 |
| | 35 | $5.13 | $3.59 | $2.56 | $1.84 | $1.35 | $1.00 |
| | | 10% | 15% | 20% | 25% | 30% | 35% |
| Revenue Multiple | 4.0 | $1.67 | $1.17 | $0.83 | $0.60 | $0.44 | $0.32 |
| | 6.0 | $2.50 | $1.75 | $1.25 | $0.90 | $0.66 | $0.49 |
| | 8.0 | $3.33 | $2.33 | $1.66 | $1.20 | $0.88 | $0.65 |
| | 10.0 | $4.16 | $2.92 | $2.08 | $1.50 | $1.09 | $0.81 |
| | 12.0 | $5.00 | $3.50 | $2.49 | $1.80 | $1.31 | $0.97 |
| | 14.0 | $5.83 | $4.09 | $2.91 | $2.10 | $1.53 | $1.13 |
| | 16.0 | $6.66 | $4.67 | $3.32 | $2.40 | $1.75 | $1.29 |
| | 18.0 | $7.50 | $5.25 | $3.74 | $2.70 | $1.97 | $1.46 |
| | 20.0 | $8.33 | $5.84 | $4.15 | $3.00 | $2.19 | $1.62 |

| **Discounted Earnings Analysis** | | |
|---|---|---|
| Estimated 2025 EPS | $ | 0.31 |
| Year | | 2025 |
| Periods (years) | | 8.0 |
| Price target | | **$2.05** |
| **Discounted Revenue Analysis** | | |
| Estimated 2025 Revenues (000s) | $ | 63,600 |
| Year | | 2025 |
| Periods (years) | | 8.0 |
| Shares outstanding (000s): | | 71,254 |
| Price target | | **$1.66** |
| **Average Price Target Combining Both Methods** | | **$1.85** |
| **Average Price Target Including Cash Per Share** | | **$1.97** |

*Source: ROTH Capital Partners*

**Valuation upside potential**. Our valuation of Mateon Therapeutics is based solely on our sales projections for CA4P in Pt-resistant ovarian cancer, but we see upside potential from several avenues. Due to its early stage of development, we are currently excluding sales projections for OXi4503 in AML. If approved, the CA4P/bevacizumab/chemotherapy combination may be used in a broader second line setting which may include patients who relapse more than six months after initial response. Additionally, we believe CA4P could attract commercial partnership(s) covering ex-U.S. territories, which could potentially include milestones and royalty payments.

**Valuation downside potential.** As with a majority of clinical stage biotechnology companies, there always exists the risk of failed or inconclusive clinical trials, which could lead to downward pressure on the stock. We note that the standard of care in second-line ovarian cancer may change before the Phase 3 FOCUS study readout. For example, the Poly ADP-Ribose Polymerase (PARP) inhibitor niraparib is slated to enter Phase 3 evaluation in first-time recurrent ovarian cancer patients later this year.

### Financial Outlook and Summary

Mateon Therapeutics had approximately $12M in cash and cash equivalents at the end of 4Q16. We estimate the company has sufficient resources to fund operations until 4Q17. We are projecting losses of $14.4M in 2017 and $15.1M in 2018 or $(0.41) and $(0.35) per share, respectively. We expect to see increases in cash burn, associated with increased clinical trial expenses the FOCUS study potentially advances into Phase 3 in 2018. For 2017, we estimate R&D expenses will be $9.7M and G&A expenses will be $4.7M. We believe the company's lead product, CA4P, could reach the market in Pt-resistant ovarian cancer in 2021.

## Risks

- **Financing risk.** As with a majority of development-stage biotechnology companies, the ability to maintain sufficient funding is critical to the progress of pipeline candidates. The company has guided that its current cash position can support operations until October 2017. Mateon may need to seek additional dilutive financing options via the capital markets. Recent delisting from The NASDAQ Capital Market may significantly impair Mateon's ability to raise capital. Should Mateon experience problems raising additional capital, its development programs' progress could be significantly impeded, leading to both delays in development timelines as well as potential negative effects on investor confidence. Each of these could have a negative impact on the share price.

- **Clinical and regulatory risk.** Mateon's future success depends on its ability to develop and commercialize new products in a timely and cost-effective manner. We believe that the company is on track to meet its milestones. However, Mateon may experience delays in initiation, enrollment, or completion of its clinical trials and regulatory authorities could impose additional conditions on these trials. Additionally, results from ongoing clinical trials may not be predictive of the results of future pivotal clinical trials. While we believe CA4P and OXi4503 have shown encouraging results in early studies, we acknowledge that both ovarian cancer and AML are challenging indications and that multiple competitor products are in development for both.

- **Commercial risk.** The company's therapeutic candidates, including CA4P, may not obtain the market penetration and sales forecasted by our estimates or those of the company given the competitive marketplace and pricing dynamics in place in the U.S. and E.U. If approved, CA4P may be subject to a narrow label that would restrict use to a smaller-than-anticipated pool of addressable patients. Further, the company currently lacks a sales force or distribution capabilities, and therefore may be unable to commercialize approved products successfully.

- **Reliance on third parties.** As of March 2017, Mateon has only 16 full-time employees and relies heavily on CROs and other third parties for clinical trial activities and outsources all of its manufacturing. Third-party CROs and manufacturers may not be able to meet the company's needs with respect to timing, quantity or quality. If Mateon is unable to contract for a sufficient supply of needed materials on acceptable terms, or encounters

delays or difficulties in its relationships with manufacturers or CROs, the company's clinical testing may be delayed, thereby delaying the submission of products for regulatory approval or the market introduction and subsequent sales of its products.

- **Intellectual Property and Licensing Risk.** The commercial success of Mateon Therapeutics depends on its ability to develop, manufacture, and commercialize proprietary technologies without infringing on the proprietary rights of third parties. Any loss of license rights to use certain critical intellectual property from licensors could have a material adverse effect on the company's business. Mateon has in-licensed exclusive worldwide rights to CA4P and OXi4503 from Arizona State University and Bristol-Myers Squibb (BMY-NC) and does not own any intellectual properties or technologies. If CA4P is approved, Mateon will be required to pay low-single-digit royalties on future net sales of products associated with the BMS patent rights until these patent rights expire. Excluding extensions and orphan exclusivity, we believe composition of matter patents on CA4P and OXi4503 expire in 2021, and a method patent on OXi4503 expires in 2028. Mateon has filed a method patent on the use of CA4P which, if approved, would expire in 2036.

## Platform Overview

Mateon's stable of vascular disrupting agents (VDAs) consists of two molecules: CA4P, also known as fosbretabulin, and OXi4503. CA4P is currently being investigated in a Phase 2/3 pivotal trial (FOCUS) in platinum-resistant ovarian cancer, while OXi4503 is being investigated in a dose-escalation Phase 1b trial in acute myeloid leukemia (AML). While the two molecules resemble each other structurally, they could have different effects mechanistically depending on the cancer type. In solid tumors, VDAs target endothelial cells that form the supportive vasculature tumors rely on to survive. In liquid tumors, VDAs appear to disrupt bone marrow endothelial cells that protect AML cells from chemotherapy. In both indications, VDAs have displayed synergy with chemotherapies and/or anti-angiogenic drugs.

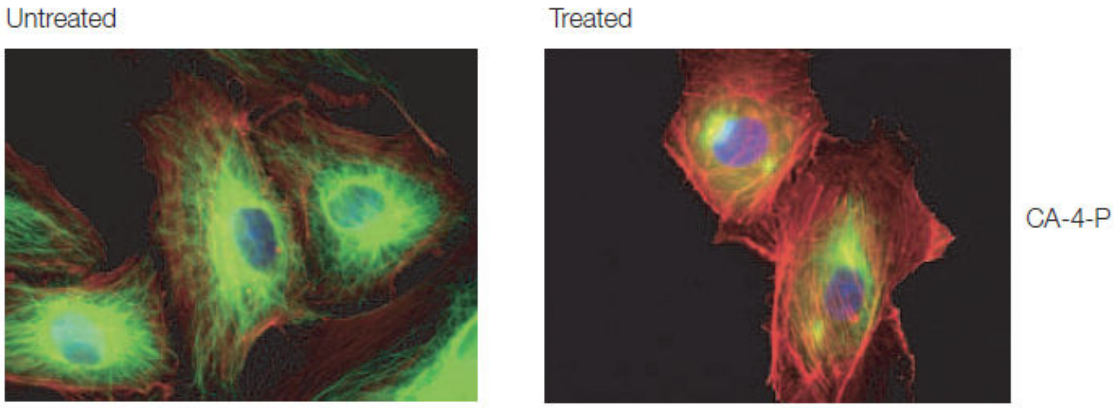
### Targeting the tumor vasculature is a validated therapeutic approach in solid tumors

CA4P targets the supportive network of tumor vasculature that tumors require to thrive. Tumor tissue, like any other tissue, needs oxygen to survive. As tumors grow, they rely on the formation of new blood vessels to supply their metabolic and respiratory needs. Disruption of the neovascularization process has led to the developmental success of drugs targeting vascular endothelial growth factor (VEGF). However, VEGF is involved in the initial signaling cascade that leads to the formation of new blood vessels, and therefore its utility as a target may be limited to reducing the growth and spread of cancers. In contrast, VDAs target the existing framework of endothelial cells in solid tumors and may represent a novel means to disrupt the tumor vasculature and subsequently attack entrenched tumors.

#### Hitting solid tumors where it hurts

VDAs reversibly target microtubules, dynamic cytoskeletal proteins that are responsible for maintaining cell shape and affecting the events of cell division. The first VDA discovered, colchicine, was found to have damaging effects on tumor vasculature decades ago; however, its therapeutic window limited its utility. Mateon's CA4P similarly binds to tubulin and inhibits its polymerization to microtubules; meanwhile, its anti-vascular activity is apparent at well below maximum tolerated doses (MTDs) in animal models. Its wider therapeutic window has been attributed to its reversible binding nature as well as its short half-life (several hours) in serum. Because microtubules are in a constant state of dynamic remodeling (microtubule ends lengthen and shorten over time in a random process called dynamic instability), inhibition of polymerization causes rapid changes in the architecture of the cell structure (see below, tubulin staining in Green).

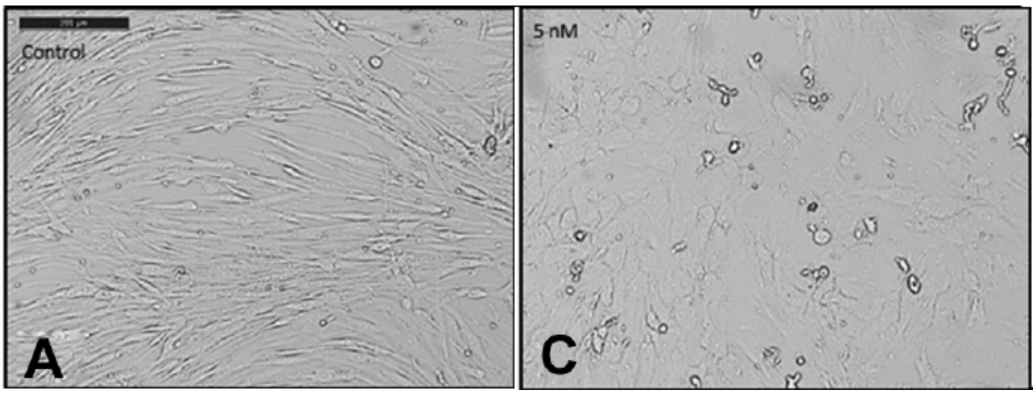
**Exhibit 3: CA4P Disrupts Tubulin Polymerization**




*Source: Nat Rev Cancer. 2005 Jun;5(6):423-35*

The structural changes include rounding, depolymerization, and detachment from supportive tissue. Adding a VDA to co-cultures of endothelial cells *in vitro* (dosed between 0.1 and 1.0 µM) resulted in rapid structural changes (see below). Other classes of therapies, such as platinum based chemotherapies, have been highly effective at targeting microtubules. However, these work by inhibiting the depolymerization of microtubules and as such suspend mitosis in dividing cells. Unfortunately, resistance to treatment develops in most cases. We believe that CA4P, by targeting the tumor's vasculature and not the tumor itself, may be less prone to resistance mechanisms.

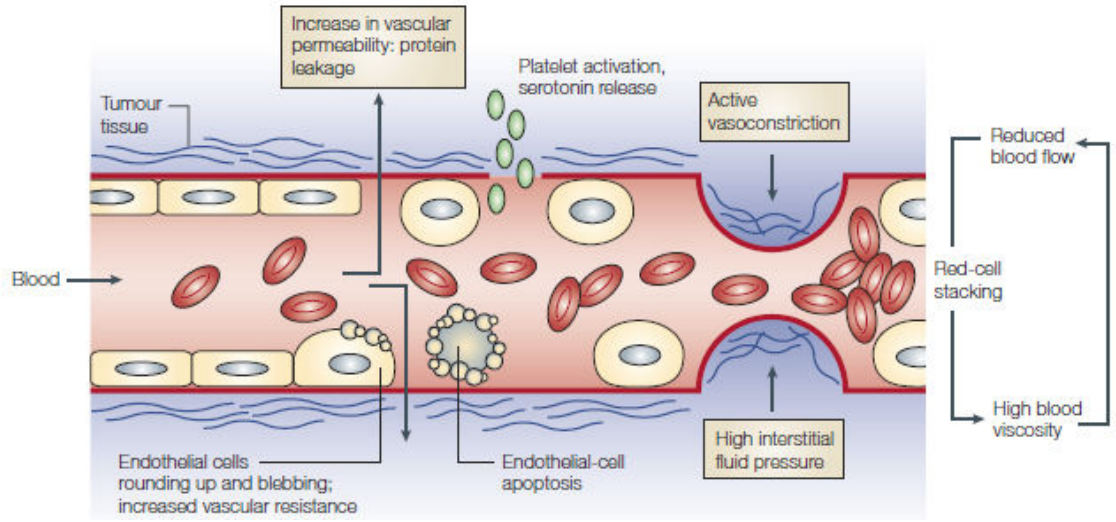
**Exhibit 4: VDAs Alter Endothelial Cell Morphology**




*Source: Exp Hematol. 2016 May;44(5):363-377*

Through mechanisms yet unknown, CA4P activates the RHO-GTP pathways resulting in the disruption of tight junctions between cells and initiation of a rapid necrotic-cell-death pathway that appears to be exclusive to endothelial cells (as a reminder, endothelial cells uniquely require plasticity to allow extravasation of proteins and solutes into tissues). *In vivo*, the loss of endothelial supportive cells cause a cascade of deleterious effects on the supportive vasculature, including occlusion and active vasoconstriction, increases in vascular permeability, and increases in blood viscosity due to vasculitis (see below), all of which restrict blood flow to the tumor.

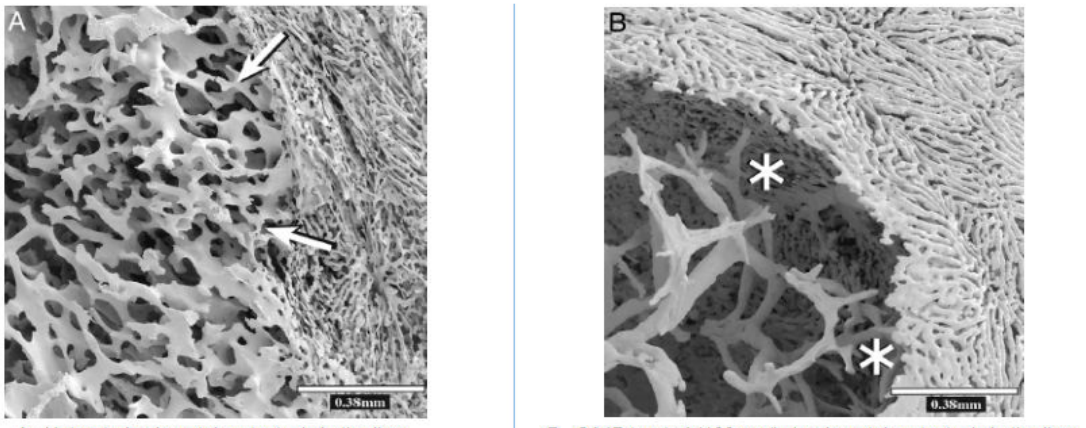
**Exhibit 5: Proposed Mechanism of Action for VDAs**




*Source: Nat Rev Cancer. 2005 Jun;5(6):423-35*

When CA4P is deployed in mouse xenograft models, vascular shutdown is rapid (within one hour) and with its blood supply cut, the tumors die of necrosis (see below). Importantly, the damaging effect of CA4P on vasculature appears to be highly selective for tumor tissues. As illustrated below, there appears to be no apparent effect on the morphology and network of healthy vasculature.

**Exhibit 6: Colorectal Liver Metastases In Mice Before and After Treatment with CA4P**




A: Untreated colorectal metastasis in the liver. B. CA4P treated (100 mg/kg) colorectal metastasis in the liver.

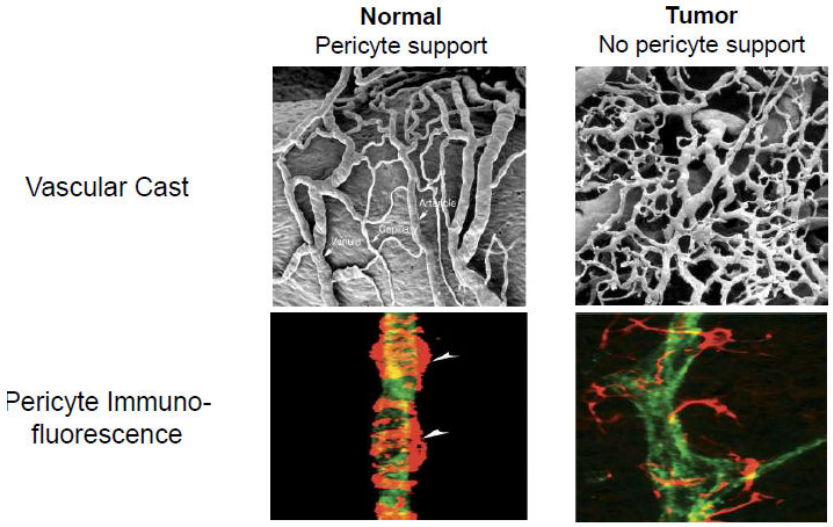
*Source: Clin Cancer Res. 2001;7:1052-1060*

**CA4P is selective for tumor vasculature**

Importantly, preclinical work in mice has demonstrated that the vascular-disrupting potential of CA4P is highly selective for tumor vasculature over normal vasculature (*Blood* 2002; 99:2060-69). Differences between the architecture of tumor vasculature and the nature of supportive tissues could explain this selectiveness. While normal vasculature is highly organized, tumor vasculature networks are chaotic, with random branching and inadequate drainage. The poor structural organization causes highly variable blood flow through capillaries with areas of high interstitial pressure that may be more susceptible to blockage by VDAs (*Nat Med*. 2003;9(6):713-25). Further, the tumor vasculature is often characterized as immature, lacking supporting pericyte and smooth muscle cells (see

below), and without this supportive framework, endothelial collapse caused by VDAs could leads to a more deleterious effect than in normal tissue.

**Exhibit 7: VDAs Selectively Act on Immature (Tumor) Vasculature**



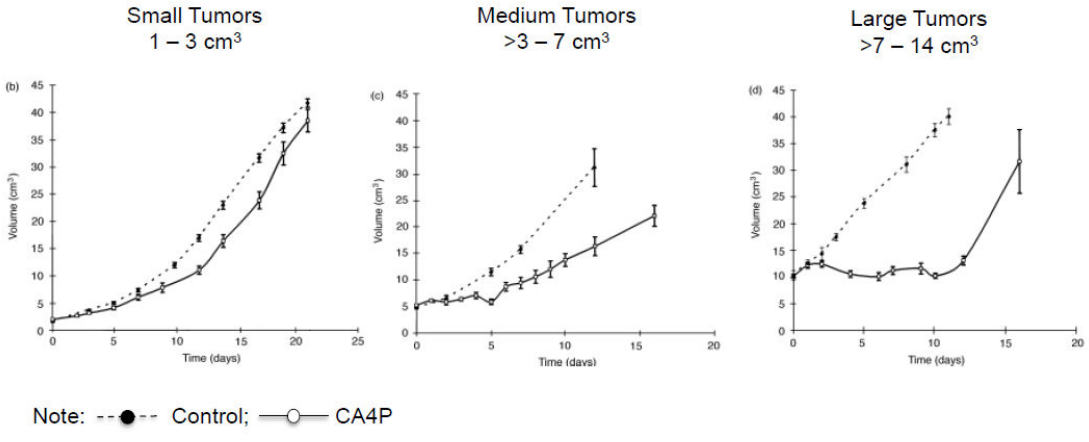

*Source: Nat Med. 2003;9(6):713-25*

In our view, preclinical work has led to two important insights on how to best deploy CA4P:

**(1) CA4P has increased efficacy in larger tumors:**

Xenograft work in mice has repeatedly shown that treatment with CA4P causes a reproducible core of necrosis in the tumors, which is surrounded by a rim of viable cells on the outside. The rationale is that peripheral tumor cells are better perfused by normal blood vessels and less dependent on tumor vasculature arterioles that supply blood to the tumor. As a result, tumors with larger diameters (and therefore more tumor vasculature-dependent cells in the core) are more targetable with CA4P (see below).

**Exhibit 8: CA4P May Work Best in Large Tumors**

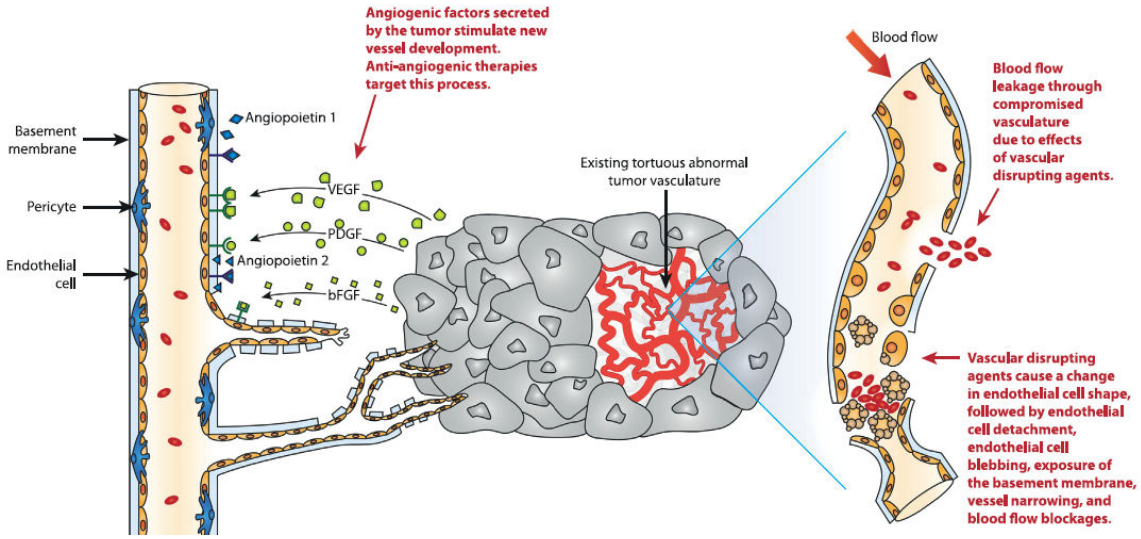



*Source: Eur J Cancer 2000;34(14):1833-43*

**(2) CA4P works best as part of a combination therapy:**

Possibly due to the viable rim of cells that remain after treatment with CA4P, the drug appears to work best when paired with other agents. Anti-VEGF agents, such as bevacizumab, could potentially choke peripheral tumor cells from forming new vasculature to support their growth. These peripheral cells may be more dependent on VEGF signaling than tumor cells in the core (see figure below).

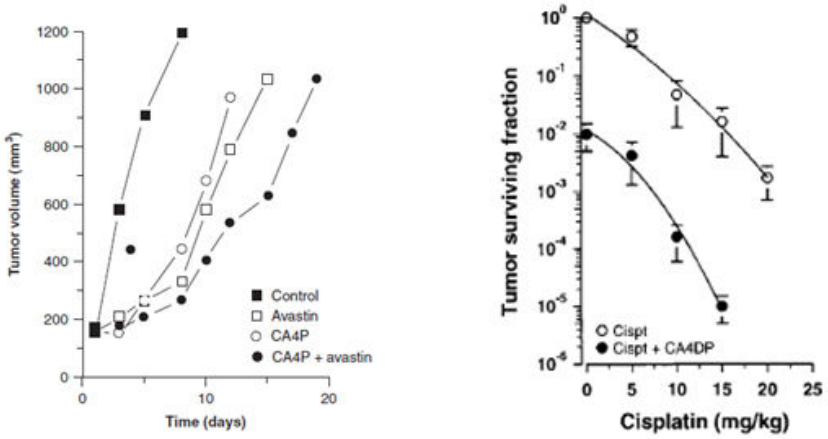
**Exhibit 9: Mechanism of Synergy with Anti-Angiogenic Drugs**




*Source: Gynecol Oncol. 2017 Feb 24. pii: S0090-8258(17)30075-6.*

Xenograft models have shown that CA4P pairs well with both bevacizumab and platinum-containing chemotherapies. As shown below (left panel), mice bearing Cakl-1 renal cell carcinoma xenografts were treated with 100 mg/kg CA4P three times per week and 2 mg/kg bevacizumab (Avastin) five times per week for two weeks. This combination yielded greater tumor volume reductions than either drug alone. As also shown below (right panel), the tumor surviving fraction of human breast cancer SKBR3 xenografts was reduced with combinations of CA4P and cisplatin, as compared to cisplatin alone.

**Exhibit 10: Preclinical Evidence of Synergies with Avastin and Chemotherapy**

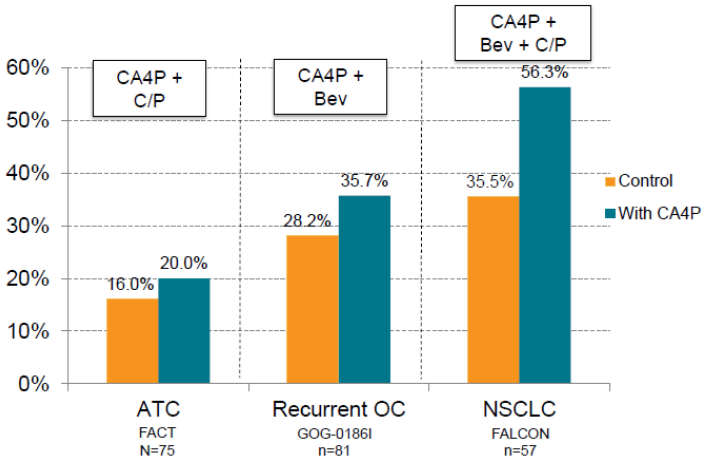



Source: *Anticanc Res 2008;28:2027-2032.and Int J Canc 2002;99(1):1-6.*

## CA4P Has Repeatedly Demonstrated Clinical Activity

CA4P has been evaluated in three separate randomized studies. In each, the treatment including CA4P yielded a higher ORR than the control arm (see below).

**Exhibit 11: Prior Randomized Clinical Trials of CA4P**




*Source: Mateon Company Presentation, February 2017*

In the FACT study, (Fosbretabulin in Anaplastic Cancer of the Thyroid), CA4P combined with standard of care carboplatin + paclitaxel (C+P) chemotherapy elicited a higher ORR than C+P alone. The study was designed to enroll 180 patients, but was cut short at 84 patients due to slow enrollment. Despite the limited enrollment, the active arm demonstrated a trend toward improved OS (median OS 5.2 versus 4.0 months, HR=0.72).

In the FALCON study, in the front-line NSCLC setting, CA4P was combined with bevacizumab and C+P and yielded an ORR of 56% compared to 36% for the B/C/P arm. This increased response rate did not translate to an improvement in PFS or OS, although we note that the trial only randomized 63 patients.

Finally, in a randomized GOG-initiated study in patients with platinum-resistant ovarian cancer, 36% of patients responded to the active treatment (CA4P + bevacizumab) compared to 28% of patients in the control arm (bevacizumab) in patients with measurable disease (for the overall patient population, the ORR was 28% in the active arm compared to 20%). This increased response translated to an improvement in mPFS (7.3 versus 4.8 months, HR= 0.685, p=0.049), despite the limited size of the trial (n=107), and overall survival trended toward significance (26.8 versus 21.2 mo, HR=0.77).

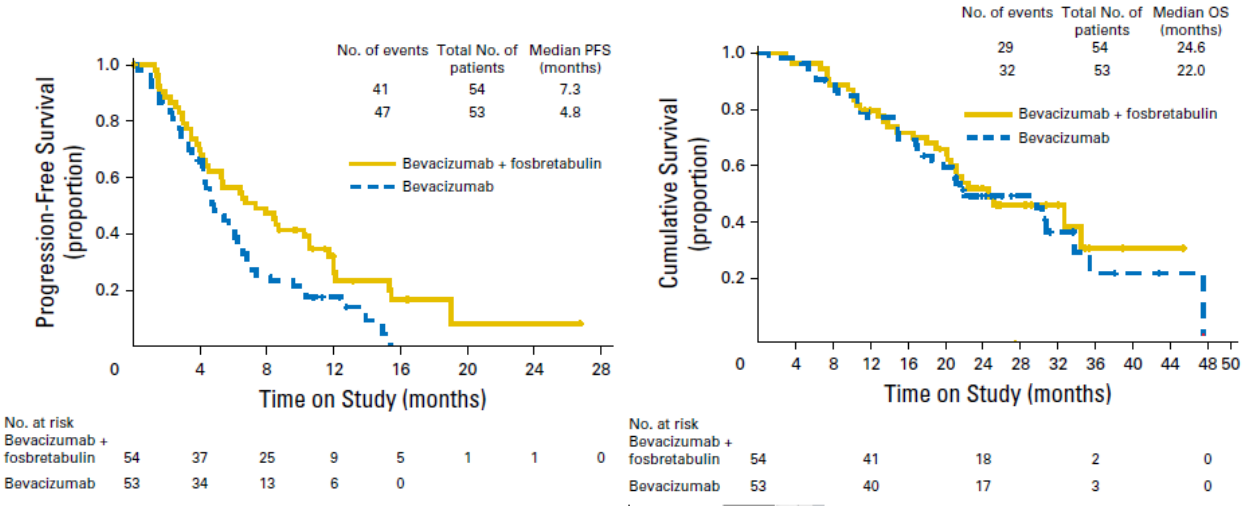
Clinically, CA4P has demonstrated a manageable safety profile. A known side-effect of VDAs is hypertension, and occurs in up to 50% of patients. In these patients, systolic and diastolic blood pressure rises approximately 10-15% (*Vasc Pharmacol* 2009;51(5-6)337-43). These cases are often transient (presenting within two hours of exposure and resolving within six hours) and manageable with anti-hypertensives.

### FOCUS is guided by prior GOG Study

The Phase 2 NRG Oncology/Gynecology Oncology Group (GOG) study randomized 107 patients with either platinum resistant or platinum sensitive metastatic ovarian cancer to treatment with either the VEGF inhibitor bevacizumab alone or bevacizumab + CA4P. Results from the trial were recently published in the Journal of Clinical Oncology (*J Clin Oncol.* 2016 Jul 1;34(19):2279-86). The study showed that adding CA4P to bevacizumab seemed to prolong PFS compared with bevacizumab alone thereby meeting the primary objective of the study (median PFS, 4.8 months for bevacizumab and 7.3 months for bevacizumab plus CA4P; HR, 0.69; two-sided 90% CI, 0.47 to 1.00; one-side P =0.049, see below). The trial arms were balanced in terms of patient platinum status, platinum interval, number of prior therapies, and age. There was a slight imbalance in baseline performance status (81% Status 0 in the

combination compared to 68% in the control). Stratifying by performance status did not seem to impact mPFS (0 vs. 1 or 2; HR=0.66). Overall survival also trended to significance (26.8 versus 21.2 months, HR=0.77).

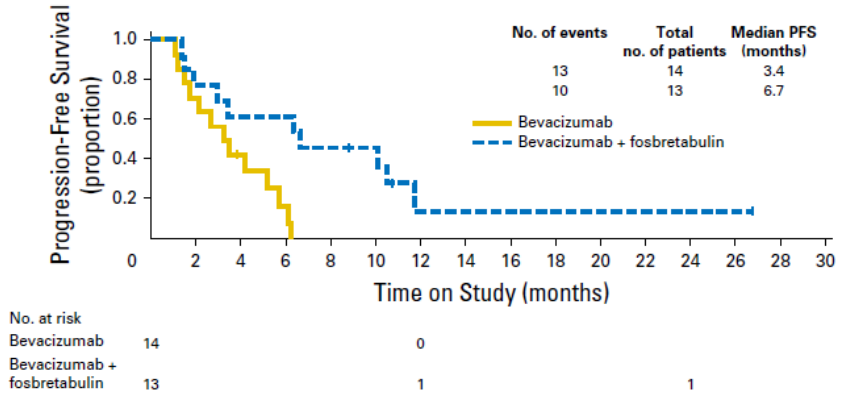
**Exhibit 12: PFS and OS Results from GOG-0186l Study**




*Source: J Clin Oncol. 2016 Jul 1;34(19):2279-86*

When stratified by those patients with platinum resistant disease (n=27), the improvement in median PFS remained significant (6.7 vs. 3.4 months; HR=0.57; Log-rank P=0.01)

**Exhibit 13: PFS Results from Pt-Resistant Patients in GOG-0186l Study**




*Source: J Clin Oncol. 2016 Jul 1;34(19):2279-86*

The combination was well tolerated. Consistent with the known safety profile of CA4P, there was a higher incidence of Grade 3+ AEs due to hypertension, and approximately a third of patients required dose reductions due to cardiovascular events. Most were classified as Grade 3 (see table below), although there was one Grade 4 event (one Grade 5 event was not classified as related to study treatment). Hypertension is a known dose-limiting toxicity associated with CA4P, although prophylactic use of hypertension medications has been implemented to minimize severity.

**Exhibit 14: Safety Profiles from GOG-0186I Study**

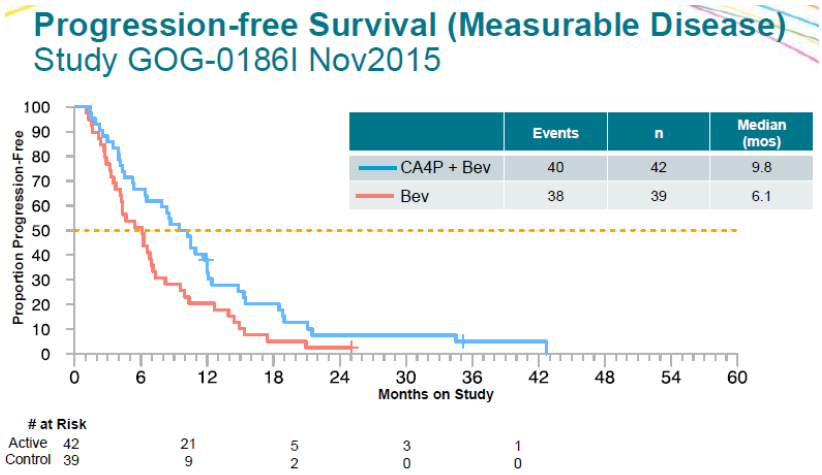
| Site | No. of Events for Bevacizumab-Only Arm | | | | No. of Events for Bevacizumab + Fosbretabulin–Arm | | | |
|---|---|---|---|---|---|---|---|---|
| | Grade 2 | Grade 3 | Grade 4 | Grade 5 | Grade 2 | Grade 3 | Grade 4 | Grade 5 |
| Blood/lymphatics | 7 | 0 | 0 | 0 | 3 | 1 | 0 | 0 |
| GI | 10 | 6 | 0 | 0 | 12 | 10 | 0 | 1* |
| General/administration site | 13 | 2 | 0 | 0 | 17 | 3 | 0 | 0 |
| Infections/infestations | 19 | 3 | 0 | 0 | 18 | 3 | 0 | 0 |
| Investigation site | 11 | 4 | 0 | 0 | 6 | 5 | 0 | 0 |
| Metabolism/nutrition | 11 | 7 | 1 | 0 | 7 | 11 | 0 | 0 |
| Musculoskeletal/connective tissue | 14 | 2 | 0 | 0 | 6 | 2 | 0 | 0 |
| Nervous system | 6 | 1 | 0 | 0 | 7 | 3 | 0 | 0 |
| Renal/urinary | 5 | 0 | 0 | 0 | 4 | 2 | 0 | 0 |
| Respiratory/thoracic/mediastinal | 10 | 2 | 0 | 0 | 5 | 1 | 0 | 0 |
| Vascular disorders | 13 | 10 | 0 | 0 | 13 | 18† | 1 | 0 |

*Grade 5 GI was gastric obstruction, considered not related to treatment.
†Includes 17 for hypertension and one for a thromboembolic event.

*Source: J Clin Oncol. 2016 Jul 1;34(19):2279-86*

Also consistent with preclinical studies, CA4P's effect appeared to be more pronounced in measurable lesions. More patients with measurable disease who were treated with bevacizumab plus CA4P responded to treatment (28.2% for bevacizumab [90%CI, 16.7%to 42.3%] vs. 35.7% for the combination [90% CI, 23.5% to 49.5%]). This difference was not statistically significant. However, when stratified by patients with measurable disease, median PFS significantly improved (see below).

**Exhibit 15: PFS Results from Patients with Measurable Disease in GOG-0186I Study**




| Treatment Comparison | HR | 95% CI | Log-rank P Value |
|---|---|---|---|
| Active vs. Control | 0.600 | [0.38, 0.95] | 0.027 |

*Source: Mateon Company Presentation, February 2017*

**FOCUSed on the best path to registration**

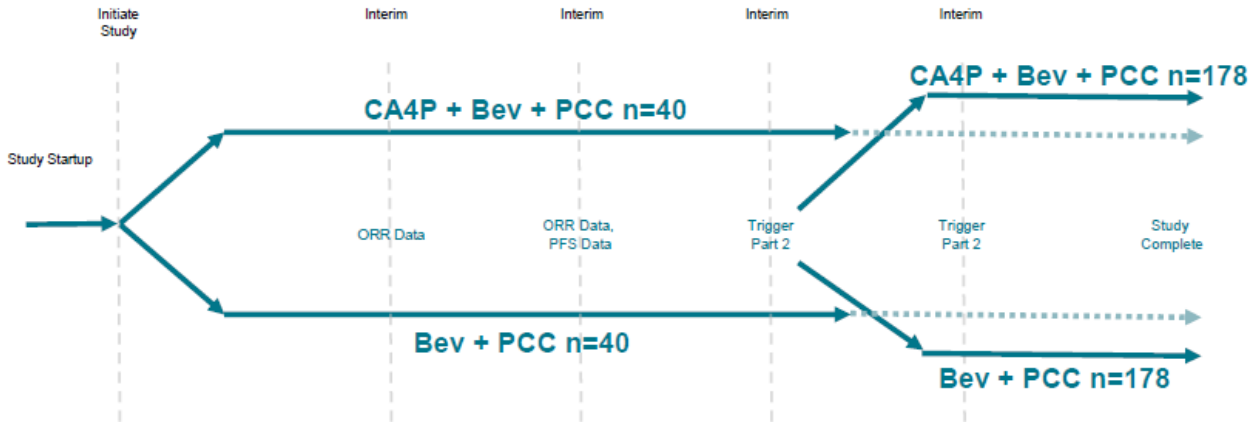
While the GOG study was being conducted, the standard of care for platinum-resistant patients changed in late 2014 when bevacizumab was approved in combination with a physician's choice of chemotherapy (paclitaxel, pegylated liposomal doxorubicin or topotecan) based on the results of the AURELIA study. That study enrolled 360 patients and demonstrated the addition of bevacizumab to chemotherapy reduced the risk of disease worsening or death by 62% compared to women who received chemotherapy alone (median PFS: 6.8 vs. 3.4 months, Hazard Ratio (HR)=0.38;

p<0.0001). Of note, despite the improvement in PFS, overall survival was not enhanced. Based on the improvement in median PFS alone, the combination was approved by the FDA in patients with platinum resistant disease.

To adapt to the new standard of care, Mateon's management team decided to focus CA4P development efforts in combination with bevacizumab and chemotherapy. Although the triplet had yet to be investigated in ovarian cancer, and we generally are cautious on jumping into large scale studies without sufficient Phase 2 support, we believe that the FOCUS trial has been designed with multiple pre-planned analyses of efficacy to support its design.

The FOCUS trial is a Phase 2/3 adaptively-designed study that incorporates a go/no-go decision prior to triggering the Phase 3 portion. The trial is designed to utilize Bayesian inference statistics on early response rates and PFS to potentially modify powering assumptions. These pre-planned efficacy analyses also give investors something to look forward to prior to topline readout.

**Exhibit 16: Design of Phase 2/3 Pivotal FOCUS Trial**




*Source: Mateon Company Presentation, February 2017*

The first interim readout (n=20) was announced in April 2017. Two of the nine patients (22%) treated with the CA4P-containing combination exhibited a PR at this time point, compared to one of 11 (9%) among bevacizumab + chemotherapy only-treated patients. In our view, the safety profile of the triplet combination is consistent with previous trials. As in the GOG trial, the most common AEs included vascular and gastrointestinal symptoms. We anticipate that three additional interim readouts (ORR and PFS, n=40, 60 and 80, respectively) will become available in 2H17. If the fourth and final planned interim analysis of the Phase 2 portion of the study is successful (n=80), we believe the study could advance into a randomized Phase 3 portion including over 300 patients. In our discussions with management, we believe an improvement in ORR could be used as a basis to trigger initiation of the Phase 3 portion. For example, the AURELIA trial had a 27% response rate, and a 50% improvement in response rate in the CA4P arm would equate to approximately a 35-40% response rate.

### FALCON Study points to safety in FOCUS triple combination

Perhaps the most important takeaway from the FALCON study was the tolerability of CA4P when combined with anti-VEGF + chemotherapy, given the same regimen is being studied in the pivotal FOCUS trial. As a reminder, FALCON investigated the utility of CA4P + bevacizumab + carboplatin and paclitaxel (CA4P/B/C/P) in the front line NSCLC indication. Importantly, the toxicities of each individual component of the combination did not appear to overlap. Although the proportion of patients who required dose adjustments was higher in the CA4P combination arm compared to the control arm (52% versus 35%), these dose reductions were due to cardiovascular events arising from the addition of CA4P to the mix. Overall, Grade 3-4 treatment-related AEs were similar in both arms, although lower degree (Grade 1-2) neutropenia was higher when CA4P was added. Equal numbers of patients in the active and control arms received growth factors for neutropenia. The cumulative study drug doses received in each arm are summarized below.

**Exhibit 17: Triple Combination Doses Used in FALCON Trial**

| Study drug (mg) | CA4P group | | Control group | |
|---|---|---|---|---|
| | Treatment phase | Maintenance phase | Treatment phase | Maintenance phase |
| CA4P | 1,317.1±679.2 (n=30) | 2,528.3±2,971.0 (n=13) | NA | NA |
| Carboplatin | 2,908.8±1,284.0 (n=31) | NA | 3,146.3±1,213.4 (n=29) | NA |
| Paclitaxel | 1,666.1±726.6 (n=31) | NA | 1,735.9±647.2 (n=29) | NA |
| Bevacizumab | 5,473.8±2,544.2 (n=31) | 8,994.2±8,710.0 (n=13) | 5,376.7±2,524.6 (n=29) | 12,944.6±10,870.0 (n=17) |

**Note:** Data shown as mean ± SD.
**Abbreviations:** CA4P, combretastatin A4-phosphate; NA, not applicable; SD, standard deviation.

*Source: Onco Targets Ther. 2016; 9: 7275–7283.*

## Putting VDAs to Work in Liquid Tumors

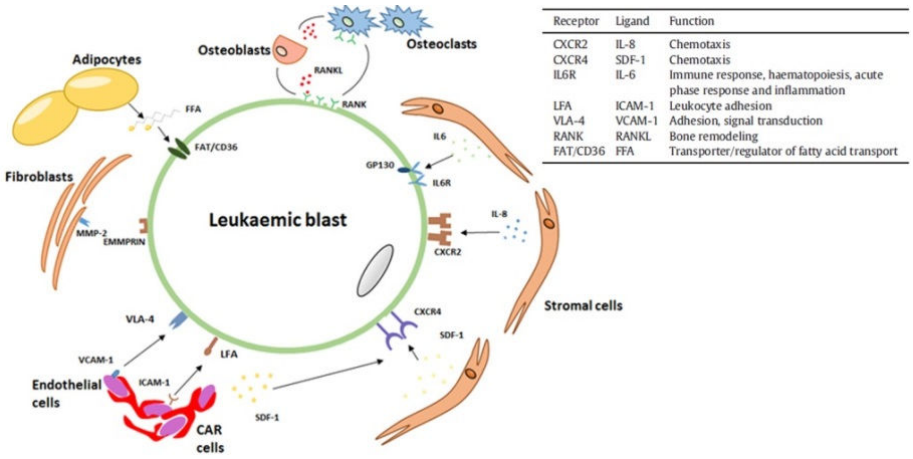
### Why use a vascular disrupting agent in a liquid tumor?

While the development of CA4P has largely centered on solid tumors, Mateon has advanced a second VDA, OXi4503, into the clinic for AML. In our view, investors should understand the mechanistic rationale for testing a VDA in hematologic malignancies.

Treatment resistance (15-20%) and relapse (40%) are a major problem in AML (Ann Hematol 2003 82:231–235), and lead to poor five-year survival (10% for previously-relapsed patients) despite relatively high CR rates (*Curr Treat Options Oncol* 2017 18(3):17).

In hematologic malignancies such as AML, the VDAs can serve as chemo-sensitizing agent by disrupting the interaction between endothelial cells of the bone marrow vasculature and AML blasts, one of the known resistance mechanisms for these tumors. Endothelial cells in the bone marrow contribute to AML resistance by maintaining reservoirs of AML blasts in the bone marrow (known as minimal residual disease, MRD) (*Curr Hematol Malig Rep* 2015 10(2):126-31). Endothelial-derived survival signals include SDF1 chemokine (CXCL12), which binds CXCR4 on blast cells, as well as the cell surface molecules VCAM-1 (binds the $\alpha_4\beta_1$ integrin VLA-4), ICAM-1 (binds LFA) and E-selectin (binds CD44 and ESL-1), which promote both AML cell survival and adhesion to endothelial cells (see below). In our view, a VDA that targets bone marrow endothelial cells could potentially disrupt these protective interactions, helping to overcome therapy resistance and decrease AML recurrence.

**Exhibit 18: AML Blast Interactions with Bone Marrow Microenvironment**




| Receptor | Ligand | Function |
|---|---|---|
| CXCR2 | IL-8 | Chemotaxis |
| CXCR4 | SDF-1 | Chemotaxis |
| IL6R | IL-6 | Immune response, haematopoiesis, acute phase response and inflammation |
| LFA | ICAM-1 | Leukocyte adhesion |
| VLA-4 | VCAM-1 | Adhesion, signal transduction |
| RANK | RANKL | Bone remodeling |
| FAT/CD36 | FFA | Transporter/regulator of fatty acid transport |

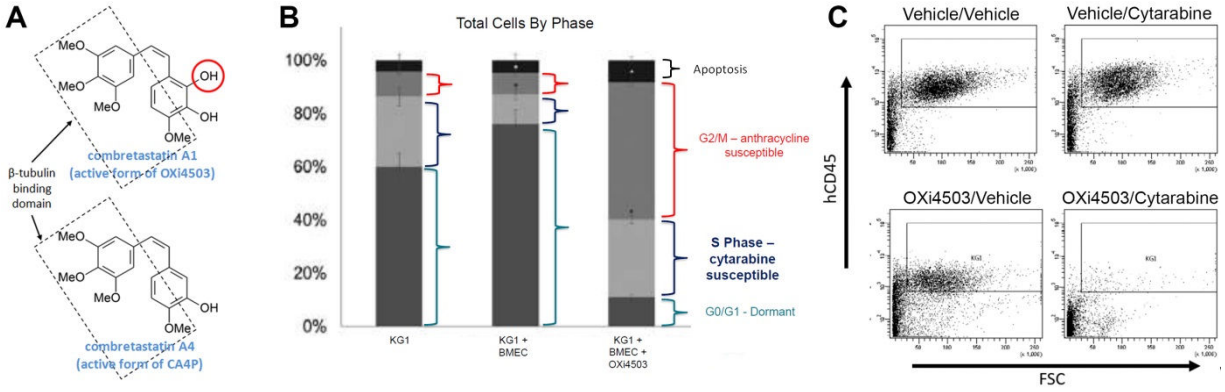
*Source: Blood Rev. 2017 Mar 12. pii: S0268-960X(16)30073-X*

Of note, AML blasts also receive support from other, non-endothelial cells in the bone marrow and there are non-marrow causes of therapy resistance, including upregulation of multi-drug resistance genes (*Best Pract Res Clin Haematol* 2001 14(1):211-33). Additionally, DNA damage repair genes (*Chromosoma* 2014 123(6):545-61), or changes in cytogenetic and molecular profile of the tumor upon relapse (*J Hematol Oncol* 2017 10(1):51) can exert protective effects. One advantage of Mateon's approach is that it is agnostic to the type of mutations present and could thus, in theory, be applicable to more AML patients.

### OXi4503 mechanism of action in AML

OXi4503 and CA4P are nearly identical, differing only by OXi4503's second phenolic moiety (see below), thought to confer higher cytotoxic activity (*Chem Res Toxicol* 2007 20(12):1885-94; *Microvasc Res* 2011 81(1):44-51). Pre-clinical experiments have shown that co-culturing human AML and bone marrow endothelial cells (BMECs) causes a significant reduction in the number of actively dividing AML cells and an increase in dormant (G0/G1) cells (*Exp Hematol* 2016 44(5):363-377). This effect was reversed in the presence of OXi4503, which also caused AML and BMEC cells to detach and decreased BMEC cell membrane localization of adhesion proteins known to mediate this interaction (VCAM-1, VE-cadherin, BCAM). Given these results, the company evaluated the anti-tumor effect of combining OXi4503 with various chemotherapies used in AML (cytarabine, anthracyclines) and found both *in vitro* and in mouse models that the combination was more effective than either therapy alone.

**Exhibit 19: OXi4503 (A) Prevents AML Cell Quiescence (B) and Sensitizes Them to Cytarabine (C)**




*Source: Mateon Therapeutics Presentation at 2017 ROTH Conference; Exp Hematol 2016 44(5):363-377*

We believe these results indicate that OXi4503 acts as a chemo-sensitizing agent by disrupting AML-stromal interactions and returning leukemic cells to the cell cycle. However, we note that OXi4503 has other potentially relevant activities, such as direct induction of apoptosis in AML cells and decrease in bone marrow microvessel density (*Blood* 2010 116(9):1539-47), as evidenced by its effects as a monotherapy. While we believe it remains unclear which of these activities is predominant *in vivo*, we believe that the preclinical evidence justifies the combination of OXi4503 with cytarabine as a therapy for AML.

The strategy of sensitizing dormant AML cells to chemotherapy by stimulating their proliferation is not new and is the reason for adding GM-CSF or other myeloid growth factors to AML induction regimens. We note that this strategy has had mixed results (*Blood* 1994 84 (10 Suppl 1): A-95, 27a; *J Clin Oncol* 1997 Dec;15(12):3496-506; *Blood* 1998 91(8):2722-30). Additional drugs attempting to reverse AML chemo-resistance by disrupting protective bone marrow interactions, including several CXCR4 inhibitors, adhesion molecule inhibitors, and hypoxia-inducible agents are currently being evaluated in the clinic (*Ther Adv Hematol* 2016 Feb; 7(1): 40–51). We note that AML remains an indication area of high unmet need; with the exception of Pfizer's (PFE-NC) gemtuzumab ozogamycin (Mylotarg, withdrawn from the market), no new drugs have been approved for the treatment of AML for over 50 years (*Curr Treat Options Oncol* 2017 18(3):17).

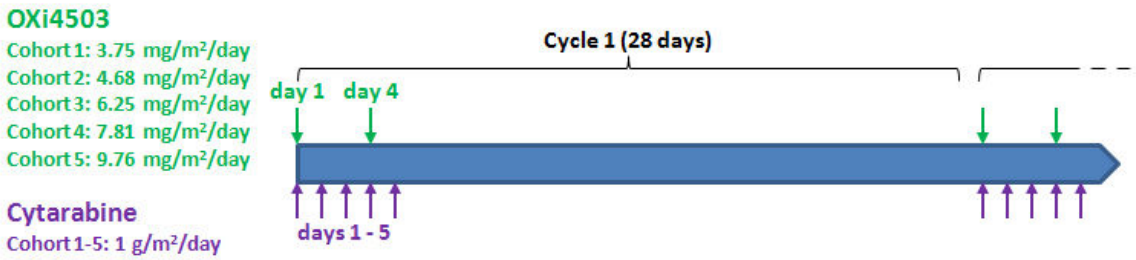
### OXi4503 shows signs of anti-AML activity in the clinic

An investigator-sponsored Phase 1 trial was initiated in 2011 to determine the safety and maximum tolerated dose (MTD) of OXi4503 monotherapy in relapsed/refractory (rr) AML and MDS patients (NCT01085656). The trial enrolled 19 patients who had received 1-5 prior therapies. The drug was administered intravenously at once weekly doses between 2.5 and 7.81 mg/m$^2$. One patient (5%) treated with the lowest dose achieved a marrow CR after one month of treatment and then died of pneumonia. Another patient (5%) treated with 5 mg/m$^2$ for 10 months achieved a partial remission (PR) (Blood 2013 122(21):1463).

The most significant drug-related grade ≥3 adverse events (AEs) were bone pain (39%), flu-like symptoms (28%), febrile neutropenia (28%) and blood clotting-related imbalances (D-dimer elevation, disseminated intravascular coagulation (DIC), increased INR, thrombocytopenia, and PTT prolongation). The MTD was never reached and the protocol was significantly amended, giving rise to a new, Phase 1b/2 company-sponsored trial named OX1222 (NCT02576301).

The ongoing OX1222 trial is designed to identify the MTD of OXi4503 in combination with cytarabine (Phase 1b primary outcome) and the overall response rate (ORR) of the combination therapy in rrAML and MDS patients (Phase 2 primary outcome). In Phase 1b, OXi4503 will be administered to five successive cohorts with doses ranging from 3.75 to 9.76 mg/m$^2$ twice monthly combined with an intermediate cytarabine dose (1 g/m$^2$/day) for five days (see Exhibit 20).

**Exhibit 20: OX1222 Trial Design**




*Source: ROTH Capital Partners research*

Enrollment in the first three cohorts has been completed and interim results (13 patients) were presented in 1Q17. Thus far, three patients achieved CRs (23%), one in each cohort, and the safety profile is markedly improved compared to the earlier Phase 1 trial (Exhibit 22). One dose-limiting toxicity (DLT) occurred in cohort 1 (pan-cytopenia). The fourth cohort (7.81 mg/m$^2$) is now enrolling and we believe that full results from Phase 1b may be presented in 2H17. Pending positive results from Phase 1b, Mateon has guided that it will seek a partner to advance into Phase 2 of the trial, which would include ~80 patients and two cohorts (rrAML and MDS).

### Efficacy of OXi4503/cytarabine combination compares favorably with current rrAML therapies

OXi4503 monotherapy achieved only one marrow CR (5%). Combination with cytarabine has substantially increased efficacy and three CRs (23%) have been reported so far. Achieving CR in rrAML patients is not uncommon and current methods range from 25% to 65% (*Curr Treat Options Oncol* 2017 18(3):17; *Blood* 2015 126(3):319-27; *Am J Hematol* 2009 84:733–737) (Exhibit 21), placing Mateon's experimental combination on the low end of the efficacy range. However, the specific age, molecular and in particular cytological profile of patients can have a significant influence in the outcome. We note that patients enrolled in Mateon's trial are heavily pre-treated (averaging four prior therapies) and more than half have cytogenetic markers of poor prognosis (trisomy 8, inv(3), p53 deletion, monosomy 17 and complex cytogenetics), normally associated with CR rates of 32% (*Blood* 2002 Dec 15;100(13):4325-36; *Leukemia* 2009 23:656–663). Encouragingly, all three patients that achieved CR in Phase 1b have poor prognosis cytogenetic markers. Additionally, we believe that future updates of OX1222 trial could lead to further CR

improvement: one patient in cohort 3 (6.25 mg/m$^2$) with blast reduction is still undergoing treatment, and greater efficacy could be seen in the remaining two cohorts with higher OXi4503 doses (7.81 and 9.76 mg/m$^2$). In our opinion, durability of response will also be an important measure of efficacy, as relapse is common in AML. Of the three CRs, two are still ongoing (one at nine months) and a third recurred after six months. These results are on par with some of the current salvage therapies (see Exhibit 21).

**Exhibit 21: Comparison of Select AML Salvage Regimens**

| Regimen | *N* | Median age | %CR1 less than 1 year | Prior allo (%) | Poor-risk cytogenetics (%) | CR (%) | CRp (%) | Median DOR/EFS (months) | Median OS (months) |
|---|---|---|---|---|---|---|---|---|---|
| FLAG-I | 23 | 48 | 60 | 43 | 57 | 39 | 13 | 16.8/7.4 | 8.8 |
| FLAG-IM | 48 | 47 | 67 | 42 | 35 | 29 | 27 | 8.3/4.1 | 5.0 |
| IDAC [4] | 62 | NR | 66 | 0 | 29 | 40 | NR | NR | 4.5 |
| HDAC [4] | 52 | | | | | 40 | NR | | |
| CLAG-M [8] | 114 | 45 | 92 | 4 | 25 | 58 | NR | 17.0 | 9.0 |
| MIDAM [28] | 62 | 56 | 37 | 3 | 18 | 50 | 13 | NR/4.4 | 9.5 |
| FLAG-IDA [29] | 46 | 41 | 100 | 4 | 33 | 52 | NR | 12.0/NR | 11.0 |
| MEC [7] | 32 | 24 | NR | 19 | NR | 66 | NR | 4.0/NR | 9.0 |
| MEC [6] | 50 | 37 | NR | 0 | NR | 68 | NR | 12.0/6.0 | 9.0 |
| MEC [15] | 97 | 55 | 100 | 6 | NR | 23 | 5 | NR | 8.0 |
| Clofarabine [20] | 31 | 54 | 52 | 3 | 29 | 55 | 13 | 6.0/NR | 6.0 |
| Clo + AraC [19] | 25 | 59 | 55 | NR | NR | 24 | 17 | NR | 5.5 |

*N*, number of patients; IDAC, intermediate-dose cytarabine; HDAC, high-dose cytarabine; CR, complete remission; CRp, complete remission with incomplete platelet recovery; EFS, event-free survival; OS, overall survival; DOR, duration of response; NR, not reported.

*Source: Am J Hematol 2009 84:733–737*

### OXi4503/cytarabine combination is safe but leaves little room for maneuvering

Despite the inclusion of cytarabine, severe AEs have been much lower in the OX1222 combination trial than what was observed in the OXi4503 monotherapy trial. In our view, this difference could be due to the twice monthly rather than once weekly dosing of OXi4503, though the slightly lower doses tested thus far in OX1222 could also be a factor (up to 6.25 mg/m$^2$ vs. 7.81 mg/m$^2$). In early trials using CA4P in solid tumors, serious cardiovascular side effects were detected in some patients (*Cancer Res* 2002 62(12):3408-16; *Clin Cancer Res* 2004 10(1 Pt 1):96-100), that were subsequently managed with calcium channel blockers. Thus far, one patient treated with OXi4503 (N=32) suffered from grade ≥3 tachychardia, considered therapy-unrelated. Based on the markedly lower drug doses in AML trials (~4-10 mg/m$^2$/day OXi4503 vs. 60-90 mg/m$^2$/day CA4P), we do not believe cardiac AEs will become a major problem for OXi4503.

**Exhibit 22: Efficacy and Safety of Approved and Experimental Therapies for rrAML and MDS**

| | OXi4503 | OXi4503/Cyt.* | Mido/Aza | Enasidenib | FLAG** | FLAG-IDA** | MEC* |
|---|---|---|---|---|---|---|---|
| **Development Status** | Phase 1 | Phase 1b/2 | NDA | NDA | Approved | Approved | Approved |
| **N Number** | 18 | 14 | 54 | 209 | 6 | 23-46 | 32-74 |
| **Complete Remission** | 5% (CRm) | 23% | 2/13% (CR/CRi) | 18/27% (CR/CRi) | 55% | 39-52% | 55-66% |
| **Bone pain** | 39% | | | | | | |
| **Febrile neutropenia** | 28% | 21% | | 26% | 100% | 100-59% | 47% |
| **Thrombocytopenia** | 28% | | 94% | 12% | | Yes# | 100% |
| **Neutropenia** | 11% | 7% | 96% | | | 100% | 100% |
| **Leukopenia** | 22% | | | | | | |
| **Anemia** | 11% | | 61% | 12% | | | |
| **Infections/Septicemia** | | 14% | 56% | | Yes# | 9-30% | 44% |
| **High D-dimer/thrombosis** | 83% | | 2% | | | | |
| **DIC/INR increase** | 28% | | | | | | |
| **Hemorrhage** | | 7% | 2% | | Yes# | 2% | 6% |
| **Liver** | 17% | | 4% | 10% | Yes# | 22-0% | 3-38% |
| **Cardiac** | | 7% | 11% | | | 0% | |
| **Mucositis** | | 7% | | | Yes# | 65% | 13% |
| **Nausea/Vomiting** | 11% | | 9% | 3% | Yes# | 0% | 22% |

*1 and **2 mg/m$^2$/day cytarabine (5 days); Yes#: common but unspecified grade; CRm: marrow CRi; CR with incomplete hematologic recovery; Cyt: cytarabine; DIC: disseminated intravascular coagulation; FLAG: Fludarabine+Cytarabine+G-CSF; FLAG-IDA: FLAG+idarubicin; MEC: mitoxantrone+etoposide+cytarabine; Mido/Aza: midostaurin/5-azacytidine; mitoxantrone+etoposide+cytarabine

*Source: ROTH Capital Partners research; Indian J Hematol Blood Transfus 2014 30(4):231-5; Blood 2015 126(3):319-327; Am J Hematol 2015 90(4):276-81; Agio R&D Day December 2015*

Overall, we believe the OXi4503/cytarabine combination has an acceptable AE profile, especially compared to common rrAML salvage therapies such as FLAG, FLAG-IDA or MEC (Exhibit 22). Nonetheless, we note that FLAG, FLAG-IDA and MEC all cause significant grade ≥3 neutropenia, thrombocytopenia, infections and/or bacteremia/septicemia, which also occur with OXi4503. We believe this overlap could limit the ability to combine OXi4503 with these common chemotherapeutic regimens or to significantly increase the dose of either OXi4503 or cytarabine in the pursuit of higher efficacy. Therefore, we do not ascribe value to Mateon's OXi4503 program in our estimates at this time.

## Key Model Assumptions

**Revenues**

Given CA4P's current development stage, we believe the drug could receive approval from U.S. regulators in 2021. Assuming an annual treatment price of $60,000, we estimate the U.S. market size for CA4P could reach $266M by 2025, the basis year of our valuation. Assuming moderate market penetration in Pt-resistant ovarian cancer patients, we estimate peak sales of $150M, yielding probability-adjusted (50%) sales revenue of $12M in 2021, increasing to $63.6M in 2025.

**Operating Expenses**

We are introducing the R&D estimate of $9.7M for 2017, ramping to $14.1M in 2025. The R&D assumptions from 2017 through 2025 take into account continued expenditure for clinical development of CA4P in Pt-resistant ovarian cancer and OXi4503 in AML. We believe the ongoing 80-patient Phase 2 segment of the FOCUS trial could advance to the 356-patient Phase 3 segment next year, and we could see a PFS readout from the trial in 2020.

We are introducing the SG&A estimates of $4.7M for 2017, increasing to $15.5M in 2025. These estimates include an increase associated with hiring a 25-person sales force. We assume COGS for CA4P will be 10% of sales revenue.

**Net Income and EPS**

We are introducing net income (loss) estimates of $(14.4)M or $(0.41) per share in 2017, ramping to $22.4M or $(0.31) per share in 2025, with the company potentially achieving sustained profitability in 2023.

**Cash**

Mateon Therapeutics had approximately $12.0M in cash and cash equivalents at the end of 4Q16. We estimate the company has sufficient resources to sustain operations until 4Q17. We have included a series of four dilutive cash raises in our model in 2017, 2018, 2019, and 2020, which could sustain operations until the company potentially achieves sustained profitability in 2023.

## Management Team

**William Schwieterman, M.D.**
President and Chief Executive Officer

Dr. Schwieterman, is President and Chief Executive Officer of Mateon Therapeutics. Dr. Schwieterman is a rheumatologist and board-certified internist, with more than 15 years of executive, regulatory, and drug development experience in the biopharmaceutical industry. He has been an independent consultant to multiple biotech and pharmaceutical clients where he helped them create their regulatory and clinical development strategies. Before joining Mateon, he was an employee of Perceptive Advisors, LLC, a hedge fund based in New York, New York. Dr. Schwieterman was also the Chief Medical Officer of Chelsea Therapeutics, Inc., acquired by Lundbeck (LUN-NC) in 2014, where he led the clinical development team toward the approval of droxidopa (Northera) for the treatment of symptomatic neurogenic orthostatic hypotension, a symptom associated with Parkinson's disease and other neurodegenerative 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). In these capacities and others, 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. Dr. Schwieterman holds a BS and MD from the University of Cincinnati.

**Matthew Loar**
Chief Financial Officer

Mr. Loar is the Chief Financial Officer of Mateon, bringing more than 20 years of experience as a financial executive with financial and accounting experience in both public and private companies in the biopharmaceutical industry. Mr. Loar was most recently CFO of KineMed, Inc., from January 2014 to July 2015. From January 2010 to January 2014, he was an independent financial consultant to companies in the biopharmaceutical industry. While consulting, he also served as acting Chief Executive and Financial Officer of Neurobiological Technologies, Inc. (NTI), beginning in February 2010, and as CFO of Virolab, Inc., from May 2011 to August 2012. Previously, he was CFO of NTI from April 2008 to December 2009. Earlier in his career, Mr. Loar was CFO of Osteologix, Inc., from 2006 to 2008 and of Genelabs Technologies, Inc., from 1995 to 2006. He received a B.A. in legal studies from the University of California, Berkeley, and is a certified public accountant (inactive) in California.

**David Chaplin, Ph.D.**
Chief Scientific Officer

Dr. Chaplin is Chief Scientific Officer of Mateon, bringing more than 30 years of experience in oncology research and drug development. In addition to Dr. Chaplin's current role as CSO and Head of Research and Development at Mateon, he has also served as President and Chief Executive Officer. He has been a leader in the field of vascular targeting and was the first to discover the vascular disrupting action of CA4P (combretastatin A4-phosphate or fosbretabulin). His original work formed the basis for not only the development of CA4P with Mateon, but also the vascular targeting development programs at AstraZeneca (AZN-NC) and Aventis (SNY-NC). 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. Dr. Chaplin also served as Section Head of Cancer Biology at Xenova in the United Kingdom and held a senior staff appointment at the British Columbia Cancer Research Centre. Educated in the United Kingdom, Dr. Chaplin has a B.Sc. in chemistry from the University of Essex, a M.S.c in pharmacology from the University of Southampton, and a Ph.D. in tumor biology from the University of London.

**Jeff Nelson**
Vice President, Program Management

Mr. Nelson joined Mateon in September 2015 as Vice President, Program Management, and brings more than 10 years of financial and operational experience in the biopharmaceutical industry. Most recently, Mr. Nelson was Senior Director of Clinical Operations at Axsome Therapeutics, Inc. (AXSM-NC). From 2009 to 2014, he served as Senior Director of Business Development and Strategic Planning at Chelsea Therapeutics, Inc. From 2005 to 2009, he was Vice President, Equity Research at Ladenburg Thalmann Financial Services Inc., covering the biotech sector. Previously Mr. Nelson was Manager, Products and Business Development at Cobalt Laboratories, the U.S. division of the Arrow Group, an international consortium of generic pharmaceutical companies. Mr. Nelson received a B.A. from

## VALUATION

We value Mateon Therapeutics using a discounted earnings per share and revenue multiple analysis. Given the current stage of development of CA4P, we project a potential commercial launch in 2021.

Profitable biotechnology companies have historically traded in a multiple range of 6-10x in revenues. For reference, an average of five profitable biotech companies with marketed products in oncology indications (including CELG-NC; AMGN-NC; SHPG-NC; NVS-NC; JNJ-NC) recently traded at an average of 5.4x sales revenue. We value Mateon Therapeutics using a revenue multiples analysis. Applying an 8x multiple to our probability-adjusted 2025 revenue estimate of $63.6M and discounting by 20% over eight periods, we obtain a $1.66 target price.

Profitable biotechnology companies have historically traded in a multiple range of 26-30x in EPS. For reference, an average of five profitable biotech companies with marketed products in oncology indications (including CELG; AMGN; SHPG; NVS; JNJ) recently traded at an average of 26.7x trailing EPS. We also value Mateon Therapeutics using a discounted earnings multiple analysis. Applying a 28x multiple to our probability adjusted 2025 EPS of $0.31 and discounting by 20% over eight periods, we obtain a $2.05 target price.

Averaging the results from these two methods and adding the projected cash per share in 12 months, we obtain a 12-month price target of $1.97 per share, which we round to $2.

Impediments to our price target include negative trial results and the possibility of changes in the standard of care in the therapeutic indications that Mateon operates.

## RISKS

· **Financing risk.** As with a majority of development-stage biotechnology companies, the ability to maintain sufficient funding is critical to the progress of pipeline candidates. The company has guided that its current cash position can support operations until October, 2017. Mateon may need to seek additional dilutive financing options via the capital markets. Recent delisting from The NASDAQ Capital Market may significantly impair Mateon's ability to raise capital. Should Mateon experience problems raising additional capital, its development programs' progress could be significantly impeded, leading to both delays in development timelines as well as potential negative effects on investor confidence. Each of these could have a negative impact on the share price.

· **Clinical and regulatory risk.** Mateon's future success depends on its ability to develop and commercialize new products in a timely and cost-effective manner. We believe that the company is on track to meet its milestones. However, Mateon may experience delays in initiation, enrollment, or completion of its clinical trials and regulatory authorities could impose additional conditions on these trials. Additionally, results from ongoing clinical trials may not be predictive of the results of future pivotal clinical trials. While we believe CA4P and OXi4503 have shown encouraging results in early studies, we acknowledge that both ovarian cancer and AML are challenging indications and that multiple competitor products are in development for both.

· **Commercial risk.** The company's therapeutic candidates, including CA4P, may not obtain the market penetration and sales forecasted by our estimates or those of the company given the competitive marketplace and pricing dynamics in place in the U.S. and E.U. If approved, CA4P may be subject to a narrow label that would restrict use to a smaller-than-anticipated pool of addressable patients. Further, the company currently lacks a sales force or distribution capabilities, and therefore may be unable to commercialize approved products successfully.

· **Reliance on third parties.** As of March 2017, Mateon has only 16 full-time employees and relies heavily on CROs and other third parties for clinical trial activities and outsources all of its manufacturing. Third-party CROs and manufacturers may not be able to meet the company's needs with respect to timing, quantity or quality. If Mateon is unable to contract for a sufficient supply of needed materials on acceptable terms, or

encounters delays or difficulties in its relationships with manufacturers or CROs, the company's clinical testing may be delayed, thereby delaying the submission of products for regulatory approval or the market introduction and subsequent sales of its products.

· **Intellectual Property and Licensing Risk.** The commercial success of Mateon Therapeutics depends on its ability to develop, manufacture, and commercialize proprietary technologies without infringing on the proprietary rights of third parties. Any loss of license rights to use certain critical intellectual property from licensors could have a material adverse effect on the company's business. Mateon has in-licensed exclusive worldwide rights to CA4P and OXi4503 from Arizona State University and Bristol-Myers Squibb (BMY-NC) and does not own any intellectual properties or technologies. If CA4P is approved, Mateon will be required to pay low-single-digit royalties on future net sales of products associated with the BMS patent rights until these patent rights expire. Excluding extensions and orphan exclusivity, we believe composition of matter patents on CA4P and OXi4503 expire in 2021, and a method patent on OXi4503 expires in 2028. Mateon has filed a method patent on the use of CA4P which, if approved, would expire in 2036.

## COMPANY DESCRIPTION

Mateon Therapeutics (formerly OXiGENE) was founded and incorporated first in New York in 1988 and then Delaware in 1992, with an IPO in August 1993. Mateon's corporate headquarters are currently located in South San Francisco, CA. As of March 2017, Mateon had a total of 16 full-time employees. The company relies on outsourcing for much of its research, development, preclinical testing and clinical trial activity, but maintains managerial and quality control over its clinical trials. Mateon is focused on the development of vascular disrupting agents (VDAs) for the treatment of cancer. Mateon's clinical-stage pipeline includes two VDAs: CA4P, also known as fosbretabulin, and OXi4503. CA4P is currently being investigated in a Phase 2/3 pivotal trial (FOCUS) in platinum-resistant ovarian cancer, while OXi4503 is being investigated in a dose-escalation Phase 1/2 trial in acute myeloid leukemia (AML). Based on evidence from prior clinical trials of CA4P and OXi4503, it appears that VDAs have anti-tumor activity and synergize with chemotherapy and anti-angiogenic drugs.

| *Figures in $ thousands except per share data* | 1Q16 | 2Q16 | 3Q16 | 4Q16 |
|---|---|---|---|---|
| **ASSETS** | | | | |
| Cash and cash equivalents | 10,276 | 7,939 | 5,167 | 3,535 |
| Short-term investments | 12,610 | 11,402 | 11,113 | 8,512 |
| Restricted cash | | | | |
| Prepaid expenses | 1,297 | 1,035 | 934 | 1,946 |
| Other current assets | | | | 77 |
| **TOTAL current assets** | 24,183 | 20,376 | 17,214 | 14,070 |
| Property and equipment, net | 25 | 19 | 14 | 11 |
| License agreements | | | | |
| Other non-current assets | 33 | 33 | 33 | 33 |
| TOTAL non-current assets | 58 | 52 | 47 | 44 |
| TOTAL assets | 24,241 | 20,428 | 17,261 | 14,114 |
| **LIABILITIES** | | | | |
| Accounts payable | 475 | 501 | 532 | 310 |
| Accrued clinical trial expenses | 757 | 236 | 46 | 64 |
| Accrued compensation and employee benefits | 459 | 474 | 497 | 842 |
| Accrued other liabilities | 333 | 406 | 410 | 398 |
| TOTAL current liabilities | 2,024 | 1,617 | 1,485 | 1,614 |
| Other long-term liabilities | | | | |
| TOTAL non-current liabilities | | | | |
| **TOTAL liabilities** | 2,024 | 1,617 | 1,485 | 1,614 |
| **STOCKHOLDER'S EQUITY** | | | | |
| Common stock | 265 | 265 | 265 | 265 |
| Additionnal paid-in capital | 290,086 | 290,321 | 290,521 | 290,698 |
| Stock subscription receivable | | | | |
| Accumulated deficit | (268,134) | (271,775) | (275,010) | (278,463) |
| **TOTAL stockholders' equity** | 22,217 | 18,811 | 15,776 | 12,500 |
| **TOTAL liabilities and stockholders' equity** | 24,241 | 20,428 | 17,261 | 14,114 |

ROTH Capital Partners, LLC
Mark Breidenbach, Ph.D.
mbreidenbach@roth.com
646-616-2786

| *Figures in $ thousands except per share data* | *2015A* | *2016A* | *1Q17E* | *2Q17E* | *3Q17E* | *4Q17E* | *2017E* | *1Q18E* | *2Q18E* | *3Q18E* | *4Q18E* | *2018E* | *2019E* | *2020E* | *2021E* | *2022E* | *2023E* | *2024E* | *2025E* |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| **Sales revenues:** | | | | | | | | | | | | | | | | | | | |
| Product sales | | | | | | | | | | | | | | | 12,000 | 21,750 | 34,950 | 50,400 | 63,600 |
| **Total Revenue** | | | | | | | | | | | | | | | 12,000 | 21,750 | 34,950 | 50,400 | 63,600 |
| **Expenses:** | | | | | | | | | | | | | | | | | | | |
| Cost of goods sold (COGS) | | | | | | | | | | | | | | | 1,200 | 2,175 | 3,495 | 5,040 | 7,065 |
| Research & development (R&D) | 9,086 | 8,764 | 2,358 | 2,406 | 2,454 | 2,503 | 9,720 | 2,528 | 2,553 | 2,579 | 2,604 | 10,264 | 10,680 | 11,114 | 11,565 | 12,035 | 11,757 | 11,088 | 14,130 |
| Selling, general & administrative (SG&A) | 4,596 | 4,995 | 1,151 | 1,163 | 1,175 | 1,186 | 4,675 | 1,198 | 1,210 | 1,222 | 1,234 | 4,865 | 5,037 | 5,470 | 8,648 | 10,802 | 10,800 | 11,088 | 15,543 |
| **Total operating expenses** | 13,682 | 13,759 | 3,510 | 3,568 | 3,628 | 3,689 | 14,395 | 3,726 | 3,763 | 3,801 | 3,839 | 15,129 | 15,718 | 16,584 | 21,413 | 25,012 | 26,052 | 27,216 | 36,738 |
| **Income (loss) from operations** | (13,682) | (13,759) | (3,510) | (3,568) | (3,628) | (3,689) | (14,395) | (3,726) | (3,763) | (3,801) | (3,839) | (15,129) | (15,718) | (16,584) | (9,413) | (3,262) | 8,898 | 23,184 | 33,912 |
| Other income (expense) | | | | | | | | | | | | | | | | | | | |
| Gain (loss) on fair value of warrants | | | | | | | | | | | | | | | | | | | |
| Interest income | 27 | 106 | | | | | | | | | | | | | | | | | |
| Other (expense) income, net | 1 | (1) | | | | | | | | | | | | | | | | | |
| **Total other income (expense)** | 28 | 105 | | | | | | | | | | | | | | | | | |
| **Profit (loss) before taxes** | (13,654) | (13,654) | (3,510) | (3,568) | (3,628) | (3,689) | (14,395) | (3,726) | (3,763) | (3,801) | (3,839) | (15,129) | (15,718) | (16,584) | (9,413) | (3,262) | 8,898 | 23,184 | 33,912 |
| Income tax benefit (expense) | | | | | | | | | | | | | | | | | 1,335 | 7,883 | 11,530 |
| Non-cash deemed dividend to preferred stock | | | | | | | | | | | | | | | | | | | |
| **Net profit (loss) attribuable to common stoc** | (13,654) | (13,654) | (3,510) | (3,568) | (3,628) | (3,689) | (14,395) | (3,726) | (3,763) | (3,801) | (3,839) | (15,129) | (15,718) | (16,584) | (9,413) | (3,262) | 7,563 | 15,301 | 22,382 |
| Earnings per share: | | | | | | | | | | | | | | | | | | | |
| Basic | (0.54) | (0.51) | (0.13) | (0.09) | (0.10) | (0.10) | (0.41) | (0.10) | (0.08) | (0.08) | (0.09) | (0.35) | (0.29) | (0.29) | (0.16) | (0.06) | 0.13 | 0.26 | 0.37 |
| Diluted | (0.54) | (0.51) | (0.13) | (0.09) | (0.10) | (0.10) | (0.41) | (0.10) | (0.08) | (0.08) | (0.09) | (0.35) | (0.29) | (0.29) | (0.16) | (0.06) | 0.11 | 0.22 | 0.31 |
| Average shares outstanding | | | | | | | | | | | | | | | | | | | |
| Basic | 25,201 | 26,545 | 26,545 | 37,856 | 38,056 | 38,256 | 35,178 | 38,456 | 44,656 | 44,856 | 45,056 | 43,256 | 53,556 | 56,856 | 58,589 | 57,856 | 59,589 | 58,856 | 60,589 |
| Diluted | 25,201 | 26,545 | 26,545 | 37,856 | 38,056 | 38,256 | 35,178 | 38,456 | 44,656 | 44,856 | 45,056 | 43,256 | 53,556 | 56,856 | 58,589 | 57,856 | 70,254 | 70,754 | 71,254 |

ROTH Capital Partners, LLC

Mark Breidenbach, PhD
mbreidenbach@roth.com
646-616-2786

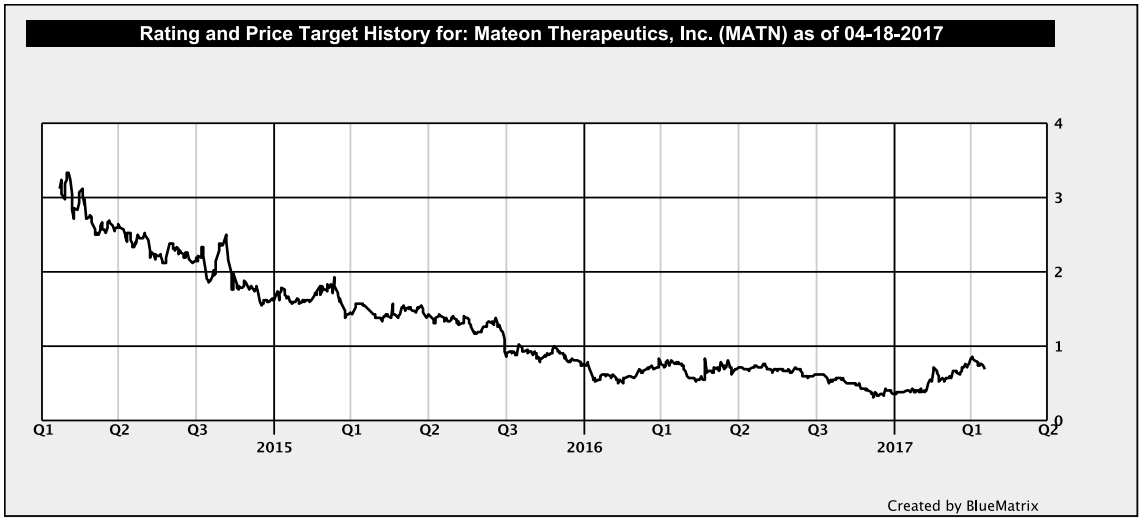
Regulation Analyst Certification ("Reg AC"): The research analyst primarily responsible for the content of this report certifies the following under Reg AC: I hereby certify that all views expressed in this report accurately reflect my personal views about the subject company or companies and its or their securities. I also certify that no part of my compensation was, is or will be, directly or indirectly, related to the specific recommendations or views expressed in this report.

## Disclosures:

ROTH makes a market in shares of Mateon Therapeutics, Inc. and as such, buys and sells from customers on a principal basis.

Shares of Mateon Therapeutics, Inc. may not be eligible for sale in one or more states.

Shares of Mateon Therapeutics, Inc. may be subject to the Securities and Exchange Commission's Penny Stock Rules, which may set forth sales practice requirements for certain low-priced securities.




Each box on the Rating and Price Target History chart above represents a date on which an analyst made a change to a rating or price target, except for the first box, which may only represent the first note written during the past three years. **Distribution Ratings/IB Services** shows the number of companies in each rating category from which Roth or an affiliate received compensation for investment banking services in the past 12 month.

**Distribution of IB Services Firmwide**

| | | | IB Serv./Past 12 Mos. as of 04/19/17 | |
|---|---|---|---|---|
| **Rating** | **Count** | **Percent** | **Count** | **Percent** |
| **Buy [B]** | **224** | **70.00** | **113** | **50.45** |
| **Neutral [N]** | **41** | **12.81** | **21** | **51.22** |
| **Sell [S]** | **7** | **2.19** | **3** | **42.86** |
| **Under Review [UR]** | **47** | **14.69** | **31** | **65.96** |

Our rating system attempts to incorporate industry, company and/or overall market risk and volatility. Consequently, at any given point in time, our investment rating on a stock and its implied price movement may not correspond to the stated 12-month price target.

Ratings System Definitions - ROTH employs a rating system based on the following:

**Buy:** A rating, which at the time it is instituted and or reiterated, that indicates an expectation of a total return of at least 10% over the next 12 months.

**Neutral:** A rating, which at the time it is instituted and or reiterated, that indicates an expectation of a total return between negative 10% and 10% over the next 12 months.

**Sell:** A rating, which at the time it is instituted and or reiterated, that indicates an expectation that the price will depreciate by more than 10% over the next 12 months.

**Under Review [UR]:** A rating, which at the time it is instituted and or reiterated, indicates the temporary removal of the prior rating, price target and estimates for the security. Prior rating, price target and estimates should no longer be relied upon for UR-rated securities.

**Not Covered [NC]:** ROTH does not publish research or have an opinion about this security.