Diagnostics

**TROV** - NASDAQ April 12, 2017

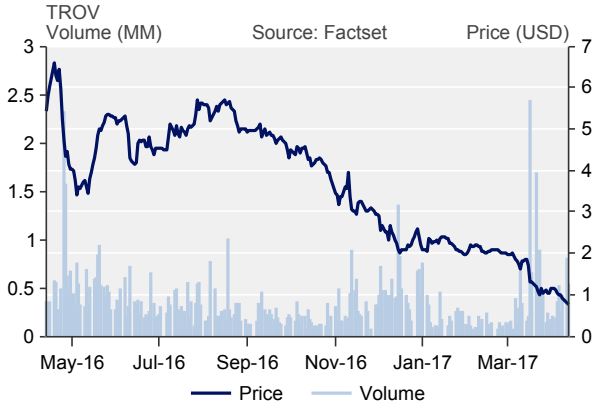
| | |
|---|---|
| **Intraday Price 04/12/2017** | **$0.79** |
| Rating: | *(prior Hold)* Buy |
| 12-Month Target Price: | *(prior NA)* $4.00 |
| 52-Week Range: | $0.65 - $6.67 |
| Market Cap (M): | 24 |
| Shares O/S (M): | 31.0 |
| Float: | 94.5% |
| Avg. Daily Volume (000): | 315 |
| Dividend: | $0.00 |
| Dividend Yield: | 0.00% |
| Risk Profile: | Speculative |
| Fiscal Year End: | December |

**Total Expenses ('000)**

| | 2016A | 2017E | 2018E |
|---|---|---|---|
| 1Q | 10,579 | 7,475 | 7,168 |
| 2Q | 10,084 | 7,800 | 7,480 |
| 3Q | 10,013 | 8,450 | 8,103 |
| 4Q | 9,850 | 8,775 | 8,415 |
| FY | 40,526 | 32,500 | 31,165 |
| *Prior* | — | 39,917 | 41,963 |

**GAAP EPS**

| | 2016A | 2017E | 2018E |
|---|---|---|---|
| 1Q | (0.35) | (0.24) | (0.23) |
| 2Q | (0.34) | (0.25) | (0.21) |
| 3Q | (0.34) | (0.27) | (0.23) |
| 4Q | (0.28) | (0.29) | (0.24) |
| FY | (1.30) | (1.06) | (0.92) |
| *Prior* | — | (1.23) | (0.86) |

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# Trovagene, Inc.

**Buy**

## Taking The Next Step - Adding in A Precision Cancer Therapeutic to the Platform - Upgrading to Buy

## Summary


- **Trovagene announced that the company in-licensed a small molecule oncology drug from Nerviano Medical Sciences (PCM-075 ), marking the beginning of a new chapter for the company in precision cancer therapeutics. Trovagene will now look to leverage its knowledge base in Precision Cancer Monitoring in combination with a therapeutic asset creating synergy for both products and the best outcome for cancer patients.**
- **PCM-075, an oral polo-like kinase 1 inhibitor (PLK1i) for Acute Myeloid Leukemia (AML). The compound looks like a fast follower to Boehringer Ingelheim's pan-PLKi, Volasertib, which demonstrated increased response rates and improved overall survival in AML patients in a pivotal study but unfortunately induced patient deaths. Unlike Volasertib, PCM-075 targets only PLK1 , has a 5X shorter half life and is oral not IV. As such we believe PCM-075 could be a safer, more efficacious PLKi for AML, as well as other cancers.**
- **Clinical pathway: Trovagene plans to conduct a P1/2 study in AML patients (N=50) ineligible for standard induction therapy, followed by a P2/3 registration study (N=600), a pathway laid out by Boehringer. The P1/2 study will evaluate dose escalation and efficacy, as well as biomarkers (initiate 2017, data 2018).**
- **The focus on the PCM platform will be twofold: 1. To partner/ out license the commercial assets and technology to a firm that has commercial infrastructure to drive market penetration. 2. To develop assays that can be used with PCM-075 to enrich responders.**
- **Model Changes: We have revised our model factoring in PCM-075 (AML drug) as the key driver of the company (expenses and revenues) going forward. We remove revenues associated with the PCM platform. Any monetization of this platform now represents upside to our forecast. We use a 30% risk cut in our therapeutic model and then triangulate our FCFF, dEPS and SOP models (at r=30%) to derive a $4 price target.**


## Details

**PCM-075, a differentiated PLK1 Inhibitor**. Polo-like kinase 1 (PLK1) plays a critical role in cancer cell cycling by facilitating the transition of cells to mitosis. PLK1 is over-expressed in a number of cancers, including Acute Myeloid Leukemia (AML) but has low expression in healthy tissues. PCM-075 is an oral, highly-selective inhibitor of PLK1 with a short half-life (24hrs) in development for AML. PCM-075 has nanomolar (nM) affinity for PLK1 but little activity against PLK2 and PLK3 (>10um, micromolar), which are highly expressed in healthy tissues. This is in contrast to Volasertib, an intravenous pan-PLK inhibitor with high affinity for all three PLKs and a long 135hr half-life. While Volasertib demonstrated activity in combination with low dose cytarabine (LDAC) for induction therapy in AML patients, the P3 trial failed as Volasertib induced a high rate of lethal infections (drug accumulation, pan-PLK, immuosuppression). PCM-075 with a differentiated mechanism of action could be a fast follower to Volasertib and demonstrate at least similar clinical activity but with a far better safety profile. In preclinical and P1 studies, PCM-075 demonstrated a positive safety profile, including reversible hematologic toxicities. A P1/2 study is planned for 2017.

**ctDNA Technology-** Trovagene's Precision Cancer Monitoring (PCM) platform can evaluate tumor DNA from both urine and blood to track patient responses to therapy. Trovagene holds the patent for an NPM1 test, a gene over-expressed in 30-40% of AML patients. The company plans to use the ctDNA technology to profile other major mutations associated with AML including FLT3, DNMT3A, NRAS and KIT to develop an AML gene panel...Precision Cancer Therapeutics.

## INVESTMENT SUMMARY

**The Bull Case. From Precision Cancer Monitoring (PCM) to Precision Cancer Therapeutics**. Bulls appreciate Trovagene's strategic decision to develop an oncology asset in combination with the PCM technology to create a best in class therapeutic in the AML space. At the same time the company will look to partner or out-license the commercial / technology base associated with the PCM platform to the right party with commercial infrastructure which can be leverage to drive PCM revenues.

Trovagene in-licensed PCM-075, a polo-like kinase 1 inhibitor (PLK1i) for the treatment of AML (Acute Myeloid Leukemia) from Nerviano Medical Sciences and could be a fast follower to Boehringer Ingelheim's (BI) pan-PLKi, Volasertib. Volasertib, while efficacious (25-30% complete response rate) in a P3 study, resulted in a high rate of fatal infections, causing the trial to fail, likely due to drug accumulation (long half-life) and pan-PLK inhibition. PCM-075 is differentiated as it only targets PLK1, has a 5X shorter half-life, has better binding to PLK1, and is oral, not IV, making it a potentially safer and more efficacious PLKi. Trovagene plans to follow the clinical blueprint left behind by Boehringer. In addition, Trovagene will leverage its PCM technology which already includes an AML gene test (NPMI) to develop a companion gene monitoring panel for precision monitoring of PCM-075 efficacy. A Phase I study in solid tumors has been completed and a P1/2 study (N=50) in treatment naïve AML patients ineligible for intensive induction therapy is expected to initiate in 2017 (data 2018, dosage and early efficacy). A P2/3 registration study (N=600) would likely follow, positioning approval and commercial launch as early as 2023. Over 20,000 patients are diagnosed with AML each year in the U.S. and the standard of care starts with first-line induction therapy. PCM-075 is an attractive alternative to harsh induction regimens and provides an option for patients that are not suitable for SOC induction therapy. With pricing in the $50K-$75K, and just 2,300 + patients we have a $130M + drug suggesting to us (after a therapeutic rate cut 30%) and a 30% risk rate that we have a compelling valuation in Trovagene versus today's sub $30M market capitalization.

**The Bear Case. Too early, too big and too expensive.** Targeting PLKs in AML has proven to be difficult, as demonstrated by the Volasertib P3 failure. Merck and GSK were also developing PLKs but currently have no active trials. PCM-075 may have the potential to be a safer option but Trovagene lacks supportive clinical data, recall that the P1 study conducted by Nerviano was in advanced solid tumors, not AML. As such, Bears point out that PCM-075 is early in the development cycle so accurately assessing probabilities of success is difficult. Bears will point out that developing an oncology drug, from phase 1, requires years and hundreds of millions of dollars. As such this tends to be a big pharma / big biotech game and partnering requires "proof of concept" to get premium terms. Trovagene with $38M in cash (YE-2016) has limited resources.

**Our Take. A Strategic positive, Early stage but could be a fast follower to Volasertib.** We believe that Trovagene made the right strategic decision to acquire a precision guided therapeutic in the oncology space that when coupled with the PCM platform could create a best in class asset in the AML space. PCM-075 is a polo-like kinase 1 inhibitor in development initially for AML (Acute Myeloid Leukemia) that could be a fast follower to Boehringer Inglheim's (BI) Volasertib, but as a more targeted PLK inhibitor it could be potentially safer. In addition, Trovagene has the strategic advantage of leveraging their existing AML monitoring test for the NPMI (AML test) gene to enrich clinical trials with the "right patients" and track patients' therapeutic response to PCM-075 at the molecular level. This should not only reduce clinical risk but could add to commercial potential. As such the company plans to expand the gene test to a proprietary gene panel that will be used as a companion diagnostic. From a clinical perspective Trovagene plans to follow the blueprint left behind by BI; a phase 1/2 dose ranging and POC study in 50 AML patients not suitable for intense induction therapy, followed by a 600-patient registration study. With the restructuring of the company to pursue development of PCM-075, costs have been reduced and we estimate the company now has ~$30M (1Q17E) on the balance sheet, or over a year of operating capital. We expect the P1/2 study to initiate in 2017 with data updates on dose ranging and early efficacy providing news flow (catalysts) for the stock. The full data set from the P2 portion of the study is expected in 2018 and if positive would represent proof of concept, setting the stage for a larger pivotal program. PCM-075 could be an option for induction therapy in AML patients not eligible for harsher SOC regimens, or an alternative for patients who elect not to undergo harsh SOC induction. As such PCM-075 could be widely adopted. We see a company at a "distressed" valuation that is trading at cash and has a newly licensed PLK1 inhibitor with attributes that suggest it could be a fast follower Volasertib. As such, we see upside.

**Finances:** Trovagene reported YE16 with a net loss of $39M. The company announced that plans for restructuring and shift in focus towards therapeutic development. Trovagene will retain the CLIA-CAP-accredited laboratory for clinical services to pharmaceutical companies and for internal programs. Initially operating expenses are expected to fall, $8M per year. Trovagene ended 2016 with $38M in cash on the balance sheet. Concurrent with reported earnings, Trovagene announced that the company has in-licensed a small molecule polo-like-Kinase 1 inhibitor (PCM-075) from Nerviano Medical Sciences for $2M upfront as well as undisclosed milestones associated with development and commercialization (up to $65M), and royalties to Nerviano. We estimate that Trovagene currently has $30M in cash, sufficient runway to 1H18 considering reduced operating expenses associated with a shift away from diagnostics and towards early stage therapeutic development.

**Exhibit 1. Trovagene Pipeline**

| Product (Therapeutics) | Indication | Development | Pre-clinical | Phase I | Phase II | Phase III | Marketed |
|---|---|---|---|---|---|---|---|
| PCM-075 | Acute Myeloid Leukemia | | | | | | |
| PCM-075 | Solid Tumors | | | | | | |

*Source: Maxim estimates and company reports*

| Product (PCM Diagnostics) | Indication | Development | Pre-clinical | Validation | Marketed |
|---|---|---|---|---|---|
| NPM1 Gene Assay | Acute Myeloid Leukemia | | | | |
| AML Gene Panel | Acute Myeloid Leukemia | | | | |

*Source: Maxim estimates and company reports*

**Exhibit 2. Upcoming Catalysts for Trovagene**

| Product | Geography | Indication | Event | Timeline | Impact |
|---|---|---|---|---|---|
| Precision Cancer Monitoring | US | Cancer diagnositcs/theragnostics | Monteize legacy PCM assays | 2017 | + |
| PCM-075 | US | Acute Myeloid Leukemia | Initiate P1/2 study | 1H17 | + |
| PCM-075 | US | Acute Myeloid Leukemia | Announce P2 data, select dose for P2 | 4Q17 | + |
| PCM-075 | US | Acute Myeloid Leukemia | Report P2 data | mid-2018 | ++ |
| AML Gene Panel | US | Cancer diagnositics/theragnostics | Announce companion diagnostic/theragnostic panel for PCM-075 in AML | 2018 | + |
| PCM-075 | US | Acute Myeloid Leukemia | Initiate P2/3 registration study | 1H19 | ++ |
| PCM-075 | US | Acute Myeloid Leukemia | Complete P2, expand to P3 | 2020 | + |
| PCM-075 | US | Acute Myeloid Leukemia | Pivotal data, file NDA | 2022 | +++ |
| PCM-075 | US | Acute Myeloid Leukemia | Commercial launch | 2023 | +++ |

Stock Significance Scale: + of moderate importance; ++ higher level; +++ highly

Source: Maxim Forecasts and Company reports.

## Polo-Like Kinase Inhibitors

**Polo-like Kinases:** Cell growth and control is a highly coordinated system of checkpoints and molecular signaling pathway crosstalk that ensures and maintains genome stability. The cell must maintain checkpoints to halt cell cycle progression if errors occur, giving the cell time to repair its DNA. The critical checkpoints in the cell cycle occur at the G1/S phase, the G2/M phase and during mitosis before cell division. This process is tightly regulated by three families of kinases; the cyclin-dependent kinases (CDKs), aurora kinases and the polo-like kinases (PLKs)[1]. The latter, PLKs, are a diverse family of protein kinases that play multiple rolls in the regulation of the cell cycle and are often dysregulated in many cancer types. As such PLKs are promising targets for anti-cancer therapies. There are five members of the PLK family, PLK1, PLK2, PLK3, PLK4 and PLK5[2]. Of these, PLK1 is the most extensively characterized PLK. PLK1 is expressed mainly during the G2 and M phases of the cell cycle where it is essential for precisely regulating cell division and maintaining genome stability in mitosis, spindle assembly and DNA damage response. PLK1 is expressed at very low levels in most normal tissues, including the liver, brain, lung, kidney and pancreas making it an attractive target for anti-cancer therapy by limiting potential off-target effects. However, PLK1 is overexpressed in rapidly dividing cells like the ovaries, testis, spleen and placenta, as well as many types of cancer (lung, breast, ovarian, B cell malignancies, H&N etc.)[3]. Overexpression of PLK1 in cancer is associated with poor prognosis.

[1] Xiaoqi Liu. 'Targeting Polo-Like Kinases: A Promising Therapeutic Approach for Cancer Treatment'. Translational Oncology. June 2015, 8(3), pp185-195.

[2] Zhixian Liu, Qingrong Sun, Xiaosheng Wang. 'PLK1, A Potential Target for Cancer Chemotherapy'. Translational Oncology. February 2017, 10(1),pp22-32.

[3] Brandwein JM. 'Targeting Polo-Like Kinase 1 in Acute Myeloid Leukemia'. Therapeutic Advances in Hematology. 2015, 6(2), pp80-87.

**Exhibit 3. Comparison of PLK1 Expression Between Cancers and Normal Tissues.** PLK1 is expression is relatively low in normal tissues where are in many cancer types it overexpressed.

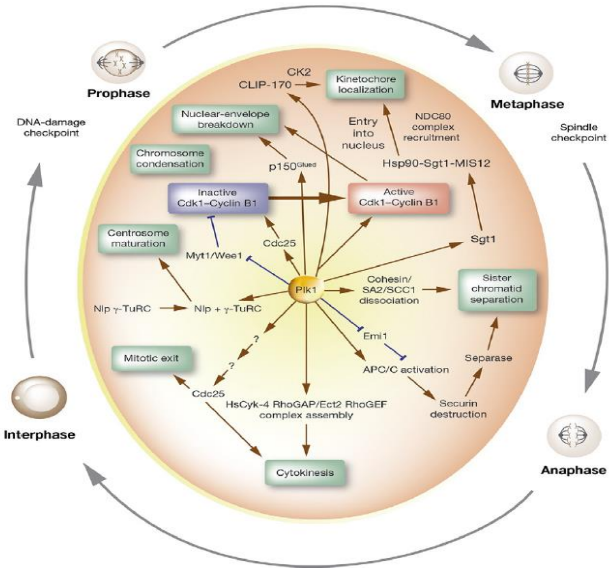
| Cancer Type | Full Name | *P* Value | Fold Change * |
|---|---|---|---|
| LUSC | Lung squamous cell carcinoma | 7.41E-157 | 20.8 |
| BRCA | Breast invasive carcinoma | 5.88E-126 | 11.3 |
| LUAD | Lung adenocarcinoma | 1.18E-63 | 9.7 |
| KIRC | Kidney renal clear cell carcinoma | 2.33E-55 | 6.1 |
| HNSC | Head and neck squamous cell carcinoma | 6.52E-50 | 4.2 |
| LIHC | Liver hepatocellular carcinoma | 3.59E-40 | 11.7 |
| UCEC | Uterine corpus endometrial carcinoma | 1.96E-36 | 21.3 |
| COAD | Colon adenocarcinoma | 5.97E-33 | 2.5 |
| STAD | Stomach adenocarcinoma | 8.45E-27 | 4.8 |
| ESCA | Esophageal carcinoma | 9.52E-27 | 10.2 |
| BLCA | Bladder urothelial carcinoma | 4.96E-26 | 9.1 |
| PRAD | Prostate adenocarcinoma | 1.29E-22 | 3.3 |
| KIRP | Kidney renal papillary cell carcinoma | 6.76E-22 | 4.7 |
| CHOL | Cholangiocarcinoma | 6.97E-14 | 24.3 |
| GBM | Glioblastoma multiforme | 5.63E-12 | 12.4 |
| KICH | Kidney chromophobe | 1.63E-06 | 3.3 |
| READ | Rectum adenocarcinoma | 1.06E-05 | 2.3 |
| PAAD | Pancreatic adenocarcinoma | 0.04 | 2.2 |

* Mean *PLK1* expression in cancer/mean *PLK1* expression in normal tissue.

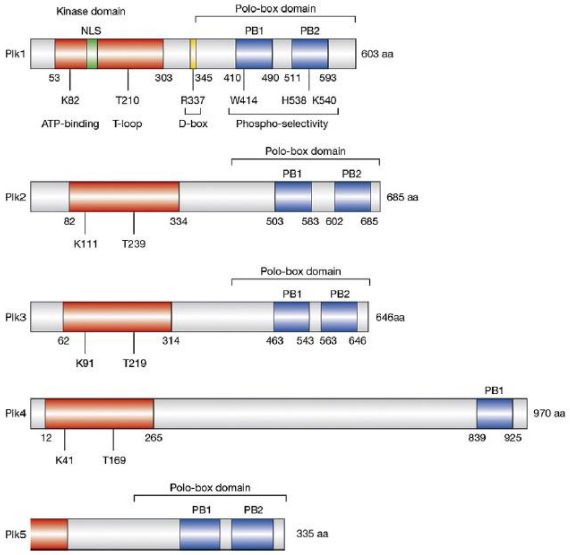
*Source: Zhixian Liu 2017*[2]

**Exhibit 4. PLK1 Multifunctional Role In Mitosis. (A)** A schematic depicting PLK1 as a regulator of several stages during mitotic progression. **(B)** Domain structures of the human PLK1 family of proteins. Kinase domains are shown in red and polo-box domains are shown in blue. Note the green bar in PLK1 which is a nuclear localization sequence. PLK1 is essential for numerous stages of mitosis, including spindle assembly, M phase entry, NERD (nuclear envelope breakdown, sister chromatid cohesions and formation of kinetochore attachments, mitotic exit and cytokinesis[4].

**(A)**




**(B)**



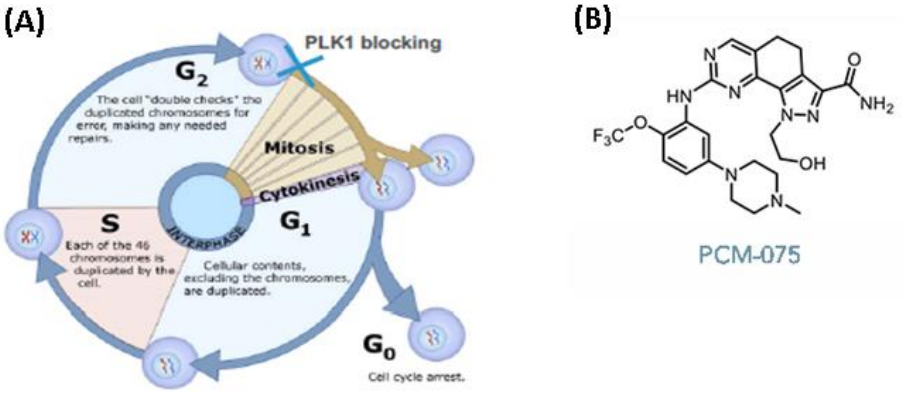

*Source: Lie Xiaoqi 2015*[4].

[4] Xiaoqi Liu. 'Targeting Polo-Like Kinases: A Promising Therapeutic Approach for Cancer Treatment'. Translational Oncology. June 2015, 8(3), pp185-195.

**PCM-075, a differentiated PLK inhibitor.:** PCM-075 is an orally available PLK1 inhibitor. PCM-075 (formerly NMS-P937) was in-licensed from Nerviano Medical Sciences by Trovagene in March 2017. Studies have demonstrated that blocking PLK1 induces tumor cell apoptosis across both solid and liquid cancers where PLK1 is often overexpressed. Blocking PLK1 arrests the cell cycle at a critical checkpoint, between G2 and progression into M phase (mitosis, cell division). PLK inhibitors that have been developed are pan-PLK inhibitors which also impact PLK2 and PLK3, the two members of the PLK family most similar to PLK1. However, less in known about the exact role of PLK2 and PLK3 in healthy, non-proliferating tissues where they are highly expressed. As such, developing a PLK1-specific inhibitor may have a superior safety profile than a pan-PLK-inhibitor[5] (i.e. Boehringer Ingleheim's Volasertib). PCM-075 is a pyrazoloquinazoline and a potent inhibitor of PLK1. PCM-075 was screened for activity against 63 protein kinases and was found to inhibit PLK1 with an $IC_{50}$ of 2 nanomolar (nM), but essentially has no activity against PLK2 and PLK3 (>10um range)[5]. This is in contrast to other PLK inhibitors in development that have nM activity against all three PLKs and are pan-PLK inhibitors with side effects.

**PLK inhibition in AML:** AML is a heterogenous blood cancer that accounts for 30% of adult leukemias. There are a wide range of genetic abnormalities associated with AML, including chromosomal imbalance (42% of cases), chromosomal translocations/inversions/deletions (15% of cases) and point mutations in specific genes like FLT3 (37% of cases), NPM1 (30% of cases), CEBPA (10% of cases) and DNMT3 (23% of cases). PLK1 is also overexpressed in AML and has become the target of PLK inhibitor drug design for this indication. Why PLKs? The standard of care in AML is for patients to undergo front-line induction chemotherapy to clear the bone marrow of AML blast cells. Intensive induction therapy consisting of cytarabine plus an anthracycline, followed by several rounds of consolidation chemotherapy can induce complete responses in up to 70% of cases in patients under the age of 60 and 50-55% in patients over the age of 60[6]. There are also a significant number of patients that are ineligible for harsh induction therapy and require an alternative that includes low dose cytarabine (LDAC). PLK inhibitors are an attractive alternative for combination with LDAC for induction therapy.

**Exhibit 5. PCM-075 Biochemical Profile. (A)** PLK1 inhibitors arrest the cell cycle at the G2/M transition and block the cell from progressing to mitosis, inducing the cell to undergo apoptosis**. (B)** Chemical structure of PCM-075. PCM-075 is a selective adenosine triphosphate (ATP) competitive inhibitor of PLK1. The selectivity of PCM-075 is driven by polar interaction with the side chain Glu140 of PLK1.




*Source: Trovagene Presentation*

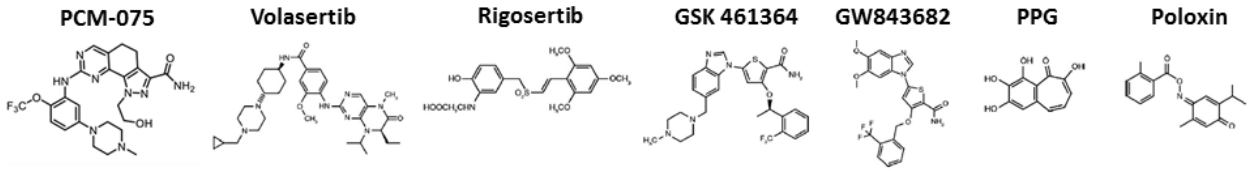
[5] Valsasina B et al. 'NMS-P937, an Orally Available, Specific Small-Molecule Polo-like Kinase Inhibitor with Antitumor Activity in Solid and Hematological Malignancies'. Molecular Cancer Therapeutics. February 2012. 11(4), pp1006-1016.

[6] Brandwein JM. 'Targeting Polo-Like Kinase 1 in Acute Myeloid Leukemia'. Therapeutic Advances in Hematology. 2015, 6(2), pp80-87.

**Exhibit 6. High Selectivity for PLK1 Differentiates PCM-075 from other PLK inhibitors in Development.** PCM-075 has a distinct advantage over other PLK inhibitors in development as PCM-075 is highly selective for the PLK1 protein with nanomolar affinity with only marginal activity against PLK2 and PLK3 but only with much more drug, >10 micromolar. This is in contrast to other PLKs, including the most clinically advanced, BI's Volasertib, which has inhibitory activity against all three PLKs and is essentially a pan-PLK inhibitor. However, while Volasertib was successful in a P2 study in AML, and in the P3 study demonstrated improved response rates and overall survival, the drug induced a high rate of lethal infections causing the trial to fail. The increased infections were likely due to drug accumulation (long half-life, 135 hrs), pan-PLK inhibition, and a lack of consistent prophylaxis of patients to prevent infections. PCM-075 has a far shorter half-life (24 hrs vs 135hrs), is selective for PLK1 with minimal activity against PLK2 and 3, and is oral (vs intravenous). Combined, PCM-075 may be a safer more efficacious PLK inhibitor.

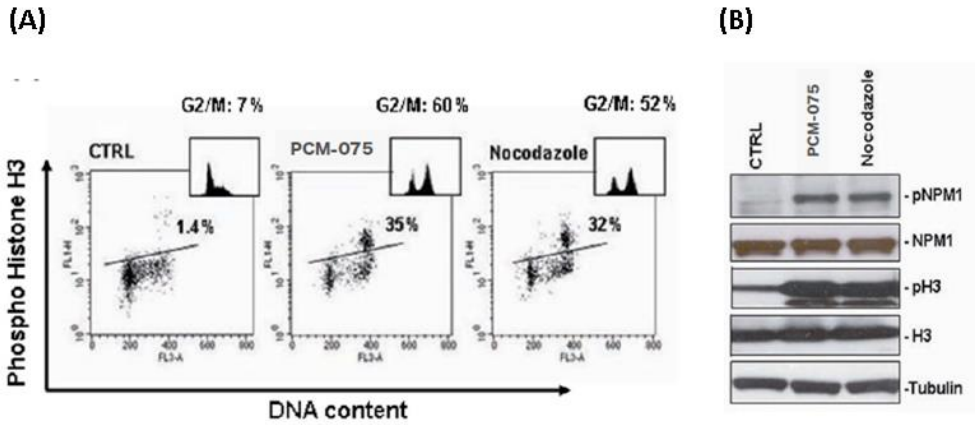
| Agent/Structure | Mechanism of Action | $IC_{50}$ PLK1 | PLK2 | PLK3 |
|---|---|---|---|---|
| **PCM-075 (pyrazoloquinazoline)** | Selective ATP-competative inhibitor | 2 nM | >10 µM | >10 µM |
| **Volasertib (BI 6727, Dihydropteridinone derivative)** | ATP-Competative Inhibitor | 0.87 nM | 5 nM | 56 nM |
| **Rigosertib (ON 01910.Na, benzylstyryl sulphone)** | Affects microtubule dynamics | 9-10 nM | ~100-200 nM | not reported |
| **GSK 461364 (thiophene derivative)** | ATP-Competative Inhibitor | <0.5 nM | 860 nM | 1 µM |
| **GW843682 (benzimidazole thiophene)** | ATP-Competative Inhibitor | 2.2 nM | n/a | 9.1 nM |
| **PPG (benzotropolone-containing compound)** | Inhibits PBD-dependent binding | 300 nM | n/a | n/a |
| **Poloxin (thymoquinone derivative)** | Interferes with PLK1 PBD | 4.8 µM | 18.7 µM | 53.9 µM |

nM- nanomolar, µM- micromolar, $IC_{50}$ Concentration for 50% Inhibition




*Source: Adapted from Xiaoqu Liu 2015[7] and Trovagene Presentation*

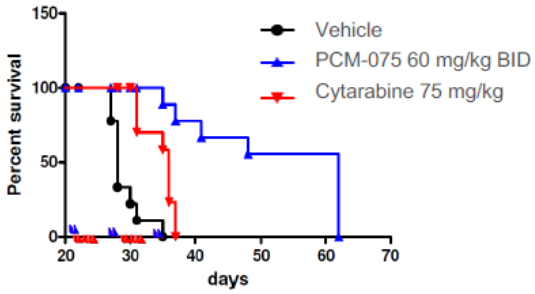
**Exhibit 7. PCM-075 Activity in AML.** PCM-075 induces mitotic inhibition and has demonstrated anti-proliferative activity in models of AML **(A)** PCM-075 arrests cells at the G2/M phase, leading to cell death. The analysis evaluated phospho-histone H3 and DNA content by flow cytometry. The panels show the correspondent cell cycle profile and indicate the percentage of cells in the G2/M phase. **(B)** Western blots analysis of total cell lysates for the following proteins; phosphor-NPM1 (pNPM1), NPM1, phosphor-histone H3 (pH3), and Histone-H3 (H3). Tubulin served as a loading control. Shown here is PCM-075 inducing in-vitro pathway modulation in patient-derived AML cells as depicted by the changes in phosphorylation of key proteins involved in AML.




*Source: Casalaro A, 2013[8] and Trovagene Presentation*

[7] Xiaoqi Liu. 'Targeting Polo-Like Kinases: A Promising Therapeutic Approach for Cancer Treatment'. Translational Oncology. June 2015, 8(3), pp185-195.

**Exhibit 8. PCM-075 Extends Survival in AML Xenograft Models.** Shown below is the response observed in various xenograft models of PCM-075 as a single agent or in combination with low-dose cytarabine. The treatment with PCM-075 started at 20-days post-inoculation at 60mg/kg BID (Days 1 and 2, 3 cycles). Median survival of the mice nearly doubled in the PCM-075 group.

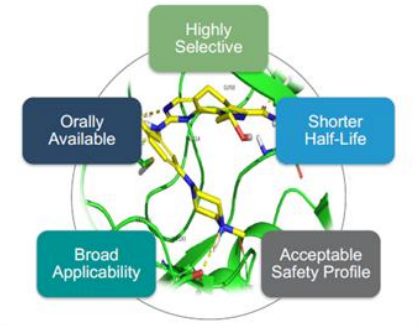



| Compound | Median Survival Time (days) | %TGI |
|---|---|---|
| Placebo | 28 | |
| Cytarabine | 38 | 128.6 |
| PCM-075 | 62 | 221.4 |

*Source: Casalaro A, 2013 and Trovagene Presentation*

**Exhibit 9. PCM-075 is differentiated from Volasertib.** As discussed above, PCM-075, with higher selectivity for PLK1, a shorter half-life and an oral formulation could potentially be a more efficacious and safer PLK inhibitor than Volasertib. Volasertib is a pan-PLK inhibitor with significant activity against other PLKs and a long half-life which combined was likely responsible for the high rate of lethal infections observed in the phase 3 study. However, Volasertib did induce a 25-30% complete response rate in a difficult to treat population. As such, PCM-075 with a differentiated MOA could be a fast-follower to Volasertib and be successful in AML.

(A)




(B)

| Compound | Selectivity | Half-Life (hrs) | Dosing | Clinical Activity in AML |
|---|---|---|---|---|
| Volasertib | pan-PLK | 135 | iv | 25-30% CR+CRi (+LDAC)* |
| PCM-075 | PLK1 | 24 | oral | TBD |

*Source: Trovagene Presentation*

[8] Casalaro et al. 'The Polo-Like Kinase Inhibitor NMS-P937 Is Effective in a New Model of Disseminated Primary CD56+ Acute Myelodysplastic Leukemia'. PLOS March 2013. 8(3).

## Precision Cancer Monitoring to Develop PCM-075

**Precision Cancer Monitoring (PCM)-** Recall that the PCM platform is based on the use of circulating tumor DNA (ctDNA) in the blood and urine monitor specific cancer gene mutations, providing the clinician with actionable results. The mutations the company focuses on are mutations, well documents in treatment guidelines.

**Circulating tumor cells (CTCs).** Circulating tumor cells, or CTCs, are cancer cells that have shed into the bloodstream from a primary tumor. Recent technological advances in the detection, isolation, capture, and characterization of CTCs from blood samples have enabled the evaluation of different CTC biomarkers, but clinical validation is still necessary. Additionally, initial sensitivities and specificities are currently insufficient and cost effective clinical tests may be challenging to develop. That said, CTC biomarkers are a promising technology that could eventually be useful in determining prognosis, monitoring the effectiveness of treatment, and as a source of the tumor identification and characterization when a surgical biopsy is unavailable. Moreover, CTC biomarkers could also eventually be used for cancer recurrence monitoring or for general population screening.

**Cell-free circulating tumor DNA (ctDNA).** In the human body, about 1011 - 1012 cells die each day primarily as a consequence of natural physiological processes for tissue and organ maintenance, but also as a result of disease. In fact, it occurs most frequently in cells that are rapidly dividing, which includes cancer cells, where cell growth is both rapid and uncontrolled. These dead and dying cells contain more than one gram of DNA, which is mostly degraded into short fragments by specific enzymes. These cell-free nucleic acids can be found in a variety of human bodily fluids. Many scientists believe that due to the heterogeneity of tumor cells, ctDNA, like CTCs, may be more representative of a patient's tumor than a surgical tissue biopsy. Analyzing ctDNA for mutations is promising as a biomarker. In addition to not requiring an invasive tissue biopsy, ctDNA does not require tumor cell capture (a key complication that researchers still need to overcome with CTCs). However, blood continually breaks down the ctDNA fragments, even after the sample is drawn from the patient, necessitating immediate testing. Blood also contains many types of cells, proteins, and other contaminants, which can further complicate analysis.

Trovagene discovered that ctDNA cross the kidney barrier and can be found in the urine as transrenal nucleic acids (TrNA). Urine does not contain many cells, proteins or other contaminants, nor does it contain components that can attack and break down cell-free nucleic acid fragments allowing for more simplified analysis. Ultra-short amplicon assays are necessary because enzymes break down ctDNA into small fragments. Many DNA isolation kits only capture DNA 100bps-200bps in length or longer. Based on Trovagene internal studies, the company's ultra-short amplicon assays are three to twenty times more efficient in isolating a 50bps target than competing assays. Trovagene is able to detect at least six times more mutations in a urine-based cell-free DNA sample than any other PCR-based assay, according to the company's internal test data. In addition to urine-based test, which the company remains focused on developing, Trovagene methods for isolating short DNA fragments should also be applicable to other small or fragmented nucleic acids, including cell-free DNA from blood and formalin-fixed, paraffin-embedded (FFPE) samples. Moreover, urine allows for a truly non-invasive collection of a sample at home (or in a hospital, physician's office or clinic) in large volume, and at a relatively low cost. These benefits of a urine-based test enable more frequent, non-invasive monitoring of oncogene mutation status, disease progression and disease recurrence.

**Mutations in AML and Trovagene's Precision Cancer Monitoring (liquid biopsy, ctDNA) platform.** Trovagene is uniquely positioned to develop an AML biomarker panel for use in clinical studies through leveraging the PCM platform. Recall that Trovagene already has an approved assay for AML to monitor the NPM1 (nucleophosmin 1) gene. The NPM1 assay is approved for the diagnosis and monitoring of AML patient responses to therapies. In addition, most other markers in AML, including FLT3, DNMT3A and NRAS genes have already been evaluated ctDNA PCM technology. Trovagene plans to combine analyses of these genes into one panel for AML. The FLT3, NMP1, DNMT3A and NRAS genes, and mutations in these genes are observed in 37%, 29%, 23% and 10% of AML cases, respectively. The World Health Organization has a classification scheme for AML that provides a framework for clinical management. While cytogenetic abnormalities (chromosomal alterations) are important, the WHO has provided two categories for changes at the genetic level, involving changes in the NPM1 gene and the CEBPA gene[9].

**NPM1,** the target of Trovagene's approved assay, is among the genetic alterations in AML that is a prognostic genetic marker. The NPM1 gene is important for many tumor-associated chromosomal translocations. The protein product of NPM1 shuttles in and out of the nucleus and several roles including binding to p53 tumor suppressor (guardian of the genome), initiation of centrosome duplication and ribosomal protein assembly (protein production). The frequency of mutation of the NPM1 gene has been reported as high as 37% in some studies. However, mutations in NMP1 are associated with a better prognosis when another important AML gene, FLT3, lacks the mutation ITD (internal tandem duplication (FLT-IDT). An intermediate prognosis is determined when FLT-ITD is present. The NPM1 mutation while found in ~30% of all AML patients, is found in 45-50% of patients who are cytogenetically normal[10]. Mutations in NPM1 have been well documented to confer chemosensitivity and are particular useful for risk monitoring minimal residual disease[11]. As such, Trovagene, which owns its own ctDNA PCM assay for NPM1 mutations has a strategic advantage for risk stratifying AML patients to enrich study populations with the "right" patients and monitor the effectiveness of PCM-075 at the molecular level. Other mutation monitoring is expected to be built into an AML gene panel, including mutations in FLT3, CEBPA and DNMT3A.

[9] Swerdlow et al. 'WHO Classification of Tumors of Haematopoietic and Lymphoid Tissues'. IARC Press 2008

[10] Gulley ML, Shea TC, Fedoriw Y., 'Genetic Test to Evaluate Prognosis and Predict Therapeutic Response in Acute Myeloid Leukemia'. Journal of Molecular Diagnostics. January 2010. 12(1).

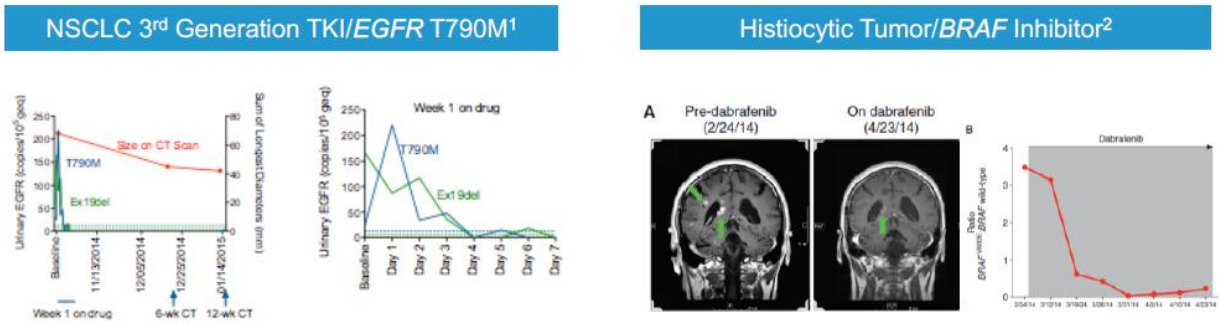
[11] Hao Z and Kota V., 'Volasertib for AML: Clinical Use and Patient Consideration'. Oncotargets and Therapy. 2015:8, pp1761-1771

**Exhibit 10. Common Mutations and Frequency of Mutations in AML.**

| Gene | Overall Frequency (%) |
|---|---|
| *FLT3* (ITD, TKD) | 37 (30, 7) |
| *NPM1* | 29 |
| *DNMT3A* | 23 |
| *NRAS* | 10 |
| *CEBPA* | 9 |
| *TET2* | 8 |
| *WT1* | 8 |
| *IDH2* | 8 |
| *IDH1* | 7 |
| *KIT* | 6 |
| *RUNX1* | 5 |
| *MLL*-PTD | 5 |
| *ASXL1* | 3 |
| *PHF6* | 3 |
| *KRAS* | 2 |
| *PTEN* | 2 |
| *TP53* | 2 |
| *HRAS* | 0 |
| *EZH2* | 0 |

*Source. Trovagene Presentation.*

**Exhibit 11. Example of Trovagene's ctDNA for Precision Cancer Monitoring.** Trovagene's ctDNA tests can be effectively used to monitor therapeutic response as demonstrated by changes in the presence or absence of specific mutations at baseline and during therapy. This is further supported with radiographic evidence demonstrating reduction in tumor size. Shown here are patient responses to a tyrosine kinase inhibitor in lung cancer tracking the T790M mutation in EGFR (endothelial growth factor receptor) and responses for a brain tumor treated with a BRAF inhibitor.




*Source. Trovagene Presentation*

## Modeling Assumptions

1- We assume that PCM-075 is commercialized in 2023 in the U.S. and 2024 in Europe.
2- Given that all AML patients go through induction chemotherapy with intense regimens, we assume a significant market penetration of up to 25% based on a large population of patients ineligible for intense therapy as well as patients in general opting for a less intense regimen (PCM-075 + low dose cytarabine).
3- We assume pricing in the U.S. of $75K and $50K in Europe with a y/y increase of 5%.
4- A risk adjustment is applied based on stage of development for PCM-075 today.

### Exhibit 12. PCM-075 AML Market Model (US)

| PCM-075, AML (U.S.) | 2016E | 2017E | 2018E | 2019E | 2020E | 2021E | 2022E | 2023E | 2024E | 2025E | 2026E | 2027E |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| Acute Myeloid Leukemia (AML) Incidence | 19,950 | 20,349 | 20,756 | 21,171 | 21,595 | 22,026 | 22,467 | 22,916 | 23,375 | 23,842 | 24,319 | 24,805 |
| Increase in incidence | 2% | 2% | 2% | 2% | 2% | 2% | 2% | 2% | 2% | 2% | 2% | 2% |
| Market pentration | | | | | | | | 5% | 10% | 18% | 22% | 25% |
| Total patients treated (Induction with PCM-075 +LDAC) | | | | | | | | 1,146 | 2,337 | 4,292 | 5,350 | 6,201 |
| Cost of treatment | | | | | | | | $ 75,000 | $ 78,750 | $ 82,688 | $ 86,822 | $ 91,163 |
| Increase/decrease in price | | | | | | | | 5% | 5% | 5% | 5% | 5% |
| Total revene ('000) | | | | | | | | $ 85,936 | $ 184,075 | $ 354,860 | $ 464,511 | $ 565,332 |
| Risk Adjustment | | | | | | | | 30% | 30% | 30% | 30% | 30% |
| **Total revenue ('000)** | | | | | | | | **$ 60,155** | **$ 128,853** | **$ 248,402** | **$ 325,158** | **$ 395,732** |

*Source: Maxim estimates*

### Exhibit 13. PCM-075 AML Market Model (EU)

| PCM-075, AML (EU) | 2016E | 2017E | 2018E | 2019E | 2020E | 2021E | 2022E | 2023E | 2024E | 2025E | 2026E | 2027E |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| Acute Myeloid Leukemia (AML) Incidence | 22,000 | 22,440 | 22,889 | 23,347 | 23,814 | 24,290 | 24,776 | 25,271 | 25,777 | 26,292 | 26,818 | 27,354 |
| Increase in incidence | 2% | 2% | 2% | 2% | 2% | 2% | 2% | 2% | 2% | 2% | 2% | 2% |
| Market pentration | | | | | | | | | 5% | 10% | 20% | 25% |
| Total patients treated (Induction with PCM-075 +LDAC) | | | | | | | | | 1,289 | 2,629 | 5,364 | 6,839 |
| Cost of treatment | | | | | | | | | $ 50,000 | $ 52,500 | $ 55,125 | $ 57,881 |
| Increase/decrease in price | | | | | | | | | 5% | 5% | 5% | 5% |
| Total revene ('000) | | | | | | | | | $ 64,441 | $ 138,033 | $ 295,667 | $ 395,824 |
| Risk Adjustment | | | | | | | | | 30% | 30% | 30% | 30% |
| **Total revenue ('000)** | | | | | | | | | **$ 45,109** | **$ 96,623** | **$ 206,967** | **$ 277,077** |

*Source: Maxim estimates*

## VALUATION

Our model assumes modest revenue from partnership / out-licensure of the diagnostics assets and retained CLIA lab services for pharma companies. We factor in PCM-075 in AML, with commercialization in the U.S. in 2023, followed by Europe in 2024, as well as revenue associated with and AML diagnostic assay. Our model assumes a risk adjustment based on stage of development. Our 12-month price target for Trovagene, Inc. is based on FCFF, dEPS and SOP models, which each use a 30% discount rate and are equally weighted to derive a 12-month price target of $4.0.

### Exhibit 14. Free Cash Flow Model

| | | |
|---|---|---|
| Average | $ | 4 |

| | | |
|---|---|---|
| Price Target | $ | 3 |
| Year | | 2017 |

**DCF Valuation Using FCF (mln):**

| | | 2015 | 2016 | 2017 | 2018 | 2019 | 2020 | 2021 | 2022 | 2023 | 2024 | 2025 | 2026 | 2027 |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| units ('000) | | 2015E | 2016E | 2017E | 2018E | 2019E | 2020E | 2021E | 2022E | 2023E | 2024E | 2025E | 2026E | 2027E |
| EBIT | | (27,447) | (39,204) | (32,500) | (31,165) | (31,477) | (31,791) | (32,109) | (32,430) | 21,385 | 130,233 | 286,109 | 458,665 | 589,444 |
| Tax Rate | | 0% | 0% | 0% | 0% | 0% | 0% | 0% | 0% | 0% | 0% | 0% | 5% | 8% |
| EBIT(1-t) | | (27,447) | (39,204) | (32,500) | (31,165) | (31,477) | (31,791) | (32,109) | (32,430) | 21,385 | 130,233 | 286,109 | 435,732 | 542,288 |
| CapEx | | (2,241) | (823) | (906) | (996) | (1,096) | (1,206) | (1,326) | (1,459) | (1,605) | (1,765) | (1,942) | (2,136) | (2,349) |
| Depreciation | | 379 | 1,070 | 659 | 692 | 726 | 762 | 801 | 841 | 883 | 927 | 973 | 1,022 | 1,073 |
| Change in NWC (minus the increase in NWC) | | | | | | | | | | | | | | |
| FCF | | (29,309) | (38,958) | (32,747) | (31,470) | (31,847) | (32,235) | (32,635) | (33,049) | 20,663 | 129,395 | 285,141 | 434,618 | 541,011 |
| PV of FCF | | (49,532) | (50,645) | (32,747) | (24,208) | (18,844) | (14,672) | (11,426) | (8,901) | 4,281 | 20,621 | 34,955 | 40,984 | 39,244 |
| Discount Rate | | 30% | | | | | | | | | | | | |
| Long Term Growth Rate | | 1% | | | | | | | | | | | | |
| Terminal Cash Flow | | 993,077 | | | | | | | | | | | | |
| Terminal Value YE2025 | | 121,741 | | | | | | | | | | | | |
| NPV | | 151,028 | | | | | | | | | | | | |
| NPV-Debt | | | | | | | | | | | | | | |
| Shares out (thousands) | | 45,851 | 2027E | | | | | | | | | | | |
| NPV Per Share | $ | 3 | | | | | | | | | | | | |

Source: Maxim estimates

### Exhibit 15. Discounted EPS Model

| | | |
|---|---|---|
| Current Year | | 2017 |
| Year of EPS | | 2027 |
| Earnings Multiple | | 10 |
| Discount Factor | | 30% |
| Selected Year EPS | $ | 6.29 |
| NPV | $ | 5 |

Source: Maxim estimates

| | | Discount Rate and Earnings Multiple Varies, Year is Constant | | | | | |
|---|---|---|---|---|---|---|---|
| | | 2027 EPS | | | | | |
| Earnings Multiple | 4.6 | 5% | 10% | 15% | 20% | 25% | 30% |
| | 0 | $0.00 | $0.00 | $0.00 | $0.00 | $0.00 | $ - |
| | 5 | $19.31 | $12.13 | $7.77 | $5.08 | $3.38 | $ 2.28 |
| | 10 | $38.62 | $24.25 | $15.55 | $10.16 | $6.75 | $ 4.56 |
| | 15 | $57.92 | $36.38 | $23.32 | $15.24 | $10.13 | $ 6.84 |
| | 20 | $77.23 | $48.50 | $31.10 | $20.32 | $13.51 | $ 9.13 |
| | 25 | $96.54 | $60.63 | $38.87 | $25.40 | $16.88 | $ 11.41 |
| | 30 | $115.85 | $72.75 | $46.64 | $30.48 | $20.26 | $ 13.69 |
| | 35 | $135.16 | $84.88 | $54.42 | $35.56 | $23.64 | $ 15.97 |

### Exhibit 16. Sum-of-the-Parts Model

| Trovagene, Inc | LT Gr | Discount Rate | Yrs. to Mkt | % Success | Peak Sales MM's | Term Val |
|---|---|---|---|---|---|---|
| **PCM-075 (AML, U.S.)** | 1% | 30% | 6 | 50% | $396 | $1,365 |
| NPV | | | | | | $2.31 |
| PCM-075 (AML, Europe) | 1% | 30% | 6 | 50% | $277 | $955 |
| NPV | | | | | | $1.62 |
| Genetic Panel for AML, Companion Diagnostic | 1% | 30% | 6 | 50% | $20 | $69 |
| NPV | | | | | | $0.12 |
| Precision Cancer Monitoring Platform | 1% | 30% | 1 | 75% | $10 | $34 |
| NPV | | | | | | $0.33 |
| Net Margin | | | | | | 75% |
| MM Shrs OS (2024E) | | | | | | 46 |
| Total | | | | | | $4 |

Source: Maxim estimates

| Trovagene, Inc. .: Income Statement ($000) | | | | | | | | | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| .: YE December 31 | 1Q16A | 2Q16A | 3Q16A | 4Q16A | 2016A | 1Q17E | 2Q17E | 3Q17E | 4Q17E | 2017E | 2018E | 2019E | 2020E | 2021E | 2022E | 2023E | 2024E | 2025E | 2026E | 2027E |
| **Revenues** | | | | | | | | | | | | | | | | | | | | |
| Royalty income | 113 | 48 | 47 | 50 | 258 | | | | | | | | | | | | | | | |
| Diagnostic service revenue | 7 | 24 | 38 | 17 | 86 | | | | | | | | | | | | | | | |
| Other revenue | | 32 | 4 | 1 | 37 | | | | | | | | | | | | | | | |
| PCM-075 (Acute Myeloid Leukemia, U.S.) | | | | | | | | | | | | | | | | 60,155 | 128,853 | 248,402 | 325,158 | 395,732 |
| PCM-075 (Acute Myeloid Leukemia, Europe) | | | | | | | | | | | | | | | | | 45,109 | 96,623 | 206,967 | 277,077 |
| PCM-075 Companion Gene Panel | | | | | | | | | | | | | | | | | 7,500 | 10,000 | 15,000 | 20,000 |
| | | | | - | | | | | | | | | | | | | | | | |
| **Total Product Sales** | 120 | 104 | 89 | 68 | 381 | - | - | - | - | - | - | - | - | - | - | 60,155 | 181,461 | 355,025 | 547,125 | 692,809 |
| **Royalties, Collaborative Revenenue, Other** | | | | | | | | | | | | | | | | | | | | |
| Precision Cancer Monitioring Platform | | | | | | | | | | | 500 | 750 | 1,500 | 2,000 | 3,000 | 4,500 | 6,000 | 7,000 | 7,500 | 10,000 |
| Total other revenue | - | - | - | - | 0 | - | - | - | - | - | 500 | 750 | 1,500 | 2,000 | 3,000 | 4,500 | 6,000 | 7,000 | 7,500 | 10,000 |
| **Total Revenues** | 120 | 104 | 89 | 68 | 380.90 | - | - | - | - | - | 500 | 750 | 1,500 | 2,000 | 3,000 | 64,655 | 187,461 | 362,025 | 554,625 | 702,809 |
| **Expenses** | | | | | | | | | | | | | | | | | | | | |
| Cost of Goods Sold | 309 | 410 | 424 | 587 | 1,730 | | | | | | | - | - | - | - | 6,016 | 18,146 | 35,503 | 54,713 | 69,281 |
| %COGS | | | | | | | | | | | | | | | | 10% | 10% | 10% | 10% | 10% |
| Research and Development | 3,208 | 4,076 | 3,937 | 3,785 | 15,006 | 3,795 | 3,960 | 4,290 | 4,455 | 16,500 | 16,665 | 16,832 | 17,000 | 17,170 | 17,342 | 17,515 | 17,690 | 17,867 | 18,046 | 18,226 |
| %R&D | | | | | | | | | | | | | | | | | | | | |
| Selling and marketing | 3,058 | 3,129 | 2,941 | 2,396 | 11,524 | 1,380 | 1,440 | 1,560 | 1,620 | 6,000 | 5,500 | 5,555 | 5,611 | 5,667 | 5,723 | 5,781 | 5,838 | 5,897 | 5,956 | 6,015 |
| General and Administrative | 4,004 | 2,469 | 2,711 | 2,292 | 11,476 | 2,300 | 2,400 | 2,600 | 2,700 | 10,000 | 9,000 | 9,090 | 9,181 | 9,273 | 9,365 | 9,459 | 9,554 | 9,649 | 9,746 | 9,843 |
| %SG&A | | | | | | | | | | | | | | | | | | | | |
| Restructuring charges | | | | 790 | 790 | | | | | | | | | | | | | | | |
| **Total expenses** | 10,579 | 10,084 | 10,013 | 9,850 | 40,526 | 7,475 | 7,800 | 8,450 | 8,775 | 32,500 | 31,165 | 31,477 | 31,791 | 32,109 | 32,430 | 38,770 | 51,228 | 68,916 | 88,460 | 103,366 |
| Operating Income (Loss) | (10,459) | (9,980) | (9,924) | (9,782) | (40,145) | (7,475) | (7,800) | (8,450) | (8,775) | (32,500) | (31,165) | (31,477) | (31,791) | (32,109) | (32,430) | 21,385 | 130,233 | 286,109 | 458,665 | 589,444 |
| Interest income | | | | | | | | | | | | | | | | | | | | |
| Interest expense | (338) | (275) | (355) | (408) | (1,376) | | | | | | | | | | | | | | | |
| Gain on disposal of equipment | | | | | | | | | | | | | | | | | | | | |
| (Loss) gain from change in fair value of derivative instruments-warrants | 534 | 53 | 88 | 1,787 | 2,462 | | | | | | | | | | | | | | | |
| Other income | | | | (145) | (145) | | | | | | | | | | | | | | | |
| **Total other income** | **196** | **(222)** | **(267)** | **1,234** | **941** | **-** | **-** | **-** | **-** | **-** | **-** | **-** | **-** | **-** | **-** | **-** | **-** | **-** | **-** | **-** |
| **Pretax Income** | (10,263) | (10,202) | (10,191) | (8,548) | (39,204) | (7,475) | (7,800) | (8,450) | (8,775) | (32,500) | (31,165) | (31,477) | (31,791) | (32,109) | (32,430) | 21,385 | 130,233 | 286,109 | 458,665 | 589,444 |
| Preferred stock dividend | (6) | (6) | (6) | (6) | (24) | | | | | | | | | | | | | | | |
| Income Tax Benefit (Provision) | - | - | - | - | - | - | - | - | - | - | | - | - | - | - | - | - | - | 22,933 | 47,155 |
| Tax Rate | | | | | | | | | | | | | | | | | | | 5% | 8% |
| **GAAP Net Income (loss)** | (10,269) | (10,208) | (10,197) | (8,554) | (39,228) | (7,475) | (7,800) | (8,450) | (8,775) | (32,500) | (31,165) | (31,477) | (31,791) | (32,109) | (32,430) | 21,385 | 130,233 | 286,109 | 435,732 | 542,288 |
| **GAAP-EPS** | (0.35) | (0.34) | (0.34) | (0.28) | (1.30) | (0.24) | (0.25) | (0.27) | (0.29) | (1.06) | (0.92) | (0.87) | (0.81) | (0.79) | (0.76) | 0.49 | 2.87 | 6.29 | 9.54 | 11.83 |
| GAAP EPS (dil) | (0.34) | (0.34) | (0.34) | (0.28) | (1.30) | (0.24) | (0.25) | (0.27) | (0.29) | (1.06) | (0.92) | (0.87) | (0.81) | (0.79) | (0.76) | 0.49 | 2.87 | 6.29 | 9.54 | 11.83 |
| Wgtd Avg Shrs (Bas) - '000s | 29,755 | 29,958 | 30,340 | 30,639 | 30,175 | 30,670 | 30,700 | 30,731 | 30,762 | 30,716 | 33,842 | 36,231 | 39,131 | 40,414 | 42,455 | 43,875 | 45,304 | 45,486 | 45,668 | 45,851 |
| Wgtd Avg Shrs (Dil) - '000s | 30,108 | 29,958 | 30,340 | 30,712 | 30,281 | 30,670 | 30,700 | 30,731 | 30,762 | 30,716 | 33,842 | 36,231 | 39,131 | 40,414 | 42,455 | 43,875 | 45,304 | 45,486 | 45,668 | 45,851 |

Source: Company reports and Maxim

## DISCLOSURES

**Trovagene, Inc. Rating History as of 04/11/2017**

powered by: BlueMatrix


| Maxim Group LLC Ratings Distribution | | | As of: 04/11/17 |
|---|---|---|---|
| | | % 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. | **74%** | **31%** |
| **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. | **23%** | **17%** |
| **Sell** | Fundamental metrics and/or identifiable catalysts exist such that we expect the stock to underperform its relevant index over the next 12 months. | **2%** | **20%** |
| | **See valuation section for company specific relevant indices* | | |

I, Jason McCarthy, Ph.D., attest that the views expressed in this research report accurately reflect my personal views about the subject security and issuer. Furthermore, no part of my compensation was, is, or will be directly or indirectly related to the specific recommendation or views expressed in this research report.

I, Jason Kolbert, attest that the views expressed in this research report accurately reflect my personal views about the subject security and issuer. Furthermore, no part of my compensation was, is, or will be directly or indirectly related to the specific recommendation or views expressed in this research report.

The research analyst(s) primarily responsible for the preparation of this research report have received compensation based upon various factors, including the firm's total revenues, a portion of which is generated by investment banking activities.

**Maxim Group makes a market in Trovagene, Inc.**

**Maxim Group expects to receive or intends to seek compensation for investment banking services from Trovagene, Inc. in the next 3 months.**

**TROV:** For Trovagene, Inc., we use the BTK (Biotechnology index) as its relevant index.

**Valuation Methods**

**TROV:** Our model assumes modest revenue from out-licensure of diagnostics assets and retained CLIA lab services for pharma companies. We factor in PCM-075 in AML, with commercialization in the U.S. in 2023, follwed by Europe in 2024. Our model assumes a risk adjusmtment based

on stage of development. Our 12-month price target for Trovagene, Inc. is based on FCFF, dEPS and SOP models, which use a 30% discount rate, equally weighted and averaged.

**Price Target and Investment Risks**

**TROV:** Aside from general market and other economic risks, risks particular to our price target and rating for Trovagene, Inc. include: (1) The companies small molecule oncology therapeutics may not be successful in clinical trials and may not reach commercialization (1) the company could fail to develop new technology, commercialize new products, or execute on growth initiatives; (2) reimbursement or other pricing pressures could negatively impact the company's margins and earnings; (3) an inability to obtain attractive financing could dilute existing shareholders; (4) the U.S. Food and Drug Administration (FDA) could exercise its jurisdictional authority with respect to the regulation of laboratory-developed tests (LDTs) as in vitro diagnostics; (5) the company could fail to adequately protect its intellectual property; (6) molecular diagnostic market growth could slow, driven by reduced investment by diagnostic labs, a lack of payer reimbursement, or a lack of physician education and acceptance; (7) legal proceedings arising from government regulations, patent infringement, class action litigation, or individual litigation could negatively impact bottom-line growth; and (8) if the company misses revenue and EPS estimates and/or Street consensus during any given time period, the stock price could be negatively impacted.

## RISK RATINGS

Risk ratings take into account both fundamental criteria and price volatility.

**Speculative** – Fundamental Criteria: This is a risk rating assigned to early-stage companies with minimal to no revenues, lack of earnings, balance sheet concerns, and/or a short operating history. Accordingly, fundamental risk is expected to be significantly above the industry. Price Volatility: Because of the inherent fundamental criteria of the companies falling within this risk category, the price volatility is expected to be significant with the possibility that the investment could eventually be worthless. Speculative stocks may not be suitable for a significant class of individual investors.

**High** – Fundamental Criteria: This is a risk rating assigned to companies having below-average revenue and earnings visibility, negative cash flow, and low market cap or public float. Accordingly, fundamental risk is expected to be above the industry. Price Volatility: The price volatility of companies falling within this category is expected to be above the industry. High-risk stocks may not be suitable for a significant class of individual investors.

**Medium** – Fundamental Criteria: This is a risk rating assigned to companies that may have average revenue and earnings visibility, positive cash flow, and is fairly liquid. Accordingly, both price volatility and fundamental risk are expected to approximate the industry average.

**Low** – Fundamental Criteria: This is a risk rating assigned to companies that may have above-average revenue and earnings visibility, positive cash flow, and is fairly liquid. Accordingly, both price volatility and fundamental risk are expected to be below the industry.

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