MetaStat, Inc.
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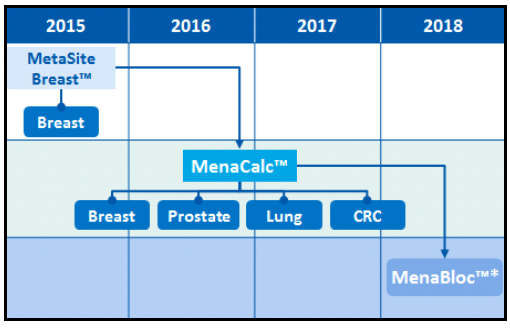
Ticker (Exchange)	MTST (OTC)
Recent Price (04/23/2014)	$1.10
52-week Range	$1.01 - $3.00
Shares Outstanding	~21.6 million
Market Capitalization	~$23.8 million
Average 3-month Volume	27,968
Insider Ownership +>5%	34.2%
Institutional Ownership	11.2%
EPS (Qtr. ended 11/30/2013)	($0.07)
Employees	7

MetaStat's Product Development Roadmap



Company Description

MetaStat, Inc. ("MetaStat" or "the Company") is a life sciences company commercializing a new approach to reliably determine a patient's individual risk of developing **systemic† metastatic** cancer and then to help reduce this risk through active intervention of the metastatic process. The Company's technology is based on a proprietary knowledge of the **mechanisms** that govern "**metastasis**" (the spread of cancer away from its primary site in the body). The technology centers on the role of the **Mena** protein in tumors, and has over 15 years of study from major medical institutions. MetaStat is initially advancing two diagnostic platforms targeting breast, prostate, lung, and colorectal cancers, which could enter the market as early as 2015, as well as a therapeutic program. The Company believes that its function-based diagnostics can lead to better treatment decisions by identifying patients with a high risk of systemic metastasis who require aggressive therapy and sparing patients with a low risk of metastasis from painful and costly therapies. The National Institutes of Health (NIH) recently concluded that a primary goal in cancer research should be to accurately define patient risk categories with the goal of being able to administer the level of treatment needed for a successful outcome.

Key Points

- In December 2013, MetaStat presented results of a large-scale study of its MetaSite *Breast* test at the San Antonio Breast Cancer Symposium, which demonstrated the test's value in predicting metastatic disease independent of other variables.
- In October 2013, the Company opened a drug discovery laboratory in affiliation with Stony Brook University in order to advance its MenaBloc therapeutic program.
- MetaStat also entered into two license agreements with MIT and other institutions for the use of **alternatively spliced mRNA** and protein **isoform** markers in the diagnosis, prognosis, and treatment of metastatic, **epithelial**-based solid tumors. This technology is leading the Company's therapeutic development.
- MetaStat has recently recruited notable individuals to its leadership, including a former senior executive from Roche, and established a highly skilled Scientific Advisory Board for Therapeutics to complement the existing Scientific Advisory Board for Diagnostics and Clinical Advisory Board.
- The Company holds rights to three issued U.S. patents and 10 patent applications pending globally.
- As of November 30, 2013, MetaStat had $488,108 in cash. The Company seeks $5-$7 million to reach key milestones or $12-$15 million for full commercialization of MetaSite *Breast* with major R&D progress on its MenaCalc and MenaBloc platforms.

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Executive Overview

MetaStat, Inc. ("MetaStat" or "the Company") is a development-stage life sciences company working to commercialize innovative cancer diagnostics as well as create new cancer therapeutics focusing on alternatively spliced proteins that have been found to drive both tumor growth and the metastatic spread of tumors. The Company's lead initiatives include its MetaSite *Breast* and MenaCalc diagnostic products as well as its MenaBloc therapeutic program. Each of these programs is built upon MetaStat's patent-protected understanding of the role of Mena in the body, which is a key protein for determining the aggressiveness of individual tumors.

Mena has been recognized as an embryonic protein that participates in the development of both the central and peripheral nervous system. It is typically only present in embryos, when it is vital to the development of the nervous system. Postpartum, its expression is heavily suppressed, becoming scarce and undetectable in healthy adults. However, scientists from MetaStat have discovered that, in approximately 80% of all solid cancers of epithelial cell origin, Mena protein is re-expressed and the relative expression of this protein determines the metastatic potential of an individual's cancer. MetaStat has identified and characterized five alternatively spliced Mena isoforms (different forms of the Mena protein). These isoforms, while only having minute differences, may actually determine which tumors have metastatic potential and which tumors are incapable of spreading through the blood vessels to distant sites in the body.

To this end, MetaStat holds platform technologies designed to enable the early and reliable prediction and treatment of systemic tumor metastasis, which are based on over 15 years of research from a consortium of scientists across the U.S. and Europe. The Company is now developing these technologies into commercial products targeting four of the five largest solid cancer markets: breast, lung, prostate, and colorectal cancers.

Tumor Metastasis

While not everyone who is diagnosed with cancer develops metastatic disease, for those who do, the cancer is much more difficult to treat and is far more likely to be fatal. "Metastasis" refers to the growth of secondary tumors away from the primary tumor location, and it is responsible for approximately 90% of cancer fatalities (Source: CancerQuest, Emory University's cancer education and outreach program). Virtually all types of cancer can develop into aggressive metastatic tumors.

Despite the well-documented relationship between metastatic spread and mortality, there are few to no options at present for determining which patient's cancer is at a higher risk of metastasis based on the tumor's unique mechanistic markers. Such knowledge could change the course of treatment for cancer, ideally preventing or effectively managing metastasis in a manner that improves the likelihood of survival.

Each of MetaStat's major programs is outlined below, with details provided in the Core Story on pages 20-48.

DIAGNOSTICS

MetaSite *Breast*

MetaSite *Breast* is a clinical laboratory **assay** (or test) to predict the likelihood of an early-stage breast cancer patient's tumor spreading to distant body parts. MetaSite *Breast* employs conventional **immunostaining** techniques to highlight unique three-cell structures in a tumor tissue **sample**. The three-cell structure (termed a "MetaSite") is composed of a **macrophage** cell, a carcinoma cell expressing the Mena protein, and an endothelial cell. This structure was identified by scientists from MIT, the Albert Einstein College of Medicine, and Weill Cornell Medical College, who reasoned that the density of MetaSites was correlated to the probability of distant tumor metastases. Research is showing that the three-celled MetaSite has a crucial role in allowing metastatic cells to enter into the bloodstream and spread through the blood to other organs in the body.

With MetaSite *Breast*, pathologists can clearly see and count the amount of MetaSites present in a sample of breast tumor tissue. Samples with a high density of MetaSites have been shown to correlate to a higher risk of metastasis. Importantly, the MetaSite *Breast* test does not require any special equipment, techniques, or procedures, and as such, is designed to be seamlessly incorporated into the standard procedures for analyzing tumor stage and grade. The test returns a "Metastasis Score" based on the number of MetaSites, and is accompanied by a rating scale used to classify each patient's test results as low, medium, or high risk as well as an interpretation of what the score indicates.

Clinical Studies

MetaSite *Breast* is the Company's first product opportunity. During 2013, MetaStat completed a favorable confirmatory trial in nearly 500 breast cancer patients using the MetaSite *Breast* diagnostic assay, for which results were presented at the 2013 San Antonio Breast Cancer Symposium. Among other findings, the trial confirmed the **prognostic** value of the MetaSite *Breast* platform technology by showing a positive correlation between the presence of MetaSites and the risk of distant metastasis in women with ER+/**HER2-negative** breast cancer (which is a strain of breast tumor that accounts for over 60% of all breast cancers) (Source: *Tumor Microenvironment of Metastasis and Risk of Distant Metastasis of Breast Cancer* poster from the 2013 San Antonio Breast Cancer Symposium).

MetaSite *Breast* has previously been validated in two other human clinical studies, both of which demonstrated a correlation between MetaSite density and metastasis and supported the test's ability to predict metastasis independently of conventional prognostic indicators, including tumor size, grade, lymph node metastasis, **lymphovascular invasion**, or hormone receptor status. Results of these studies are provided on pages 33-36, which include results from an unpublished study of a comparison to Genomic Health, Inc.'s (GHDX-NASDAQ) Onco*type* DX® Breast Cancer Assay. Notably, high-risk patients identified by MetaSite *Breast* were 22 times more likely to experience metastasis than people who the test deemed to be low-risk. In contrast, Onco*type* DX's high-risk group was only 4.5 times more likely to recur than the low-risk group.

Product Differentiation

An important distinction between MetaStat's technology and whole genome-based assays such as Onco*type* DX® and others is that MetaStat focuses on predicting the risk of cancer metastasis based on the tumor's underlying mechanisms. Historically, cancer cells have entered the blood vessels via unknown means. The research supporting the Company's technology has sought to identify the structural and behavioral mechanisms that allow cancer cells to move and determine how this information can be used in prognosis. To MetaStat's knowledge, its technology is the only technique to focus on such mechanistic markers.

Initially, the Company anticipates that its MetaSite *Breast* test, which analyzes metastatic risk by taking mechanistic factors of metastasis into account, could be used in conjunction with existing diagnostics, including the Onco*type* DX® Breast Cancer Assay, which largely estimate metastatic risk based on cancer cell proliferation. As further data for MetaSite *Breast* is published and the test gains awareness in the clinical community over time, MetaStat believes that it could ultimately be used as a standalone product—potentially becoming directly competitive to existing assays at a lower cost than currently available products.

MenaCalc Platform

The MenaCalc diagnostic technology platform is intended for use in determining individual levels of variants of the Mena protein (also called "Mena isoforms") in cancer tissue. Mena has at least five isoforms, and measuring the relationship between these variants can help create an individual metastatic profile as early on in disease progression as possible. With MenaCalc, tumor cells extracted from a patient through a biopsy can be evaluated for the presence and ratio of the various Mena isoforms in order to find an initial "MenaCalc Metastasis Score." Over time, patients' Mena isoform profiles could identify trends and detect stability or progression of disease as well as detect the efficacy of various therapies in real time.

MetaStat is developing multiple product candidates based on the MenaCalc platform to target common epithelial cancers: (1) MenaCalc *Breast*; (2) MenaCalc *Lung*; (3) MenaCalc *Prostate*; and (4) MenaCalc *Colorectal*. The Company reports that research to date in breast cancer has illustrated a correlation between the MenaCalc *Breast* Metastasis Score and the MetaSite *Breast* Metastasis Score, which was confirmed through a 797-patient study conducted at MIT, the Yale University School of Medicine, and the Albert Einstein College of Medicine (Source: Agarwal et al., *Breast Cancer Research* 2012; 14[5]:R124).

In addition, an abstract presented at the 2014 Annual Meeting of the U.S. and Canadian Academy of Pathology further confirmed the potential of the MenaCalc platform. Researchers from the University of Toronto, Yale, MIT, and Albert Einstein College of Medicine applied MenaCalc to 406 breast cancer tissue samples from individuals who had been patients at eight Toronto hospitals between 1987 and 1996. The results showed that high MenaCalc levels were associated with decreased overall survival in axillary node-negative breast cancer patients independent of other factors, indicating that MenaCalc may be an effective prognostic biomarker for this patient population (Source: *Mena^{calc}, a Quantitative Method of Metastasis Assessment, as a Prognostic Marker for Axillary Node-Negative Breast Cancer*, 2014). This team of 10 investigators included Dr. John Condeelis, Dr. Frank Gertler, and Dr. Thomas Rohan who together comprise MetaStat's Scientific Advisory Board for Diagnostics.

MetaStat's development plans for MenaCalc going forward include conducting several confirmatory clinical studies for its MenaCalc product lines: (1) a large-population validation study of the MenaCalc *Breast* test with metastatic risk as primary endpoint during 2014; (2) a large-scale proof-of-concept study in **adenocarcinoma** of the lung during 2015/2016 to confirm earlier findings that MenaCalc predicts cancer spread and survival in lung adenocarcinomas; and (3) a confirmatory proof-of-concept trial in 2015 for the MenaCalc *Prostate* candidate, which builds upon a favorable pilot study with this product candidate that was completed at MIT.

THERAPEUTICS

MenaBloc Therapeutic Program

In December 2013, MetaStat licensed a collection of alternatively spliced therapeutic targets that have a role in the epithelial to mesenchymal transition (EMT) of tumor cells. EMT is an early event in the metastatic process, which also contributes to therapeutic resistance in breast and other cancers. Scientific research has shown that EMT–dependent splicing changes occur in human breast cancer tumors, and have the ability to impact tumor progression and resistance (Source: Shapiro et. al., *PLoS Genetics*, August 2011, Volume 7, Issue 8). MetaStat believes this discovery presents a novel opportunity for new cancer therapeutics that target alternatively spliced **oncogenes**.

The license agreements include patent and technology licenses from MIT, the Koch Institute for Integrative Cancer Research, the Albert Einstein College of Medicine, and the Montefiore Medical Center, and include the use of alternatively spliced mRNA and protein isoform markers in the diagnosis, prognosis, and treatment of metastatic, epithelial-based solid tumors.

As evidenced by the high mortality rate of metastatic cancers, there is an unmet medical need for therapies based on a solid understanding of the process of metastatic disease, including techniques to kill or stop the spread of metastatic cancer cells or to disrupt individual steps in the metastatic process. The Company believes that commercializing its MenaCalc diagnostic products in multiple tumor types would teach the market about the importance of understanding the Mena protein when diagnosing and treating cancer, and could thus help aid adoption of therapeutics designed to down-regulate Mena expression. MetaStat anticipates that a MenaBloc therapeutic could ultimately prevent metastasis among high-risk patients when it is administered as a maintenance therapy after surgery or in conjunction with **chemotherapy** and other targeted therapies. Moreover, under the MIT license agreements from December 2013, MetaStat has the potential to develop companion diagnostic and therapeutic products, which could both detect alternatively spliced isoforms and then be linked to a therapeutic to address the isoforms in order to delay patients' tumor progression and decrease metastatic spread.

Appointing Industry Experts to Lead Therapeutic Development

Since Crystal Research Associates published its first Executive Informational Overview® (EIO) on MetaStat in January 2013, the Company has made considerable progress on both the diagnostics and therapeutics sides of its business. This progress commenced with Chief Executive Officer Dr. Oscar M. Bronsther's commitment in 2013 to strengthening corporate leadership by appointing skilled individuals from the oncology and life sciences fields. In February 2014, the Company also launched a Scientific Advisory Board (SAB) for Therapeutics, which complements the existing SAB for Diagnostics and Clinical Advisory Board. Pages 13-19 detail the biographies and experience of the Company's executive management and advisors, which include recent additions of Dr. Heiner Dreismann as the head of diagnostics and Dr. David M. Epstein as the chairman of the newly formed SAB for Therapeutics.

Dr. Dreismann was previously the president and CEO of Roche Molecular Systems and head of global business development for Roche Diagnostics. Dr. Epstein is the former senior vice president and chief scientific officer (CSO) for OSI Pharmaceuticals, Inc. (acquired by Astellas Pharma Inc. [4503-Toyko] in 2010 for roughly $4 billion) and is currently an associate professor in cancer and stem cell biology at Duke-NUS Medical School where he is also director of the Center for Technology and Development.

Likewise, the recently appointed members of MetaStat's SAB for Therapeutics are skilled in the fields of alternatively spliced proteins, therapeutic resistance in cancer, and **RNA** biology. These advisors include the individuals listed below (complete biographies provided on pages 16-17).

- Dr. Eric Winer, a Harvard Medical School professor and chief of the Division of Women's Cancers at the Dana-Farber Cancer Institute
- Dr. Adrian Krainer, from the Cold Spring Harbor Laboratory, who discovered the SRSF1 splicing factor
- Dr. Mariano Garcia-Blanco, the director of the Center for RNA Biology at Duke University, who has discovered the role of alternative splicing in tumor progression and metastasis pathways
- Dr. Michael Hemann, an associate professor of biology at MIT with expertise in modeling tumors' therapeutic resistance to drugs in order to identify new drug targets
- Dr. Frank Gertler, a professor in the Koch Institute for Integrative Cancer Research at MIT, who has over two decades of experience working with Mena and who is also on MetaStat's SAB for Diagnostics

Establishing a Drug Discovery Laboratory for Therapeutics Products

MetaStat opened a drug discovery laboratory at Stony Brook University's Long Island High-Technology Incubator in September 2013, which became operational in October 2013. The incubator is designed for early stage, high-tech companies during their development and growth periods. It has housed over 70 companies in the past 16 years, which the Long Island High-Technology Incubator reports have a high survival rate upon leaving the program (Source: Stony Brook Research, www.stonybrook.edu/research/vpr/incubators.shtml).

MetaStat's laboratory is staffed with experienced Ph.D.'s formerly from Dr. Epstein's team at OSI Pharmaceuticals, where they were focused on molecular, targeted cancer therapies. The laboratory's leadership includes Dr. Elizabeth Buck and Mr. Matthew O'Connor (MetaStat's lead scientist of therapeutics) among other research scientists. Dr. Buck brings to MetaStat experience in drug discovery as well as due diligence for in-licensing opportunities and the evaluation of technology for corporate collaborations. Previously, she was the assistant director of advanced preclinical pharmacology at OSI Pharmaceuticals, where she led a scientific team focused on preclinical pharmacology and translational research. She is experienced in advancing preclinical oncology molecules to the Investigational New Drug (IND) submission stage. Mr. O'Conner is also a skilled research scientist in oncology drug discovery and development, translational research, clinical biomarkers, and management.

HEADQUARTERS AND EMPLOYEES

MetaStat was incorporated in Texas in 2009 and re-incorporated in Delaware in 2010. The Company has principal executive offices in Montclair, New Jersey, and a drug discovery laboratory in Stony Brook, New York. The Company trades as "MTST" on the Over-the-Counter (OTC.QB) exchange.

The Company employs seven full-time individuals as well as Drs. Dreismann and Epstein as consultants. MetaStat also has relationships with numerous medical doctors, scientists, and engineers—some of whom are consultants and some of whom are full-time researchers from MIT and other institutions, funded by MetaStat's research and development collaborations. For example, the Company's recent large-population validation study for MetaSite *Breast* was conducted at MIT, the Albert Einstein College of Medicine, and Weill Cornell Medical College, and studies using MenaCalc for breast and lung have been performed by MIT, the Albert Einstein College of Medicine, and Yale University.

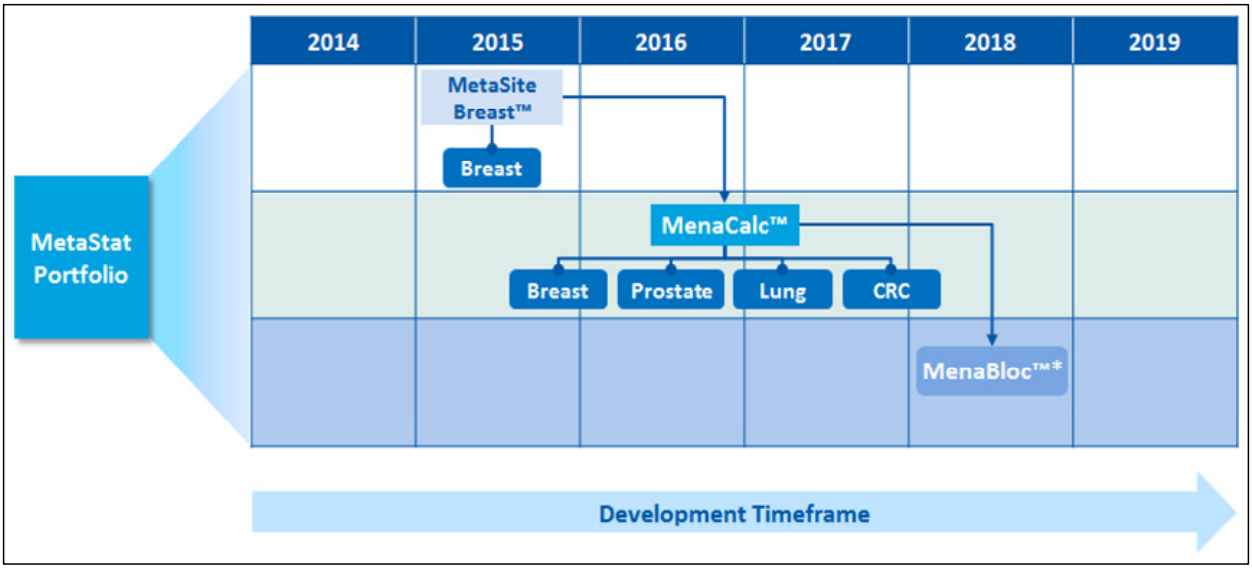
Growth Strategy

Product Development Strategies and Milestones

MetaStat is following a tiered strategy with regard to the launch of its diagnostic and therapeutic products. The Company intends to first launch MetaSite *Breast* in order to establish its corporate brand and generate the cash necessary to fully develop and commercialize MenaCalc, which in itself could help create a market for the therapeutic MenaBloc candidate. The Company's anticipated development timeline for its three core programs is overviewed in Figure 1 and detailed following the Figure.

Figure 1

PRODUCT ROADMAP: COMMERCIALIZATION PATH IN 2015 AND BEYOND



MetaSite *Breast:*	A diagnostic platform to predict the likelihood of cancer spreading based on the density of "MetaSites"
MenaCalc:	A diagnostic platform to predict metastasis using measurement of the relative levels of Mena protein isoforms in a tumor sample
MenaBloc:	Small molecule therapeutics to slow or potentially shut down the metastatic process

Source: MetaStat, Inc.

MetaSite Breast

In October 2013, MetaStat reported that its MetaSite *Breast* test was found to have value in the prediction of metastatic disease in a nearly 500-patient, large-scale validation study. Results of this study have been submitted for publication and were presented in December 2013 at the San Antonio Breast Cancer Symposium. Initial findings, as reported in a poster presentation at the Symposium, are presented on page 36. The Company plans to initiate an additional validation study and chemotherapy benefit trial with certain identified cohort(s).

MetaStat further anticipates establishing a central laboratory that meets both Clinical Laboratory Improvement Amendments (CLIA) and Good Laboratory Practice (GLP) standards. This would likely entail a separate laboratory located at the Company's existing drug discovery laboratory facility, as there could be a synergy between the two labs and significant benefits for having them in proximity to each other.

The MetaSite *Breast* test is MetaStat's closest candidate to market, and the Company aims to commence marketing in 2015. Details of MetaStat's initial marketing program for MetaSite *Breast* are provided on page 39. MetaStat is exploring two strategic options for full commercialization of the MetaSite *Breast* test: (1) either establishing internal sales and marketing and other functions needed to sell the product candidate solely from MetaStat; or (2) partnering with another entity to commercialize the product in order to benefit from a partner's capabilities in laboratory services, sales and marketing, or pricing and reimbursement.

MenaCalc

MetaStat plans to launch MenaCalc *Breast* during 2015, following a large-population validation study of the test during 2014. MenaCalc programs for lung and prostate cancer are expected to be in clinical trials by 2015/2016.

MenaBloc

With the opening of a drug discovery laboratory in October 2013 in Stony Brook and the hiring of a team of highly experienced scientists, MetaStat is ahead of schedule on its therapeutic initiatives. Over the next 12-24 months, the Company aims to perform a functional screening program for a small molecule Mena inhibitor, and complete medicinal chemistry and lead optimization with the goal of entering a MenaBloc therapeutic into Phase I clinical trials within three to four years.

Figure 2

TIERED GROWTH STRATEGY

Source: MetaStat, Inc.

Fundraising

As with most development-stage companies, fundraising is a crucial component of MetaStat's growth strategy. The Company estimates that it needs between $5 million and $7 million to reach important milestones. A larger amount of $12 million to $15 million would likely take MetaStat through two years of operations, including commercialization of the first diagnostic product, completion of R&D on a second diagnostic candidate, and through the 18 months that the Company estimates it needs to reach important therapeutic program milestones.

Milestones

Recent Milestones

MetaStat continues to advance toward introducing innovative new products to diagnose and treat metastatic disease, as shown by the progress made over the past year.

- Entered into exclusive worldwide patent and technology license agreements with MIT, including MIT's David H. Koch Institute for Integrative Cancer Research and MIT's Department of Biology; the Albert Einstein College of Medicine; and the Montefiore Medical Center, which are key to MetaStat's efforts for developing companion diagnostic and therapeutic products

- Issued two key U.S. patents in late 2013/early 2014 that cover the MenaCalc diagnostic platform as well as the MetaSite *Breast* diagnostic assay—for which the earliest patent does not expire until 2029

- Established a Scientific Advisory Board for Therapeutics to aid development of the Company's therapeutic program focused on alternatively spliced proteins. Appointments to the Board include scientific and clinical leaders from the fields of RNA biology, alternative splicing, and therapeutic resistance in cancer, notably Dr. David M. Epstein, an associate professor in cancer and stem cell biology and director of the Center for Technology and Development at the Duke-NUS Medical School in Singapore and former senior vice president and CSO for oncology research at OSI Pharmaceuticals. Other members on MetaStat's SAB for Therapeutics are equally as accomplished in the field, and are credited with discoveries related to alternative splicing and cancer research. Biographies are provided on pages 16-17.

- Recruited Dr. Heiner Dreismann, former president and CEO of Roche Molecular Systems and the head of global business development for Roche Diagnostics, as the head of diagnostics for MetaStat

- Recruited Dr. Elizabeth Buck as the CSO for therapeutics and Mr. Matthew O'Connor as lead scientist of therapeutics, both of whom are formerly of OSI Pharmaceuticals, to work in collaboration with Dr. Epstein on MetaStat's therapeutic drug development

- Opened a drug discovery laboratory in affiliation with Stony Brook University's incubators in Stony Brook, New York

- Completed a 500-patient confirmatory trial in breast cancer using the MetaSite *Breast* assay, and presented data at the 2013 San Antonio Breast Cancer Symposium in Poster Session P2-11-03

- Received a third-party, published verification of MenaCalc in March 2014 that validated the MenaCalc platform by demonstrating that high MenaCalc levels were associated with decreased overall survival in axillary node-negative breast cancer patients independent of other factors, leading researchers to state this could be a "useful independent prognostic biomarker" in this patient population (Source: *Mena^{calc}, a Quantitative Method of Metastasis Assessment, as a Prognostic Marker for Axillary Node-Negative Breast Cancer*, 2014)

- Granted eligibility status by the Depository Trust Corporation (DTC) as of February 20, 2013, which the Company expects could simplify the process of trading and exchanging its common stock

- Completed private placements with accredited investors for total gross proceeds of roughly $3 million

Intellectual Property

Broad patent protection is essential in the evolving and expanding field for metastasis treatment. MetaStat seeks domestic and international coverage for its technologies, and invests in qualified patent counsel. Much of MetaStat's current intellectual property is held under a license agreement with MIT, the Albert Einstein College of Medicine of Yeshiva University, Cornell University, and the IFO-Regina Elena Cancer Institute, as described below. The Company's portfolio spans over 15 years of research through these institutions.

Technology License Agreements

The platform technology employed by MetaStat for analyzing tissue samples, among other sponsored research initiatives, was initially developed by a consortium of scientific institutions: MIT, the Albert Einstein College of Medicine of Yeshiva University, Cornell University, and the IFO-Regina Elena Cancer Institute. The Company licensed rights to the technology from these institutions in August 2010 under a Patent and Technology License Agreement.

Altogether, the August 2010 license agreement served to provide MetaStat with global, exclusive rights to materials and methods developed by the institutions for use in diagnosing and treating the metastasis of solid tumor cancers. It covered pending patent applications, patent disclosures, cell lines, and technology associated with understanding the underlying mechanisms of metastasis in solid tumor epithelial cancers. In exchange, MetaStat issued equity to the licensors, reimbursed them for patent expenses, agreed to pay future patent expenses, annual license maintenance fees, future royalties, and milestone payments.

Separately, MetaStat also entered into a Sponsored Research Agreement with the Albert Einstein College of Medicine and Weill Cornell Medical College in April 2011 for the large-population validation study of the MetaSite *Breast* test, for which final payments to both colleges were made in September 2012.

In March 2012, MetaStat licensed additional pending patent applications, patent disclosures, cell lines, and related technology from the Albert Einstein College of Medicine. These patent rights were obtained via two license agreements between MetaStat and the College, both made effective in March 2012. Financial terms are similar to the initial agreement in August 2010, and include reimbursement of patent expenses, license maintenance fees, royalties, and milestone payments. These agreements provided drug developers at MetaStat and potential corporate partners with tools to develop anti-metastatic drugs, an artificial blood vessel technology to isolate, collect, genetically profile, and determine chemotherapeutic resistance of a pure population of metastatic cancer cells from a tumor, as well as includes a license to a patent that describes the actual causative gene signature of metastatic cells.

Most Recent Technology License Agreements: MIT Licenses

The Company's most recent technology license agreements were executed in December 2013 with MIT, including MIT's David H. Koch Institute for Integrative Cancer Research and MIT's Department of Biology, the Albert Einstein College of Medicine, and the Montefiore Medical Center. This transaction included two exclusive worldwide patent and technology license agreements—a Diagnostic License Agreement and a Therapeutic License Agreement. The agreements cover pending patent applications, patent disclosures, and technology surrounding discoveries of alternatively spliced mRNA and protein isoform markers for the diagnosis, prognosis, and treatment of metastasis in epithelial solid tumor cancers. These latest license agreements are key to MetaStat's efforts for developing companion diagnostic and therapeutic products, where the Company seeks to link a targeted therapeutic to its companion diagnostic based on the detection and targeting of alternatively spliced oncogenes.

Patent Coverage

MetaStat holds three issued U.S. patents and 10 pending patent applications globally. The most recently issued U.S. patents, from December 2013 and February 2014, protect the Company's lead programs—its MenaCalc diagnostic platform and its MetaSite *Breast* test. The MenaCalc patent (#8,603,738, expires in July 2029) covers the use of Mena isoforms for diagnosis and prognosis of metastatic epithelial cancers, including breast, pancreas, prostate, colon, brain, liver, lung, and head/neck, and the use of the isoforms for determining efficacy of oncologic drug candidates as well as potential use of MenaCalc as a companion diagnostic to anti-metastatic therapeutics. The MetaSite *Breast* patent (#8,642,277, expires in November 2031) covers the Company's diagnostic breast assay to detect and quantify the mechanisms by which metastatic cells spread through the bloodstream.

Figure 3

PATENT COVERAGE SUMMARY

Jurisdiction	Title	Type and Number	Status
	Issued Patents		
U.S.	Tumor microenvironment of metastasis (TMEM) and uses thereof in diagnosis, prognosis, and treatment of tumors	U.S. Patent No. 8,642,277	Issued: 2/4/2014
U.S.	Metastasis specific splice variants of Mena and uses thereof in diagnosis and prognosis of tumors	U.S. Patent No. 8,603,738	Issued: 12/10/2013
U.S.	Isolation, gene expression, and chemotherapeutic resistance of motile cancer cells	U.S. Patent No. 8,298,756	Issued: 10/30/2012
	Patent Applications		
U.S.	Metastasis specific splice variants of Mena and uses thereof in the treatment of tumors	Patent Application No. 14/074,089 Divisional of U.S. Pat. App. No. 12/462,324	Filed: 11/7/2013 Priority Date: 2/2/2007
Europe	Metastasis specific splice variants of Mena and uses thereof in diagnosis, prognosis, and treatment of tumors	Patent Application No. 08713370.8	Filed: 1/31/2008
Canada	Metastasis specific splice variants of Mena and uses thereof in diagnosis, prognosis, and treatment of tumors	Patent Application No. 2,676,179	Filed: 1/31/2008
Canada	Isolation, gene expression, and chemotherapeutic resistance of motile cancer cells	Patent Application No. 2,576,702	Filed: 8/4/2005
Europe	Method for identifying metastasis in motile cells	European Patent No. 1784646 / App. No. 05807467.5	Filed: 8/4/2005 Publication: 6/13/2012
U.S.	Human invasion signature for prognosis of metastatic risk	Patent Application No. 14/115,928	PCT Filed: 11/15/2012
U.S.	An in vivo quantitative screening test for anti-metastasis treatment efficacy	Patent Application No. 12/998,237	Filed: 10/28/2009
U.S.	Alternatively spliced mRNA isoforms as prognostic indicators for metastatic cancer	Patent Application No. 14/000,995	Filed: 2/24/2012
Europe	Alternatively spliced mRNA isoforms as prognostic indicators for metastatic cancer	Patent Application No. 12749944.0	Filed: 2/24/2012
Singapore	Alternatively spliced mRNA isoforms as prognostic indicators for metastatic cancer	Patent Application No. 201306378-9	Filed: 2/24/2012

Source: MetaStat, Inc.

Company Leadership

Management

MetaStat's recent management and Board additions are believed to be a testament to the quality of the Company's science and the progress that has been made over the past several years. Figure 4 summarizes the Company's executive leadership, followed by brief biographies. In addition, the Company's scientific and clinical advisors (profiled on pages 15-17) include individuals who have held key roles in the supporting research behind MetaStat's platform technologies over the past 15 years.

Figure 4
EXECUTIVE MANAGEMENT

Oscar L. Bronsther, M.D., F.A.C.S.	Chief Executive Officer, Chief Medical Officer, and Director
Heiner Dreismann, Ph.D.	Head of Diagnostics
Elizabeth Buck, Ph.D.	Chief Scientific Officer for Therapeutics
Warren C. Lau	President, Chief Financial Officer, and Director
Daniel H. Schneiderman	Vice President of Finance and Comptroller

Source: MetaStat, Inc.

Oscar L. Bronsther, M.D., F.A.C.S., Chief Executive Officer, Chief Medical Officer, and Director

Dr. Bronsther is a diplomat, American Board of Surgery, and is the chairman, Section of General Surgery, at Inova Fairfax Hospital. He is a clinical professor of surgery at George Washington University in Washington, D.C. From 2005 to 2007, he served as chairman of the Board of National Transplant Network. Dr. Bronsther received a B.A. from the University of Rochester in 1973, an M.D. from Downstate Medical Center in 1978, was a Fellow in Kidney Transplantation at Downstate Medical Center, and was a Fellow in Liver Transplantation at the University of Pittsburgh Center. His editorial positions include reviewer, *Journal of the American College of Surgeons*, *Transplantation*, *Transplant Proceedings*, *Liver Transplantation and Surgery*, and *American Journal of Kidney Disease*. Dr. Bronsther is the author of 63 peer-reviewed publications, seven books and book chapters, and has participated in over 30 invited lectures.

Heiner Dreismann, Ph.D., Head of Diagnostics

Dr. Dreismann joined MetaStat as head of diagnostics in October 2013. Dr. Dreismann has more than 24 years of experience in the healthcare industry, and is regarded as a pioneer in the early adoption of the polymerase chain reaction (PCR) technique, one of the most ubiquitous technologies in molecular biology and genetics research today. Dr. Dreismann had a successful career at the Roche Group from 1985 to 2006, where he held several senior positions, including president and CEO of Roche Molecular Systems, head of global business development for Roche Diagnostics, and member of Roche's Global Diagnostic Executive Committee. Dr. Dreismann currently serves on the boards of several public and private healthcare companies. He earned an M.S. in biology and a Ph.D. in microbiology/molecular biology (*summa cum laude*) from Westfaelische Wilhelms University (The University of Münster) in Germany.

Elizabeth Buck, Ph.D., Chief Scientific Officer for Therapeutics

Dr. Buck brings to the Company expertise in preclinical pharmacology, translational research, and biomarker discovery in oncology. Previously, she was assistant director of advanced preclinical pharmacology at OSI Pharmaceuticals, a wholly owned subsidiary of Astellas Pharma US Inc., where she served since 2005. In this role, Dr. Buck was responsible for the advancement of preclinical lead molecules in oncology to IND nomination. She drove the translational research and biomarker strategy for multiple programs including linsitinib, currently in Phase II development, and is author of more than 30 peer-reviewed publications and patents. Among Dr. Buck's research accomplishments is an improved understanding for how compensatory signaling between receptor tyrosine kinases can lead to resistance to cancer therapeutics including the EGFR inhibitor erlotinib. Dr. Buck received a B.S. in physics from the University of New Hampshire, a Ph.D. in pharmacology from New York University/Mount Sinai School of Medicine, and a fellowship at Sunesis Pharmaceuticals.

Warren C. Lau, Founder, President, Chief Financial Officer, and Director

Mr. Lau is MetaStat's founder, president, chief financial officer, and a director. He also served as chief executive officer (CEO) from the Company's formation in July 2009 through December 2012. From October 2005 to March 2008, Mr. Lau served as a director and as the founder, president, and CEO of HoustonPharma, Inc. Previously, he was the president and CEO as well as a director of Opexa Therapeutics (OPXA-NASDAQ), which formed as a result of the acquisition of Opexa Pharmaceuticals by PharmaFrontiers Corp., a company founded by Mr. Lau in February 2003. Mr. Lau was the founder of Adventrx Pharmaceuticals, Inc. (ANX-NYSE) in 1996. He served as its president and CEO and as a member of its Board of Directors from July 1996 through November 2001. During his time as president and CEO, Adventrx consummated two acquisitions, including Biokeys Pharmaceuticals, Inc. and Immune Complex Corporation, which was later spun off to shareholders. From November 1997 to September 1998, Mr. Lau served as a director of Immune Complex Corporation and Synthetic Genetics, Inc., both privately held biotechnology companies.

Daniel H. Schneiderman, Vice President of Finance, Comptroller, and Secretary

Mr. Schneiderman has served as MetaStat's vice president and comptroller since February 27, 2012. He was appointed vice president of finance in December 2012. Mr. Schneiderman has 10 years of investment banking and corporate finance experience. He has focused on private and public equity for small/mid-market capitalization companies mainly in the healthcare and life sciences sectors. During his career, Mr. Schneiderman has participated in public and private financings of approximately $500 million. Prior to joining MetaStat, he was senior vice president of investment banking for Burnham Hill Partners LLC, where he worked since 2008. From 2004 through 2008, Mr. Schneiderman was vice president of investment banking at Burnham Hill Partners, a division of Pali Capital, Inc. Previously, he worked at H.C. Wainwright & Co. in 2004 as an analyst. Mr. Schneiderman holds a Bachelor's degree from Tulane University.

Scientific Advisory Board for Diagnostics

Figure 5
SCIENTIFIC ADVISORY BOARD (SAB) FOR DIAGNOSTICS

John S. Condeelis, Ph.D.	Chairman of the SAB for Diagnostics
Frank B. Gertler, Ph.D.	Advisor, SAB for Diagnostics and SAB for Therapeutics
Thomas E. Rohan, M.D., Ph.D.	Advisor

Source: MetaStat, Inc.

John S. Condeelis, Ph.D., Chairman of the Scientific Advisory Board for Diagnostics

Dr. Condeelis is the Judith and Burton P. Resnick Chair in Translational Research, professor and co-chairman of the Department of Anatomy and Structural Biology at the Albert Einstein College of Medicine. He is the director of the Cancer Center program "Tumor Microenvironment and Metastasis" and co-director of the Gruss Lipper Biophotonics Center of the Albert Einstein College of Medicine. His current research interests are in tumor cell **motility**, chemotaxis, invasion, and intravasation during metastasis. He has combined multiphoton imaging with expression analysis to derive gene expression signatures. This Human Breast Cancer Invasion Signature defines the pathways used by tumor cells in mammary tumors to move and invade blood vessels. The tumor cells are followed using multiphoton imaging for these studies using novel caged-enzymes and biosensors to test, *in vivo*, the predictions of the invasion signature regarding the mechanisms of tumor cell chemotaxis to **epidermal growth factor (EGF)**. Dr. Condeelis has authored more than 250 scientific papers on various aspects of cell and cancer biology, prognostic marker development, and optical imaging.

Frank B. Gertler, Ph.D., Scientific Advisory Boards for Diagnostics and Therapeutics

Dr. Gertler received a B.S. from the University of Wisconsin-Madison in 1985. During his post-graduate thesis work at the University of Wisconsin-Madison, Dr. Gertler discovered the Enabled (Ena) gene in a search for functional downstream targets of signaling by the Drosophila homolog of the c-Abl proto-oncogene. He proceeded to demonstrate that Abl and Ena function were key components of the machinery required to establish normal connections during development of the nervous system. After receiving a Ph.D. in oncology and genetics in 1992, Dr. Gertler trained as a Postdoctoral Fellow in the laboratory of Philippe Soriano at the Fred Hutchinson Center for Cancer Research from 1993 through 1997. During this time, he cloned Mena, the mammalian homolog of Drosophila Ena, and discovered a family of related molecules, the "Ena/VASP" proteins. In 1997, Dr. Gertler joined the Biology Department at MIT. His laboratory continued to work on Mena and the related Ena/VASP proteins and described pivotal roles for these proteins in controlling cell movement, shape, and adhesion during fetal development. In 2005, Dr. Gertler moved to the MIT Center for Cancer Research and began to work on the role of Mena in metastatic progression and launched other efforts geared at understanding how the control of cell motility is dysregulated during metastatic diseases. Currently, Dr. Gertler is a full professor in the Koch Institute for Integrative Cancer Research at MIT and a member of the MIT Biology Department.

Thomas E. Rohan, M.D., Ph.D., Scientific Advisory Board for Diagnostics

Dr. Rohan is professor and chairman of the Department of Epidemiology and Population Health at Albert Einstein College of Medicine. He is also associate director for population sciences, program leader of the Cancer Epidemiology Program (CEP) and faculty director, Epidemiology Informatics Core Facility at the Albert Einstein Cancer Center. Dr. Rohan is an M.D. with a Ph.D. in epidemiology and an M.Sc. in medical statistics. He has published more than 300 scientific articles and two books on various aspects of epidemiology. He has a particular interest in the molecular pathogenesis of breast cancer. Dr. Rohan is associate editor of the journal *Cancer Epidemiology, Biomarkers, and Prevention* and several other journals, including a new journal, *Cancer Medicine*, which has a focus on personalized medicine. He has served on many grant review panels, served a four-year term on the Epidemiology of Cancer Study Section at the National Cancer Institute (NCI), and is currently a member of the Board of Scientific Counselors of NCI.

Scientific Advisory Board for Therapeutics

Figure 6
SCIENTIFIC ADVISORY BOARD (SAB) FOR THERAPEUTICS

David Epstein, Ph.D.	Chairman of the Scientific Advisory Board for Therapeutics and Director (Board of Directors)
Eric Winer, M.D.	Scientific Advisory Board for Therapeutics
Adrian Krainer, Ph.D.	Scientific Advisory Board for Therapeutics
Mariano A. Garcia-Blanco, M.D., Ph.D.	Scientific Advisory Board for Therapeutics
Michael T. Hemann, Ph.D.	Scientific Advisory Board for Therapeutics
Frank B. Gertler, Ph.D.	Scientific Advisory Boards for Diagnostics and Therapeutics

Source: MetaStat, Inc.

David Epstein, Ph.D., Chairman of the Scientific Advisory Board for Therapeutics and Director (Board of Directors)

Dr. Epstein was appointed to MetaStat's Board of Directors and as chair of the drug discovery and development team effective April 16, 2013, and as chairman of the Scientific Advisory Board for Therapeutics on February 4, 2014. Dr. Epstein is currently associate professor in cancer and stem cell biology and director of the Center for Technology and Development at the Duke-NUS Medical School in Singapore. From May 2006 to March 2013, Dr. Epstein served as senior vice president and CSO for OSI Pharmaceuticals, now a wholly owned subsidiary of Astellas Pharma US, Inc., where he had strategic and operational oversight of OSI's oncology discovery research and translational medicine programs. From May 2001 to April 2006, Dr. Epstein served as vice president of Archemix Corp., an **aptamer** therapeutics-focused discovery and development company, where he was responsible for overseeing Archemix's aptamer research and preclinical development programs. Dr. Epstein's experience is believed to give him a broad and deep understanding of the science underlying MetaStat's business and its competitors' efforts.

Eric Winer, M.D., Scientific Advisory Board for Therapeutics

Dr. Winer is professor, Department of Medicine, at Harvard Medical School. He is chief of the Division of Women's Cancers, director of the Breast Oncology Program, and the Thompson Chair in Breast Cancer Research at the Dana-Farber Cancer Institute. Dr. Winer received an M.D. from Yale University. Under his leadership, the program at Dana-Farber has played a critical role in the development of targeted therapies for **HER2-positive** breast cancer. The group at Dana-Farber is also investigating a wide range of targeted therapies for all subtypes of breast cancer. Dr. Winer has authored over 200 publications relating to clinical cancer research.

Adrian Krainer, Ph.D., Scientific Advisory Board for Therapeutics

Dr. Krainer is the St. Giles Foundation Professor of Molecular Genetics and the Program Chair of Cancer and Molecular Biology at the Cold Spring Harbor Laboratory. Dr. Krainer's expertise is in the fundamental mechanisms and regulation of human pre-mRNA splicing, and understanding the role of defective splicing in cancer. Dr. Krainer discovered SRSF1, the founding member of a conserved family of splicing factors, and his work has provided evidence of splicing factors driving cancer and the role of alternative splicing in cancer-cell metabolism. Dr. Krainer's laboratory has also developed novel antisense therapeutics to correct disease-causing splicing defects, and application of this method is currently being assessed in the clinic. Dr. Krainer is considered an expert in this area, with over 150 research articles published to date.

Mariano A. Garcia-Blanco, M.D., Ph.D., Scientific Advisory Board for Therapeutics

Dr. Garcia-Blanco is the Charles D. Watts professor of molecular genetics and microbiology, and medicine, and director of the Center for RNA Biology at Duke University. Dr. Garcia-Blanco's expertise is in RNA biology. His laboratory has pioneered the use of reporters to image alternative splicing of RNA *in vivo*. He has discovered the role of alternative splicing in epithelial to mesenchymal transition (EMT), a process essential for tumor progression and metastasis. A major focus of his laboratory is elucidating signaling pathways that mediate changes in alternative splicing as tumor cells undergo EMT.

Michael T. Hemann, Ph.D., Scientific Advisory Board for Therapeutics

Dr. Hemann is the Eisen and Chang Career Development associate professor of biology at MIT. Dr. Hemann brings expertise in modeling therapeutic resistance in order to identify new drug targets where inhibition can synergize with existing therapies. His laboratory uses RNAi to study the roles of cancer relevant genes in mediating sensitivity and resistance to chemotherapeutic agents. Dr. Hemann's interests are also focused on understanding the role of genetic instability on acquired and intrinsic drug resistance.

Frank B. Gertler, Ph.D., Scientific Advisory Boards for Diagnostics and Therapeutics

Biography provided on page 15.

Clinical Advisory Board

Joan Jones, M.D., Clinical Advisory Board

Dr. Jones is professor, Department of Pathology, Department of Anatomy and Structural Biology, Department of Epidemiology and Population Health at the Albert Einstein College of Medicine and is an attending pathologist at New York Presbyterian Hospital. Dr. Jones is a former professor of clinical pathology and laboratory medicine at Weill Cornell Medical College. She is an anatomic pathologist with clinical experience in breast pathology and an interest in the contribution of cell migration and the microvasculature to metastatic progression. Dr. Jones' work with the metastasis group at the Albert Einstein College of Medicine began in 1991 when parallels were first being drawn between events in amoeboid chemotaxis and the behavior of metastatic tumor cells. Her role has been to provide the histologic and human disease context for observations both in culture systems and animal models. Dr. Jones was one of the originators, along with Dr. Condeelis, on the use of intra-vital imaging (IVI) of live mammary tumors to identify vascular landmarks around which tumor cells migrate and intravasate. Dr. Jones' application of these IVI observations to human breast cancer samples led to confirmation of the concept of TMEM in humans, a microanatomic landmark consisting of a tumor cell, an endothelial cell, and a macrophage, initially observed *in vivo* in animals. She developed both the methodology and the approach to quantitation of this landmark in human samples. Dr. Jones continues to work on the application of Mena-related biomarkers and TMEM to the prediction of metastatic risk in breast cancer.

Joseph Sparano, M.D., Clinical Advisory Board

Dr. Sparano is professor of medicine and women's health at the Albert Einstein College of Medicine, associate director for clinical research at the Albert Einstein Cancer Center, and associate chairman of the Department of Oncology at Montefiore Medical Center. He is a medical oncologist and clinical researcher who has been involved in the development of numerous Phase I, II, and III NCI-sponsored, investigator-initiated, and industry-sponsored trials, with expertise in breast cancer, lymphoma, HIV-associated cancer, developmental therapeutics, and development and validation of prognostic and predictive biomarkers. He serves as chair of the Eastern Cooperative Oncology Group Breast Cancer Committee, vice-chair of the NCI Breast Cancer Correlative Science Committee, and member of the NCI Breast Cancer Steering Committee.

Board of Directors

The Board of Directors oversees the conduct of and supervises the Company's management. Figure 7 provides a summary of Board members, followed by detailed biographies.

Figure 7
BOARD OF DIRECTORS

Johan M. (Thijs) Spoor, MBA	Chairman of the Board
David Epstein, Ph.D.	Director on the Board of Directors and Chairman (Scientific Advisory Board for Therapeutics)
Oscar L. Bronsther, M.D., F.A.C.S.	Chief Executive Officer, Chief Medical Officer, and Director
Warren C. Lau	President, Chief Financial Officer, and Director
David N. Siegel	Director
Patrick T. Mooney, M.D.	Director

Source: MetaStat, Inc.

Johan M. (Thijs) Spoor, MBA, Chairman of the Board

Mr. Spoor was appointed to MetaStat's Board of Directors on February 27, 2012, effective as of April 7, 2012, and was appointed chairman of the Board on December 21, 2012. Mr. Spoor is currently the CEO, president, CFO, and director of FluoroPharma Medical Inc. (FPMI-OTC). He has held these positions at FluoroPharma since May 2011. Mr. Spoor holds a nuclear pharmacy degree from the University of Toronto as well as an MBA from Columbia University with concentrations in finance and accounting. Mr. Spoor has been a guest lecturer at Columbia Business School, Kings College in London, and the University of Newcastle in Australia. He previously held the title of CFO for Sunstone BioSciences, Inc. for the period from February 2010 through September 2010. Prior to joining Sunstone BioSciences, he worked as a consultant at Oliver Wyman from December 2008 through February 2010 focusing on helping pharmaceutical and medical device companies evaluate their global revenue potential given the complex interplay of regulatory approvals, the reimbursement environment, as well as the impact of physician preference within constantly evolving standards of care. He further specialized on the implications of healthcare reform on new product approval and health insurance reform. Mr. Spoor has also been an equity research analyst at J.P. Morgan from July 2007 through October 2008 and Credit Suisse from November 2005 through July 2007 covering the biotechnology and medical device industries. Prior to his career on Wall Street, Mr. Spoor worked in the pharmaceutical industry, spending 11 years with Amersham plc/GE Healthcare where he worked in seven countries in a variety of roles including setting up Good Manufacturing Practice (GMP) facilities, accountability for the nuclear cardiology portfolio, and most recently as the director of new product opportunities leading the positron emission tomography (PET) strategic plan. Mr. Spoor also sits on the Board of Directors of AtheroNova, Inc. (AHRO-OTC) and Protea Biosciences Group, Inc. (PRGB-OTC). Mr. Spoor's background in nuclear pharmacy, finance, and accounting, and as a healthcare research analyst, as well as his experience at both large and small healthcare companies provides him with a broad familiarity of the range of issues confronting a developing biotechnology company, which makes him a qualified member of MetaStat's Board of Directors.

David Epstein, Ph.D., Director on the Board of Directors and Chairman (Scientific Advisory Board for Therapeutics)

Biography provided on page 16.

Oscar L. Bronsther, M.D., F.A.C.S., Chief Executive Officer, Chief Medical Officer, and Director

Biography provided on page 13.

Warren C. Lau, President, Chief Financial Officer, and Director

Biography provided on page 14.

David N. Siegel, Director

Mr. Siegel was appointed to MetaStat's Board of Directors on February 27, 2012, effective as of April 7, 2012. Mr. Siegel was appointed president and CEO of Frontier Airlines, Inc. (part of Republic Airways Holdings Inc. [RJET-NASDAQ]) in January 2012. Previously, he was a managing director of Hyannis Port Capital, Inc. from June 2010 to December 2011. Mr. Siegel served as chairman and CEO of XOJET, Inc., a TPG Capital-funded private aviation company, from October 2008 until May 2010. Before joining XOJET, Mr. Siegel was chairman and CEO of Gategroup, AG, based in Zurich, from June 2004 to March 2009. Mr. Siegel was chairman and CEO of Gate Gourmet Group, Inc., an independent airline catering, hospitality, and logistics company. Prior to Gate Gourmet Group, Mr. Siegel served as president, chief executive, and a member of the Board of US Airways Group, Inc. (LCC-NYSE) and US Airways, Inc., the airline operating unit. Prior to joining US Airways, Mr. Siegel was chairman and CEO of Avis Rent A Car System, Inc. (part of Avis Budget Group, Inc. [CAR-NASDAQ]).

Patrick T. Mooney, M.D., Director

Dr. Mooney was appointed to MetaStat's Board of Directors on February 27, 2012, effective as of April 7, 2012. From September 2007 to September 2013, Dr. Mooney served as the CEO and chairman of the Board of Directors of Echo Therapeutics, Inc. (ECTE-NASDAQ). Dr. Mooney previously served as president, CEO, and director of Echo Therapeutics (a privately-held company prior to its merger with Sontra Medical Corporation) from September 2006 to September 2007. Prior to joining Echo Therapeutics, Dr. Mooney was president, CEO, and chairman of Aphton Corporation, a biopharmaceutical company, from January 2004 to November 2006. Aphton declared bankruptcy under Chapter 11 of the United States Bankruptcy Code. Dr. Mooney served as senior biotechnology analyst at Thomas Weisel Partners, LLC, a full-service merchant banking firm, and as senior biotechnology analyst at Janney Montgomery Scott, LLC, a full services investment banking firm. He graduated from the Jefferson Medical College of Thomas Jefferson University and trained as a surgical resident at Thomas Jefferson University Hospital. From June to September 2010, Dr. Mooney was a member of the Board of Directors of Quantrx Biomedical Corp. (QTXB-OTC). Dr. Mooney's medical education and experience as practicing clinician, as well as his industry-specific management experience, provides him with a broad and deep understanding of the science underlying MetaStat's business and competitors' efforts, which is a beneficial resource to the Company's Board of Directors.

Core Story

MetaStat, Inc. ("MetaStat" or "the Company") is a life sciences company seeking to develop and commercialize new diagnostic products and new therapeutics (focusing on alternatively spliced proteins) for systemic cancer metastasis—the spread of cancer from a primary tumor to other parts of the body via the bloodstream. The technologies center on MetaStat's patent-protected understanding of the role of the Mena protein in the body: (1) its re-expression in approximately 80% of all solid cancers of epithelial cell origin; (2) that the relative expression of Mena may determine the metastatic potential of an individual's cancer; and (3) that there are five alternatively spliced isoforms (variations) of the Mena protein that have been found to drive both tumor growth and tumor metastasis.

With this knowledge and over 15 years of research from a consortium of scientists across the U.S. and Europe, MetaStat has advanced several platform technologies designed to enable the early and reliable prediction and treatment of systemic tumor metastasis. The Company's lead initiatives include its MetaSite *Breast* and MenaCalc diagnostic products as well as its MenaBloc therapeutic program. Each of these programs are based on direct microscopic observation of the mechanisms and behaviors of metastatic cells in living, functioning human-derived tumors, which the Company believes is one of the competitive advantages for its product candidates.

MetaStat is currently targeting its diagnostic and therapeutic products toward four of the five largest solid cancer markets: breast, lung, prostate, and colorectal cancers.

The accompanying pages detail the core Mena protein and each of MetaStat's product candidates, followed by a discussion of the Company's market opportunities. Please note that the scientific information and results presented on the accompanying pages are summarized for easier reading. Additional information can be obtained from MetaStat or from any of the scientific publications listed in the Appendix on page 65.

Mena Protein Family

MetaStat's business centers on an intimate understanding of the underlying biology (the pathways, mechanics, and genetics) of systemic metastasis, which centers on the Mena protein family. The key discovery forming the foundation for MetaStat's product candidates is that the Mena protein isoforms (variants) are important regulators of the metastatic cascade. By continuing to research and leverage the Mena protein, MetaStat believes that its two active diagnostic programs—MetaSite *Breast* and MenaCalc—and its active therapeutic program MenaBloc could ultimately provide important clinical benefits, such as new and better diagnostic information, improved treatment decisions based on better knowledge, and improved economics of cancer care.

A number of genes have been identified that must be up- or down-regulated in invasive tumor cells in order to cause metastasis. One of the key upregulated genes encodes the protein Mena, which belongs to the vasodilator-stimulated phosphoprotein (VASP) family of proteins. VASP proteins have a role in cell migration (motility) by promoting the assembly of **actin** fiber networks. Actin fibers form a cellular "skeleton" in the cytoplasm of all cells, and are involved in motion of certain cell types.

In most individuals, the Mena protein is only present in developing embryos where the protein supports nervous system branching, and becomes scarce and undetectable in healthy adults. Mena helps embryonic nerve cells, or neurons, organize the formation of axons—nerve fibers that send messages from one neuron to other neurons in the nervous system. In this instance, Mena supports positive and healthy processes and is an essential component in nervous system formation. It is also an important element of cell migration. However, Mena can also appear in cancer cells, where it is much more harmful and supports cancer invasion and metastasis by enabling cancer cells to invade surrounding tissues and migrate toward and penetrate blood vessels.

Mena's Function

Mena functions as a regulatory protein of the actin network. The growth and elongation of actin fibers is controlled by a process that caps their ends. Mena interferes with (or "antagonizes") the actin capping, allowing the actin fibers to continue to lengthen, protruding the front edge of the cell forward. Greater details concerning the structure and function of the Mena protein are available at www.ncbi.nlm.nih.gov/pmc/articles/PMC3042857/.

Mena's Role in Cancer Metastasis

Mena influences a number of intracellular (inside the cell) signaling programs and serves as part of the pathway that regulates a primary tumor cell's transition to a metastatic cell. Mena has been shown to enhance a cancer cell's ability to invade surrounding tissues in a collaborative study by Dr. John S. Condeelis (biography on page 15) at the Albert Einstein College of Medicine and Dr. Frank Gertler (biography on page 15) at the David H. Koch Institute for Integrative Cancer Research at MIT (Source: Philippar et al., *Developmental Cell* 2008; 15:813-828).

Dr. Condeelis is chairman of MetaStat's Scientific Advisory Board for Diagnostics. He has pioneered microscope techniques for use in intravital imaging and currently serves as the scientific director of the Analytical Imaging Facility at the Albert Einstein College of Medicine as well as the director of the Tumor Microenvironment and Metastasis Program at the Albert Einstein Cancer Center. Dr. Gertler, a professor of biology at the Koch Institute, is on the Company's Scientific Advisory Boards for both Diagnostics and Therapeutics.

Mena modulates three elements of migratory behavior in metastatic cancer cells:

(1) regulation of actin fibers, which affect cancer cell movement and invasion;

(2) formation of **invadopodia**—specialized membrane **protrusions** that degrade the **extracellular matrix** and support invasion into surrounding tissues and blood vessels; and

(3) sensitivity to vascular **chemoattractant**, which induces the cancer cell to migrate toward blood vessels.

Collectively, these elements support invasion and metastasis. During the process of invasion, continuous degradation of the cellular matrix must occur along the advancing front of the migrating cells, in combination with active cell locomotion. The formation of invadopodia degrades the extracellular matrix, subsequently allowing the tumor cell to send out a well-organized protrusion that invades surrounding tissue and pulls the remainder of the cell behind it. This aspect is largely achieved through Mena's actin fiber regulation. All cells in the human body are able to move due to their actin cytoskeleton, which helps organize the shape of the cell while containing cellular organelles within the cell and in their proper position. However, in metastatic cells, Mena modulates the growth of the leading edge of the cell, allowing actin fibers to continuously grow in one direction and supporting the invasion of cancer cells into other areas of the body. The strength and direction of this invasion is modulated by Mena.

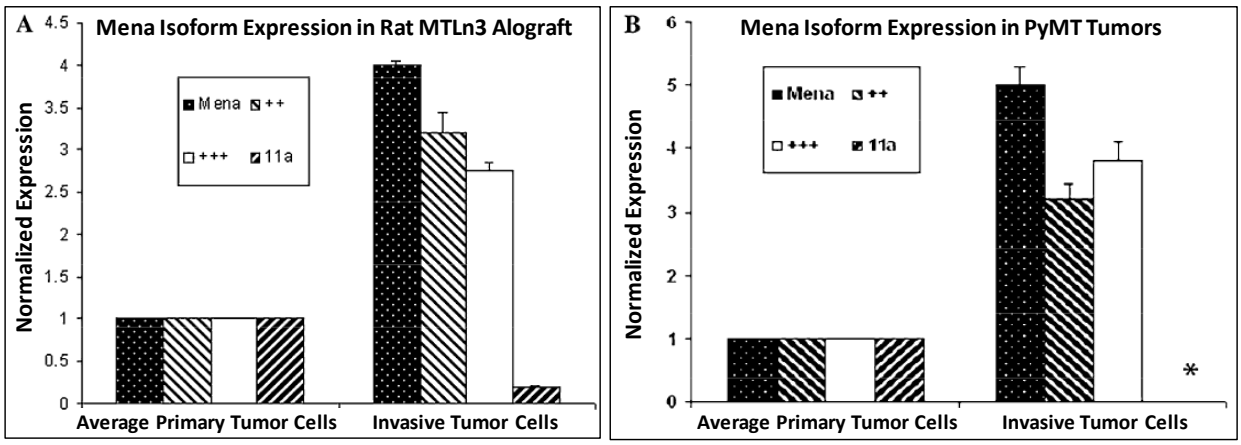
Mena is also responsible for migration of cancer cells toward the vascular system and into other areas of the body. Mena makes cancer cells more sensitive to epidermal growth factor (EGF), which attracts the cells toward blood vessels and is secreted by **perivascular** macrophages—one of the three cell types that constitute a MetaSite (described on pages 30-32). Thus, metastatic tumor cells are guided to the MetaSite where they are able to gain entry into the blood vessel and spread to other areas.

Mena Isoforms

Continued research by MetaStat's university collaborators revealed that, in cancer cells, Mena presents in different varieties (called "isoforms" or "splice variants")—sequences of Mena that have slight structural variations—resulting in more dangerous forms of the Mena protein (Source: *Developmental Cell* 2008; 15[6]:813-828). Small quantities of several Mena isoforms—Mena^{++}, $^{+++}$, and 11a—are found in average primary tumor cells (APTCs), as shown in Figure 8. However, there is a change in expression when APTCs become invasive. As illustrated in Figure 8, two isoforms of Mena are upregulated in invasive tumor cells ($^{++}$ and $^{+++}$), while one type (11a) is downregulated in three different primary mammary tumors, which is consistent with observations in human breast cancers and cancer cell lines (Source: *Clinical & Experimental Metastasis* 2009; 26:125-159). This pattern continues when invasive tumor cells begin to circulate and metastasize to other areas of the body. Mena expression results in a higher ratio of mobile carcinoma cells *in vivo*, and increases invasive potential both *in vitro* and *in vivo* (Source: Philippar et al., *Developmental Cell* 2008; 15:813-828).

Figure 8

MENA ISOFORM EXPRESSION IN AVERAGE PRIMARY TUMOR CELLS VERSUS INVASIVE TUMOR CELLS



Source: Clinical & Experimental Metastasis 2009; 26:153-159.

The Importance of the Mena^{INV} Isoform

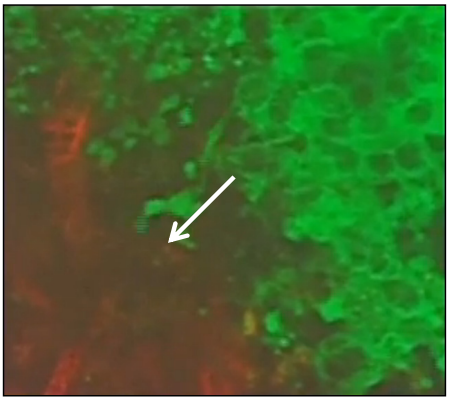
Animal models were used to evaluate the various effects of Mena isoforms. The most dangerous isoform of Mena is Mena^{+++}, which has been termed Mena^{INV} due to its potent effects at increasing invasion (*in vivo* and *in vitro*) and metastasis, including metastasis to the lung (Source: *Developmental Cell* 2008; 15[6]:813-828). In a study published in 2009, two different rodent models showed that levels of Mena and Mena^{INV} were three to four times higher in invasive cells versus primary tumor cells (Source: Phillipar et al., *Clinical Experimental Metastasis* 2009; 26[2]:153-159). In animals with Mena^{INV}, there were seven times as many circulating cancer cells in the bloodstream (Source: Roussos et al., *Breast Cancer Research* 2010; 12[6]:R101).

Mena^{INV} also promotes motility *in vivo* and localizes to the leading edge of motile tumor cells. It further increases the matrix degradation activity of tumor cells as a result of its role in the stabilization of invadopodia, providing opportunities for tumor cells to advance into new territories (Source: *Developmental Cell* 2008; 15[6]:813-828).

Mena^{INV} further increases tumor cells' sensitivity to chemoattractants. Mena^{INV} increases a cancer cell's motility responses stimulated by EGF, the chemical attractant that leads the metastatic cell to blood vessels (Source: *Clinical & Experimental Metastasis* 2009; 26:125-159). In one experiment, Mena^{INV} made metastatic cancer cells 25 times more sensitive to EGF (Source: Philippar et al., *Developmental Cell* 2008; 15[6]:813-828). An article in the *Journal of Cell Science* offers intravital images of Mena^{INV}-expressing tumor cells as they migrate toward a blood vessel (Source: Roussos et al., *Journal of Cell Science* 2011; 124:2120-2131). As well, Figure 9 illustrates one of these images as presented by Dr. Gertler at the 2009 Koch Institute Symposium.

Figure 9

INTRAVITAL IMAGE OF TUMOR CELLS WITH MENA^{INV} INTRAVASATING TOWARD BLOOD VESSELS



GREEN = Tumor Cells with Mena^{INV}

RED = Blood Vessels

* To view this Figure in color, please download an electronic version of this report from www.crystalra.com.

Source: MIT TechTV's "2009 Koch Institute Symposium - Frank Gertler."

Resistance to therapies that inhibit the EGF receptor (EGFR) (e.g., Tarceva® and Irressa®) is common in patients with metastatic cancer. Because cells expressing Mena or Mena^{INV} have an increased sensitivity to EGF, researchers have theorized that upregulation of Mena or Mena^{INV} may enable tumor cells to metastasize without the presence of a strong EGF signal, thus escaping the action of these cancer treatments (Source: *Developmental Cell* 2008; 15[6]:813-828).

Alternative Splicing Events (ASEs)

Data collected to date suggest that splicing regulates the activity of Mena during metastasis and a growing body of evidence has implicated alternative splicing events (ASEs) in the progression of cancer. As such, **exon**-specific antibodies to Mena^{INV} and 11a may be beneficial in identifying tumors with a high risk of invasion, potentially improving the accuracy of diagnosis and prognosis in cancer patients (Source: *Developmental Cell* 2008; 15[6]:813-828). To this end, MetaStat's MenaBloc therapeutic platform described on pages 44-46 focuses on Mena biology by targeting the ASEs that drive the epithelial to mesenchymal transition (EMT) process. EMT is a biologic process whereby an epithelial cell goes through multiple biochemical changes to become a mesenchymal cell. The resultant mesenchymal cell is characterized by greater invasiveness, resistance to apoptosis (programmed cell death), and an enhanced migratory capacity, meaning that these cells can migrate away from the epithelial layers they originated in. MetaStat believes that targeting the ASEs that start the EMT process with a therapeutic approach can delay tumor progression and decrease metastatic spread.

This approach was substantiated by an August 2011 article published in *PLoS Genetics*, called "An EMT–Driven Alternative Splicing Program Occurs in Human Breast Cancer and Modulates Cellular Phenotype." This research, referred to by MetaStat as the "Shapiro et. al. paper" after its primary author Irina M. Shapiro from the Koch Institute for Integrative Cancer Research at MIT, describes changes in the alternative splicing of genes involved in EMT progression, which have been found to occur *in vitro* and *in vivo* in breast cancer samples. These changes affect isoform expression of the genes that control the morphology and motility of epithelial and mesenchymal cells, enabling EMT processes. The authors found that EMT-dependent splicing changes occurred in breast cancer cell lines and in primary human breast cancer samples, and was likely regulated by splicing factors, including epithelial specific splicing factors (ESRPs) and the RBFOX, CELF, MBNL, and hnRNP splicing factor classes. A primary conclusion of the research is that alternative splicing has a major role in EMT and tumor progression, since EMT is an early step in the metastatic process.

In December 2013, MetaStat entered into exclusive worldwide patent and technology license agreements with MIT, the Koch Institute for Integrative Cancer Research, the Albert Einstein College of Medicine, and the Montefiore Medical Center. These agreements relate to the use of alternatively spliced mRNA and protein isoform markers in the diagnosis, prognosis, and treatment of metastatic, epithelial-based solid tumors. The findings from the Shapiro et. al. research, these MIT licenses, and MetaStat's intellectual property on the use of the Mena protein isoforms in the diagnosis, prognosis, and treatment of metastasis in epithelial cancers are at the heart of MetaStat's therapeutic development work.

Mena's Potential as a Biomarker for Metastatic Cancer

The increased expression of Mena^{INV} and decreased expression of 11a in both invasive and metastatic cells could provide opportunities for new diagnostic and prognostic biomarkers to detect the presence of metastatic cancer cells or predict metastatic disease. Recent research has demonstrated the potential of splice variants as cancer biomarkers. As well, analysis of the relative levels of the three isoforms—Mena^{INV}, Mena^{++}, Mena 11a—in tumor tissues could be used to create a new ratiometric prognostic marker to predict metastatic risk (Source: *Clinical & Experimental Metastasis* 2009; 26:125-159). As well, an 800-patient study performed to confirm the potential of these changes in expression as biomarkers has been published (Source: Agarwal et al., *Breast Cancer Research* 2012; 14:R124), and in 2014, an abstract presented at the Annual Meeting of the U.S. and Canadian Academy of Pathology further validated Mena's potential as a prognostic biomarker. This concept forms the foundation for MetaStat's MenaCalc diagnostic platform (described on pages 40-43).

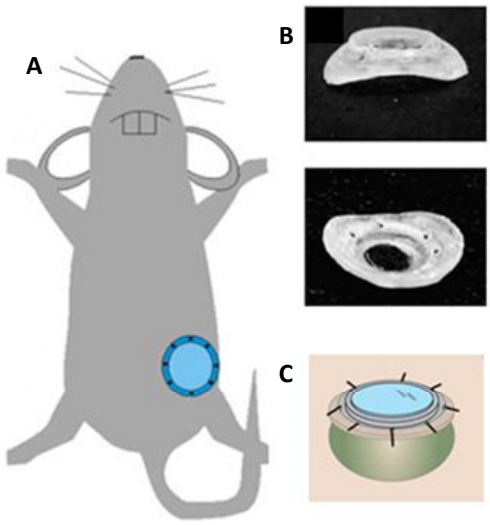
Mena Development Work is Enabled by Novel, New Life Science Research Tools

The processes behind metastasis are highly complex and can be challenging to study in the lab. While the invasion, intravasation, and dissemination aspects of metastatic cancer are known, researchers for a long time lacked an in-depth understanding of the mechanisms of invasion and migration and the molecular and behavioral phenotypes as well as how to detect these changes and how these changes affect the behavior of cancer cells. It was believed that an improved understanding of what molecules change to enable metastasis and how these changes affect the behavior of cancer cells could be beneficial for various aspects of metastatic cancer treatment, detection, and prognosis. In order to develop the technology platform that MetaStat possesses into viable products, the Company and numerous researchers from MIT, Albert Einstein College of Medicine of Yeshiva University, Cornell University, and Italy's IFO-Regina Elena Cancer Institute had to first create the tools for observing the behavior of metastatic cancer cells in tumors, which have since been licensed to MetaStat.

Intravital Imaging Window

A patented intravital imaging tool allows scientists to capture images from within a live animal. The imaging window has been used to observe and analyze the mechanisms of invasion for metastatic cancer. It was conceived by Dr. Condeelis and researchers at the Albert Einstein College of Medicine for use in combination with multiphoton microscopy in living mice to observe metastatic cell movement inside a tumor over a long period. The tool was first featured in an article published in *Nature Methods* in December 2008 (Source: *Nature Methods* 2008; 5(12):1019-21).

Figure 10
INTRAVITAL IMAGING WINDOW



Source: MetaStat, Inc.

Once the imaging window is implanted, a multiphoton microscope captures focused images of optically marked cells within the tumor. Multiphoton microscopy employs multiple light sources at various angles and offers the ability to obtain three-dimensional images as well as images from within live tissue. Researchers have used the intravital imaging window to help identify patterns of gene expression that relate to tumor cell behavior in vivo (within a living organism) and to observe cell behavior, particularly the elements that lead to invasion and metastasis, in tumors developed directly from the mammary epithelium in transgenic animals.

Artificial Blood Vessel

The teams who researched MetaStat's technology are believed to have been the first to understand how and why metastatic cells are attracted to blood vessels. With this knowledge, they invented and patented an artificial blood vessel—a microneedle filled with chemoattractants that could be used to attract, and thus isolate, a specific population of highly metastatic cancer cells from primary tumors within living animals. Microneedles containing gradients of either EGF or colony-stimulating factor 1 (CSF-1) were used to collect invasive cells from live primary tumors (Source: Wyckoff et al., *Cancer Research* 2007; 67(6):7022-7029). These chemoattractants mimicked **chemotactic** signals from blood vessels and surrounding tissue. To isolate a pure population of metastatic cells, a microneedle with chemoattractant is placed inside a tumor, left for a short period, and then withdrawn. MetaStat has received Notices of Allowance for its U.S. and European patent applications covering this artificial blood vessel technology. To the Company's knowledge, this is the first method to effectively isolate, collect, genetically profile, and determine chemotherapeutic resistance of a pure population of metastatic tumor cells. Historically, researchers have been unable to completely isolate metastatic tumor cells, and thus had challenges genetically profiling this population.

The artificial blood vessel technology is important to MetaStat's current product development, as it allowed researchers to narrow down the top 19 genes predicting systemic metastasis but not local recurrence in tumor cells. The gene signature includes upregulation of Mena and a number of other key regulators of actin polymerization and motility. Genes related to apoptosis and cell proliferation were downregulated in invasive cells (Sources: Wang et al., *Cancer Research* 2004; 64:8585-8594; Wang et al., *Cancer Research* 2007; 67[8]; Patsialou et al., *Cancer Research* 2012 72[8]). As such, conventional chemotherapeutic agents that are designed to interrupt cell division are not effective for these non-dividing cells.

According to MetaStat, this approach is believed to describe the actual causative gene signature of metastatic cells versus currently available technology, which claims to evaluate the risk of cancer recurrence by relying upon correlative mathematical algorithms developed by analyzing whole tumor tissue samples. To the Company's knowledge, none of the identified invasion signature genes are detectable by Genomic Health's Onco*type* DX® test.

MetaSite *Breast*

MetaStat's near-term product opportunity is MetaSite *Breast*, a diagnostic test designed to predict the likelihood of cancer spreading through the bloodstream to other organs in the body—also called systemic or **hematogenous** metastasis—in early stage breast cancer. This spread, and the resulting growth of breast cancer tumors in new organs, is responsible for up to 90% of breast cancer fatalities.

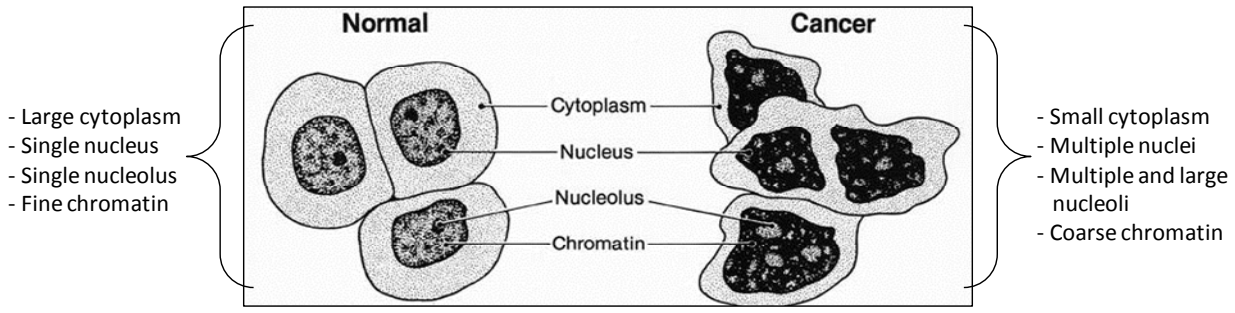
Current Methods for Diagnosing and Monitoring Metastatic Breast Cancer

While earlier detection and improved treatments have led to lower death rates over the past two decades, breast cancer was estimated to be linked to 40,000 deaths during 2013 in the U.S. alone (Source: American Cancer Society, Surveillance and Health Services Research, 2013). Physicians currently use various prognostic and predictive factors to diagnose and evaluate patients' risk of cancer progression and metastasis. Professional medical organizations, such as the College of American Pathologists (CAP), have identified a number of factors that are used to help guide physicians when evaluating breast cancer patients, including tumor grade (the degree to which the tumor cells under the microscope look different than normal breast tissue), tumor size, lymph node involvement (number of nodes), hormone status (estrogen/progesterone), HER2 status, and patient age.

For epithelial solid tumor cancers, such as breast cancer, a sample of tumor tissue is removed by core biopsy, lumpectomy, or mastectomy, and then evaluated by a pathologist under the microscope. In general, the less the breast tumor resembles normal breast tissue, the more aggressive the cancer may be. Figure 11 illustrates differences in normal cells versus cancer cells.

Figure 11

STRUCTURE OF NORMAL CELLS VERSUS CANCER CELLS



Source: National Cancer Institute.

Staining techniques may also be employed by pathologists to identify receptor sites (locations on the cell surface where molecules can interact with cellular components) for hormones that help some types of cancer cells grow, such as estrogen or HER2 (breast cancer). Approximately 20% of breast cancers produce excess HER2 due to a gene mutation, frequently resulting in a more aggressive cancer (Source: the Mayo Foundation for Medical Education and Research). In contrast to many competitive approaches, which are targeted to gene-specific breast cancers such as only HER2-positive or HER2-negative tumor types, MetaStat's MenaCalc diagnostic platform described on pages 40-43 is expected to be applicable to all breast tumor subtypes, which has the potential to separate MetaStat from its competition.

Currently, the American Joint Committee on Cancer (AJCC) guidelines for assessing tumor stage uses the TNM system, where "T" stands for tumor size and relationship to adjacent anatomic structures; "N" represents lymph node status; and "M" signifies whether or not distant spread is detected using biopsy or imaging techniques.

Room for Enhanced Prognostic Tools

At present, physicians rely primarily on tumor grade and stage to estimate the likelihood of metastasis, but are well aware of the shortcomings in this approach. Given their experience working with cancer patients, some physicians report that this method results in improper classification for some patients. Patients who are improperly classified as "high risk" may be exposed to months of aggressive chemotherapy and **radiation** treatments that cause severe side effects (as summarized in Figure 12) and can dramatically impact quality of life. Similarly, patients incorrectly classified as "low risk" may not receive timely and necessary treatment. As the efficacy of treatment varies significantly across cancer populations, the cost of the therapy and the physical and mental burdens associated with treatment may not always be justified.

Figure 12

A SELECTION OF POTENTIAL SIDE EFFECTS OF CHEMOTHERAPY AND RADIATION THERAPY

Chemotherapy Side Effects			Radiation Therapy Side Effects
▪ anemia	▪ infection	▪ neuropathy	▪ skin reactions
▪ bone loss/osteoporosis	▪ memory loss	▪ taste and smell changes	▪ armpit discomfort
▪ diarrhea	▪ menopausal symptoms	▪ vaginal dryness	▪ chest pain
▪ fatigue	▪ mouth and throat sores	▪ vision/eye problems	▪ fatigue
▪ fertility issues	▪ nail changes	▪ vomiting	▪ heart problems
▪ hair changes	▪ nausea	▪ weight changes	▪ lowered white blood cell counts
▪ heart problems			▪ lung problems

Source: Breastcancer.org (http://www.breastcancer.org).

One prognostic tool currently available for breast cancer is Genomic Health's Onco*type* DX® Breast Cancer Assay, which evaluates 21 genes to predict the likelihood of a patient benefitting from chemotherapy or suffering from cancer recurrence in early stage breast cancer. Onco*type* DX® assigns a numerical value to the risk of recurrence and the likelihood that the patient will benefit from chemotherapy in addition to hormonal therapy. Some physicians argue that Onco*type* DX® does not add to data collected by routine immunohistochemistry—a laboratory test performed on tumor tissue to detect the amount of a specific genetic protein in the cancer calls—and that the confidence intervals for predicting outcome for an individual patient are broad (Source: http://www.onclive.com, May 4, 2011). Regardless, since its launch in 2004, Onco*type* DX® has been endorsed by both the American Society of Clinical Oncology (ASCO) and the National Comprehensive Caner Network (NCCN). The test is reimbursed by many insurance companies, a contributing factor in its adoption. Based on clinical results to date, MetaStat believes its MetaSite *Breast* test could provide valuable added information for pathologists, physicians, and patients beyond what Onco*type* DX® provides due to their very different mechanisms of action. Results from a comparative study of the two tests are provided on page 35.

MetaStat's Diagnostic Assays are Fundamentally Different

MetaStat's technology focuses on identifying markers that can predict the risk of cancer advancing based on the mechanisms involved in cancer development and metastasis. Historically, cancer cells have entered blood vessels via unknown means. The research supporting the Company's technology has sought to identify structural and behavioral mechanisms that allow cancer cells to move and to understand how this information can be used in prognosis. To MetaStat's knowledge, its technology is the only technique at present that focuses on mechanistic markers.

Specifically, unlike MetaSite *Breast*, the Onco*type* DX® algorithm is not mechanism based. Rather, it is a mathematical algorithm that relies heavily on proliferation markers and hormone receptor and HER2 status. In contrast, MetaSite *Breast* is mechanism based, which the Company believes could better predict a breast cancer patient's prognosis.

Figure 13 shows a comparison of MetaSite *Breast* and the Company's other diagnostic platform, MenaCalc, to existing gene-based tests.

Figure 13

METASITE *BREAST* AND MENACALC ARE MECHANISTICALLY DIFFERENT FROM EXISTING GENE-BASED TESTS

MetaSite *Breast* and MenaCalc assess metastatic risk by directly measuring the activity of the metastatic process at the cellular level and protein level, respectively.

	MetaSite *Breast*	MenaCalc	Oncotype DX	MammaPrint	IHC4
Manufacturer	**MetaStat**	**MetaStat**	Genomic Health	Agendia	Genoptix
Indication	**Breast**	**Breast and potentially prostate, colorectal, and lung**	Breast, colon, prostate	Breast	Breast
Hormone Receptor Status Independent?		√		√	
Tissue Requirement	**Excisional biopsy**	**Needle biopsy or excisional biopsy**	Excisional Biopsy	Needle biopsy or excisional biopsy	Excisional biopsy
Prognostic for Distant Metastasis	√	√	√	√	√
Direct Measure of Metastatic Activity	√	√	X	X	X
Assess Potential for Proliferation	X	X	√	√	√

Source: MetaStat's data from product websites of Oncotype DX, MammaPrint, and IHC4 by Aqua, and Medscape News, January 2013.

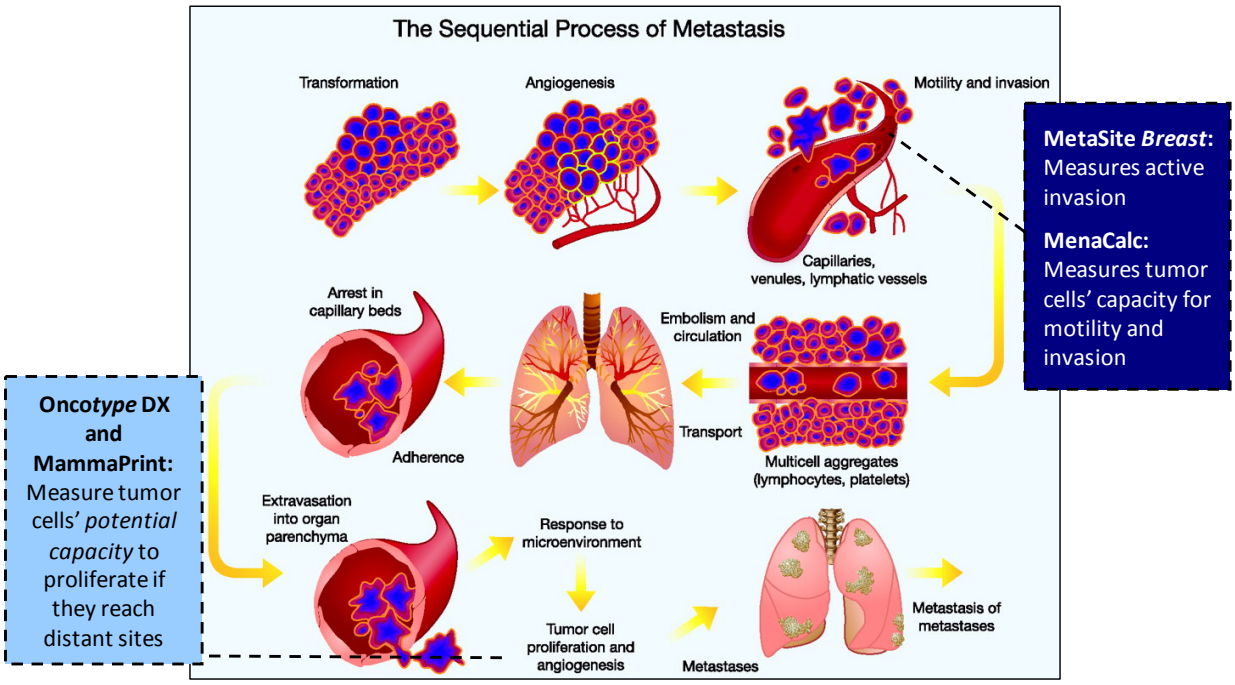
A major difference in these tests listed in Figure 13 above is that existing gene-based tests measure tumor cells' potential to proliferate while MetaStat's diagnostic focus is on creating tests to directly measure metastatic activity, i.e., whether the tumor cells will or are actually entering the vasculature. As shown in Figure 14 (page 30), these entail two different stages of the metastatic process.

By measuring the metastatic potential of a distinct tumor cell population unlike a whole genome assay such as Oncotype DX®, MammaPrint®, or IHC4, MetaStat believes that its MetaSites and Mena^{INV}-based approaches offer several novel capabilities in terms of measuring the following tumor characteristics:

- the proprietary invasive signature—a set of genes where their expression is altered in invasive tumor cells;
- the potential for motility and cell dissemination;
- the potential for an epithelial to mesenchymal transition (EMT);
- the modulation of α5β1 integrin function and enhanced ECM bidirectional signaling;
- the chemotactic and invasive functions driven at benign EGF concentrations;
- the resistance to EGFR targeted therapies; and
- the resistance to chemotherapy and radiation.

One of the core components of the Company's approach is studying the distinct population of cells that express the Mena^{INV} isoform and are in the act of hematogenous dissemination and metastasis, yet these cells are not believed to be accounted for in whole genome assay-based diagnostics (Source: MetaStat's Investor Presentation, April 2013).

Figure 14
PROLIFERATION VERSUS METASTATIC ACTIVITY



Source: MetaStat's data from Talmadge et al. "AACR Centennial Series: The Biology of Cancer Metastasis: Historical Perspective." J Cancer Res 2010;70:5649-5669.

THE METASITE *BREAST* TEST

MetaSite *Breast* is a clinical laboratory assay (or test) to predict the likelihood of an early-stage breast cancer patient's tumor spreading to distant body parts. MetaSite *Breast* employs conventional immunostaining techniques to highlight unique three-cell structures in a tumor tissue sample. The three-cell structure (termed a "MetaSite") is composed of a macrophage cell, a carcinoma cell expressing the Mena protein, and an endothelial cell. This structure was identified by scientists from MIT, the Albert Einstein College of Medicine, and Weill Cornell Medical College, who reasoned that the density of MetaSites was correlated to the probability of distant tumor metastases. Research is showing that the three-celled MetaSite has a crucial role in allowing metastatic cells to enter into the bloodstream and spread through the blood to other organs in the body.

MetaSites

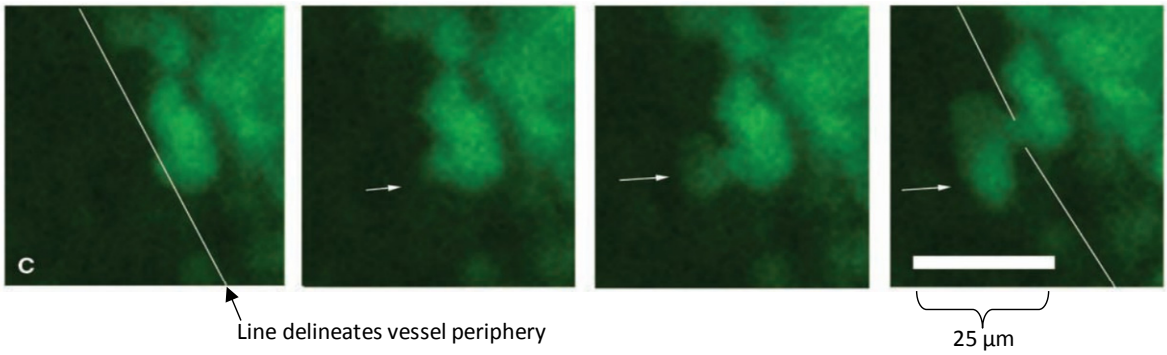
It was long thought that macrophages congregate in tumors to attempt to perform some of the immune system functioning to get rid of foreign cancer cells. However, researchers have learned that cancer has co-opted this process and turned the macrophages into helper cells that help facilitate metastatic movement of cells (Source: *Cancer Research* 2004; 64:7022-7029). Researchers behind MetaStat's licensed technology, including Dr. Condeelis, discovered the biochemical signaling that occurs between macrophages and labeled it the "paracrine loop." Macrophage provides EGF to the tumor cell, which in turn provides colony-stimulating factor (CSF) to the macrophage (Source: MIT TechTV's "2009 Koch Institute Symposium - Frank Gertler"). The findings were published in a 2004 *Cancer Research* article entitled, "A Paracrine Loop between Tumor Cells and Macrophages Is Required for Tumor Cell Migration in Mammary Tumors."

The notion that macrophages contribute to, rather than combat, the metastatic process has been further confirmed in preclinical research. While observing invasive cells through the intravital imaging window (described on page 25), researchers behind MetaStat's licensed technology discovered the essential components enabling metastatic cells to enter the bloodstream and metastasize to other areas of the body. Using the intravital imaging window, researchers observed and recorded a metastatic cell and the macrophage being attracted toward one another and linking together. As viewed from a blood vessel within a mouse, researchers watched as a metastatic cell penetrated the wall of the blood vessel and was swept away by the bloodstream.

Figure 15 illustrates an invasive tumor cell entering the bloodstream by penetrating a nearby blood vessel.

Figure 15

A TUMOR CELL IS IMAGED INTRAVASATING INTO A BLOOD VESSEL



* To view this Figure in color, please download an electronic version of this report from www.crystalra.com.

Source: Cancer Research 2007; 67:8.

In order to enter a blood vessel (intravasate), three types of cells must present together in the same micro-anatomical site, as shown in Figure 16:

(1) an endothelial cell (a type of cell that lines blood vessels);

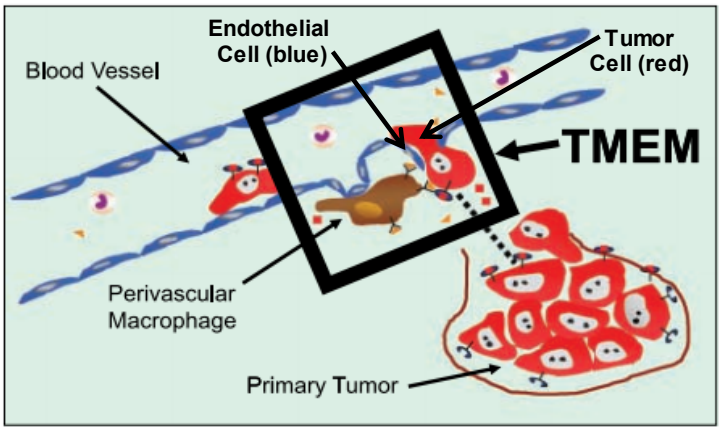
(2) a perivascular macrophage (a type of immune cell that guides tumor cells to blood vessels); and

(3) a tumor cell that produces the Mena protein.

Collectively, this entity is called the Tumor Microenvironment of Metastasis (TMEM) or "MetaSite."

Figure 16

TUMOR MACROENVIRONMENT OF METASTASIS (TMEM) OR "METASITE"



Source: Clinical Cancer Research 2009; 15(7).

Using the MetaSites to Predict Tumor Metastasis

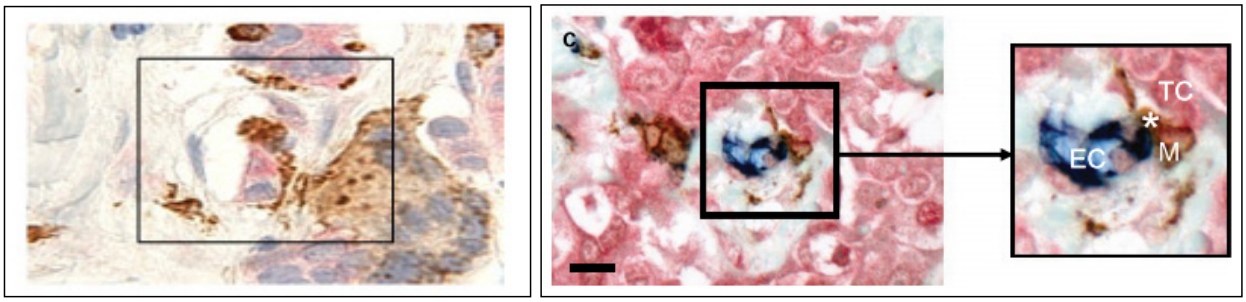
With MetaSite *Breast*, pathologists can clearly see and count the amount of MetaSites present in a sample of breast tumor tissue. Samples with a high density of MetaSites have been shown thus far to correlate to a higher risk of metastasis. Moreover, this cancer marker is believed to be among the first to reliably predict metastatic outcome in a case-controlled study, and may have potential to dramatically change the approach for breast cancer treatment (Source: Albert Einstein College of Medicine press release, March 24, 2009).

The Company's MetaSite *Breast* assay is designed to provide physicians and patients with critical information on metastatic potential that is highly specific to the individual tumor. Test results would provide both a quantitative score based on the number of MetaSites as well as an accompanying interpretation of what the score indicates in terms of the risk of developing metastasis and classification of the patient into a low, medium, or high-risk group.

Importantly, the MetaSite *Breast* test does not require any special equipment, techniques, or procedures, and as such, is designed to be seamlessly incorporated into the standard procedures for analyzing tumor stage and grade. It employs conventional immunostaining techniques that use antibodies to the three cell types found in the MetaSite in order to allow the pathologist to visualize the three-cell MetaSite structures in the tumor tissue samples (as shown in Figure 17).

Figure 17

METASITES VISUALIZED AFTER METASITE *BREAST'S* TRIPLE IMMUNOHISTOCHEMICAL STAIN



Red = Tumor Cell (TC) Blue = Endothelial Cell (EC) Brown = Macrophage (M)

* To view this Figure in color, please download an electronic version of this report from www.crystalra.com.

Sources: MetaStat, Inc. and Clinical Cancer Research 2009; 15(7).

Clinical Studies

MetaSite *Breast* has already been studied in several human trials in order to demonstrate its efficacy at identifying the density of MetaSites as a prognostic marker of the patient's risk of developing metastatic disease. In addition, the test has been favorably compared to a current market leader, Genomic Health's Onco*type* DX® Breast Cancer Assay (results of which are presented on page 35), and has recently completed a large-population validation study (described on page 36).

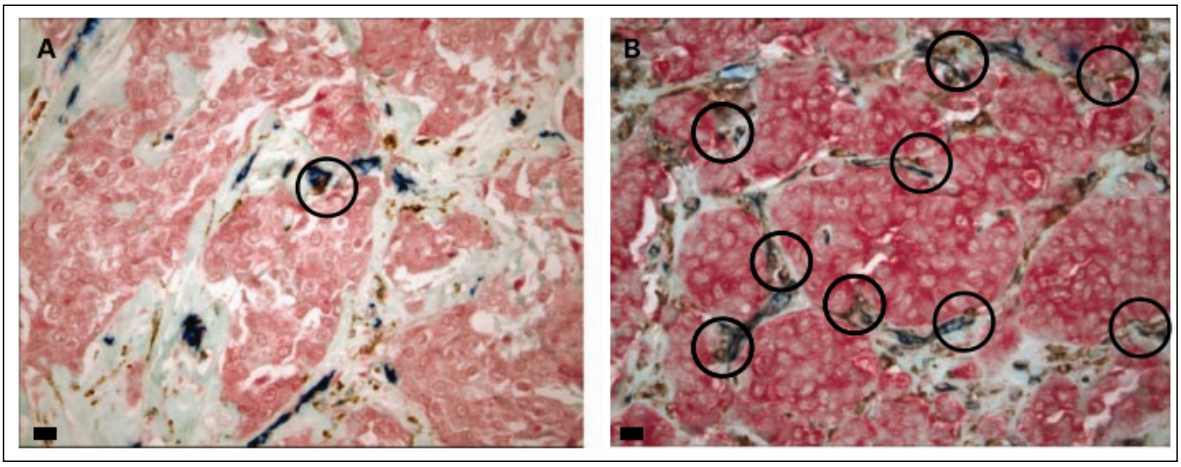
Finding a Correlation Between TMEM Density (MetaSites) and Metastatic Disease

In this study, researchers used the MetaSite *Breast* test to compare 30 paraffin-embedded samples from primary breast cancer patients who eventually developed metastatic breast cancer and 30 samples from breast cancer patients without metastatic disease. The case-control study was designed to compare breast cancer patients who suffered metastatic disease with patients who did not develop metastases but were otherwise similar based on currently used prognostic criteria (the control group). All patients were followed for a minimum of five years. Without knowing which tissue sample belonged to which group, pathologists applied the triple immunohistochemical stain to each sample to identify MetaSites (or TMEMs). Each patient's MetaSites were counted, and increased density of TMEMs correlated significantly with metastasis.

An example of two patients' tissue samples—one with metastasis and the other without—is shown in Figure 18. The patients had a similar histological tumor grade and both samples were stained for TMEM. The non-metastatic patient had low TMEM density (A, left side) while the patient who later developed metastases showed significantly more MetaSites (B, right side). The density of any of three MetaSite components individually—a carcinoma cell, macrophage, or endothelial cell—was not sufficient to predict the clinical outcome.

Figure 18

COMPARISON OF TMEM COUNTS* IN A METASTATIC AND A NON-METASTATIC TUMOR WITH SIMILAR HISTOLOGICAL GRADES



** Each circle depicts one TMEM (or three-cell "MetaSite") after triple immunostaining. Original magnification, 400; bar, 20 μm; circles, 60 μm in diameter.*

Source: Clinical Cancer Research 2009;15(7).

As a result, researchers concluded that TMEM density was significantly correlated with hematogenous metastases, and may be useful as a prognostic marker for patients with breast cancer. The number of MetaSites varied widely from 12 (low risk) to over 240 (high risk). Data from the study showed that the patient group that eventually developed metastasis had higher MetaSite density (median 105) versus the control group (median 50), as shown in Figure 19 (page 34).

For every 10 TMEMs, the odds of systemic metastasis nearly doubled (with a 95% confidence interval). Importantly, the ability of TMEM to predict distant metastasis was independent of conventional prognostic indicators, including tumor size, grade, lymph node metastasis, lymphovascular invasion, or hormone receptor status. In 2009, the study was published in *Clinical Cancer Research* and entitled "Tumor Microenvironment of Metastasis in Human Breast Carcinoma: A Potential Prognostic Marker Linked to Hematogenous Dissemination." Three of the study's authors now reside on MetaStat's Scientific Advisory Board for Diagnostics, including Dr. Condeelis (chairman), Dr. Rohan, and Dr. Gertler. As well, Dr. Joan Jones is a member of the Company's Clinical Advisory Board.

Figure 19

TMEM DENSITY IN THE NON-METASTATIC AND METASTATIC PATIENT GROUPS

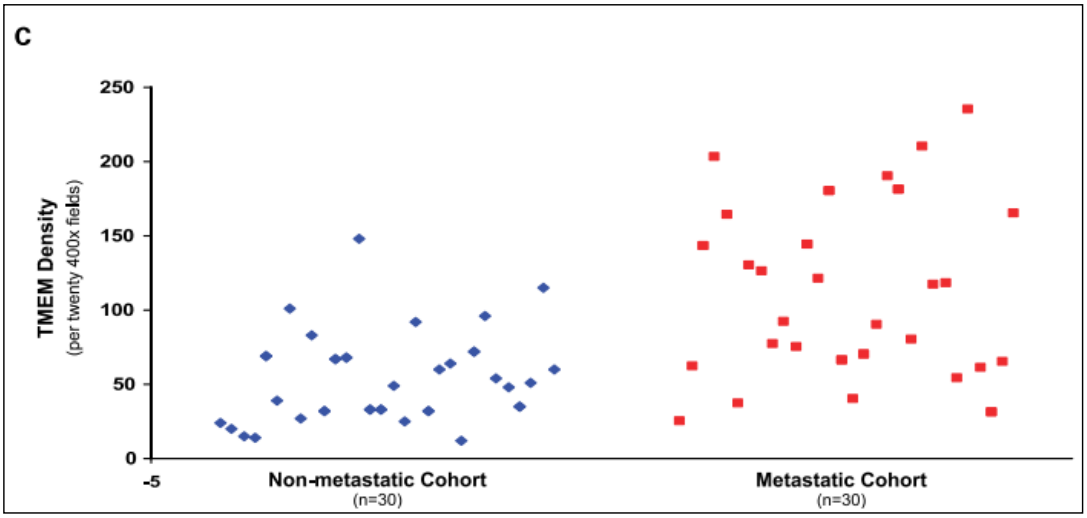


Table 2. Case-control study results

(A) TMEM density

	Metastatic cohort (n = 30)	Nonmetastatic cohort (n = 30)	P
Median (5th percentile, 95th percentile)	105 (28.3, 221)	50 (13.1, 130)	0.00006

(B) Increase in risk of metastasis per 10-unit increase in TMEM

Adjusted for	Odds ratio (95% confidence interval)
(Unadjusted)	1.9 (1.1-3.4)
Age at diagnosis	1.9 (1.1-3.4)
Tumor grade	1.9 (1.1-3.4)
Tumor size	1.9 (1.1-3.3)
Lymphovascular invasion	1.5 (0.95-2.3)
Lymph node metastasis	1.9 (1.0-3.6)
Estrogen receptor status	2.0 (1.1-3.7)
Progesterone receptor status	1.9 (1.0-3.6)
HER-2/*neu* status	2.2 (1.1-4.7)

NOTE: TMEM density was significantly higher in the group of patients who developed distant metastasis compared with those with localized breast cancer (A). Additionally, for every 10-unit increase in TMEM, the odds of metastasis almost doubled (B). This estimate was robust to adjustment (separately) for the commonly used prognostic criteria listed in the table, including tumor grade, emphasizing that TMEM is not a surrogate for grade and may be a useful new independent prognostic factor.

Source: Clinical Cancer Research 2009;15(7).

Clinical Justification to Complement Tests like Oncotype DX® with MetaSite Breast

MetaStat has evaluated the MetaSite *Breast* test against Genomic Health's Onco*type* DX® Breast Cancer Assay. Importantly, after a review of information and data available on the Genomic Health, Inc. website, MetaStat estimates that the gene signature of Onco*type* DX® is approximately 60% related to cell proliferation, 38% response to hormones, and 2% to motility and degradation of the extracellular matrix, which varies greatly from the MetaSite *Breast* test. As a result, MetaStat anticipates that its test's TMEM count (or "MetaStasis Score") may not correlate with the Onco*type* DX® results, thus it could be used to provide new information to physicians that both complements and potentially competes against Genomic Health's recurrence score (RS).

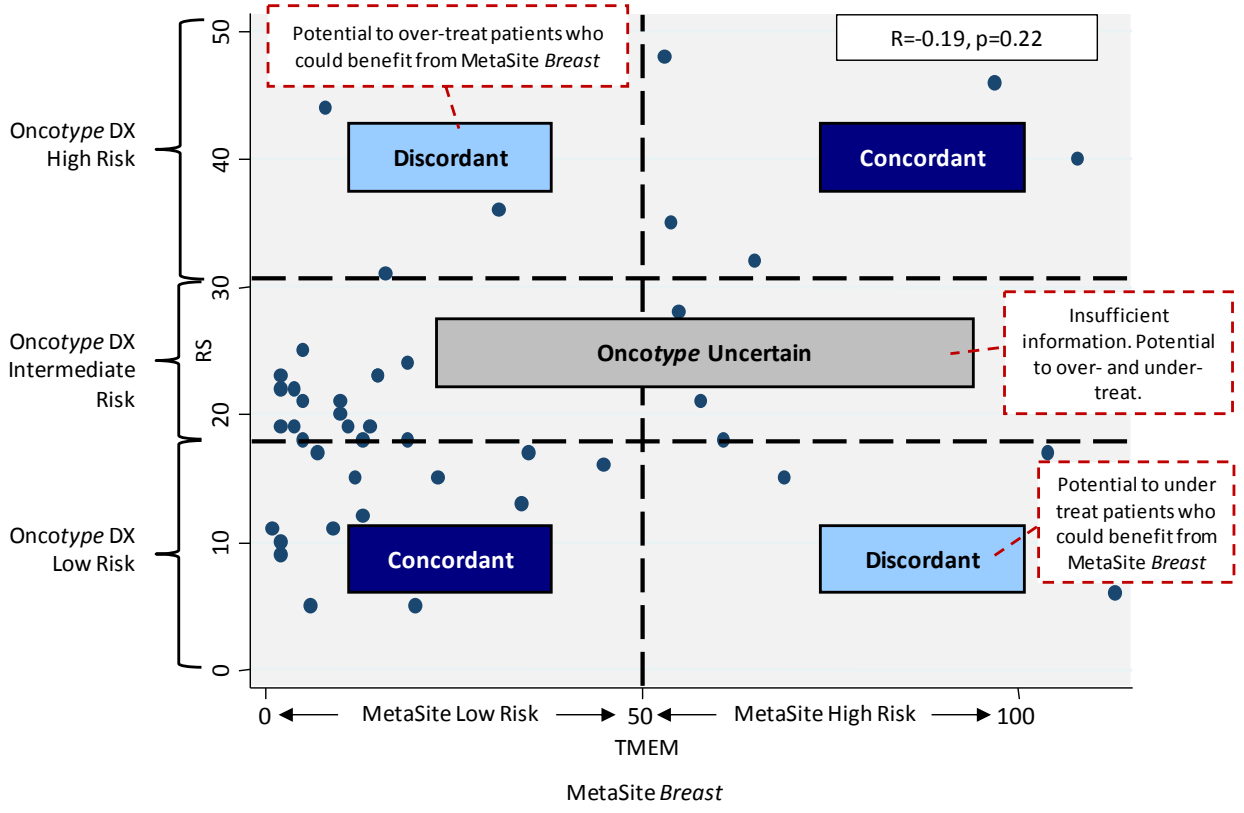
To illustrate this variation, in an unpublished study of 44 breast cancer patients, the Onco*type* DX® Recurrence Score was compared to the MetaSite count. Data from the study revealed a correlation coefficient of 0.19, indicating that the two tests are largely independent. MetaStat believes that, if repeated in a larger study, the data could indicate that MetaSite *Breast* supplies valuable information beyond the Onco*type* DX® test and holds the potential to become an important element in the clinical care and stratification of breast cancer patients.

In both trials (MetaSite *Breast* independently and in comparison to Onco*type* DX®), MetaStat divided participants into three cohorts based on the MetaSite count: low, medium, and high risk. The Company found that patient samples classified as high-risk were 22 times more likely to experience metastasis than the low-risk group. Onco*type* DX® also separates patients into three risk-level groups; however, with a group average of a 31% risk of recurrence, the high-risk group is believed to be only 4.5 times more likely to recur than the low-risk group (7%).

Accordingly, MetaStat believes that its MetaSite *Breast* test can help stratify the approximately 40% of patients who were identified by the Onco*type* DX® test to be intermediate risk, noted as "Onco*type* Uncertain" in Figure 20.

Figure 20

COMPARISON OF METASITE *BREAST* AND ONCO*TYPE* ® DX TESTS



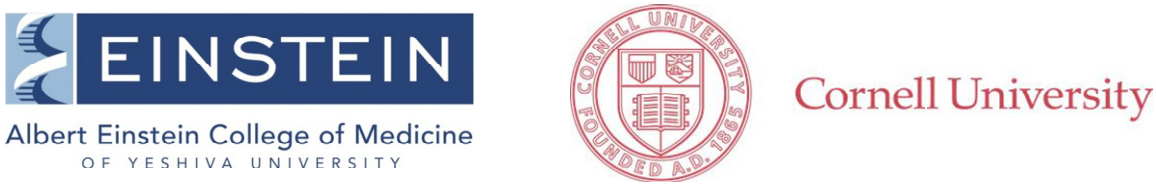
Source: MetaStat, Inc.

Large-Population Validation Study in Breast Cancer Patients

In January 2013, MetaStat completed a study of MetaSite *Breast* in nearly 500 patients. The trial was performed in conjunction with two sponsored research partners: the Albert Einstein College of Medicine and Weill Cornell Medical College. The trial used tumor tissue samples that had previously been collected from 481 female breast cancer patients, and each patient's medical history for a minimum of a five-year period after the tumor tissue had been excised. This type of trial structure is called a retrospective study, as it allows cancer researchers to access and use archived tumor tissues and medical records without personal identifying information attached to the patient histories and follow-ups. This negated the need to obtain informed consent, and is commonly employed by major cancer research centers. When a tumor is excised at a major cancer research center, sections of the tumor are often preserved and archived to support ongoing and future cancer research. The tissue samples for MetaStat's study were provided by Kaiser Permanente, a nonprofit health plan provider.

Figure 21

SPONSORED RESEARCH PARTNERS FOR THE LARGE-POPULATION VALIDATION STUDY



Source: MetaStat, Inc.

The trial had two main objectives:

(1) to further study the relationship between TMEM count at initial diagnosis of invasive **ductal carcinoma** (the most common type of breast cancer) and a patient's risk of systemic metastasis; and

(2) to identify a cut-point for stratifying patients into low-, medium-, and high-risk groups (based on TMEM count) based on the likelihood of developing systemic metastasis and to determine the cut-point's sensitivity and specificity.

MetaStat used samples from 250 metastatic breast cancer patients and 250 non-metastatic individuals and paired them as closely as possible based on tumor size, grade, lymph node involvement, and hormone receptor status. MetaStat then performed the MetaSite *Breast* test on the tissues, and determined the TMEM count and density. The MetaSite density could then be compared to the known outcome from the medical records—the equivalent of five-year follow-up data acquired in significantly less time and lower cost than performing a prospective human trial. This technique has been utilized for the validation of a number of products on the market today, including Onco*type* DX®.

Initial results of this study were announced in October 2013, and have been submitted for publication in a peer-reviewed scientific journal. They were also presented in a poster session at the San Antonio Breast Cancer Symposium on December 10-14, 2013. Results showed the value of the MetaSite *Breast* test in the prediction of metastatic disease by confirming that the Metastasis Score (TMEM count) returned by the test was associated with a statistically significant increase in the risk of distant metastasis in patients with ER+/HER2-negative breast cancer. Importantly, the prognostic information provided from the MetaSite *Breast* test was stated to be independent of and thus complementary to clinicopathologic variables, such as lymph node spread, tumor size, and an IHC-4 score (generated by a currently marketed diagnostic assay) (Source: *Tumor Microenvironment of Metastasis and Risk of Distant Metastasis of Breast Cancer* poster, 2013). This relationship between TMEM count and distant metastasis was identified in a predefined subgroup of women with ER+/HER2-negative breast cancer, which represents over 60% of all breast tumors.

Competitive Advantages for Marketing and Commercializing the MetaSite *Breast* Test

MetaStat's products are being designed to help improve the quality of treatment decisions as well as to improve the economics of cancer care. The MetaSite *Breast* test differs from existing tests by assessing metastatic risk rather than proliferation risk (as a result of measuring the activity of the metastatic process). The Company is developing its diagnostic based on tested theories that this type of measurement can be a better predictor of metastasis, and has accordingly found in clinical studies that MetaSite *Breast* appears to enable a higher degree of precision in stratifying risk than competitive products. Thus, it may have a considerable competitive advantage in improving the quality of treatment decisions by more accurately classifying patients as high risk versus low risk for systemic metastasis—ideally combatting issues of overtreatment for some patients and not enough treatment for others.

Figure 22

POTENTIAL COMPETITIVE ADVANTAGES OF METASITE *BREAST*

- The number of MetaSites correlates with metastatic risk (every 10 point increase in TMEM score doubles the risk of metastasis).
- It directly examines the active metastatic process as it occurs.
- It readily fits into the current diagnostic paradigm and does not require additional surgical procedures.
- It does not require FDA approval (CLIA GLP certification).
- It is differentiated from existing tests.
  - It assesses metastatic risk rather than proliferation risk by measuring the activity of the metastatic process.
  - When compared to existing tests, MetaSite *Breast* enables a higher degree of precision in stratifying risk.
  - Low cost of goods and broad IP may help prevent the emergence of direct competitors and substitute tests.

Source: MetaStat, Inc.

Low List Price

MetaStat anticipates that it can price its breast cancer diagnostics very competitively in the market. The Company has calculated its costs, including processing tumor tissue samples, the wholesale price of reagents, the machinery involved in the staining of MetaSites, technician and administrative time, professional fees for pathologists, sales and marketing expenses, and profit margin, and believes that its list price for its breast cancer diagnostics will likely be $2,595. This is significantly less than existing diagnostic products for breast cancer, including Onco*type* DX®, which has a list price of roughly $4,290 (Source: MetaStat's Form 10-K filed with the SEC on May 28, 2013).

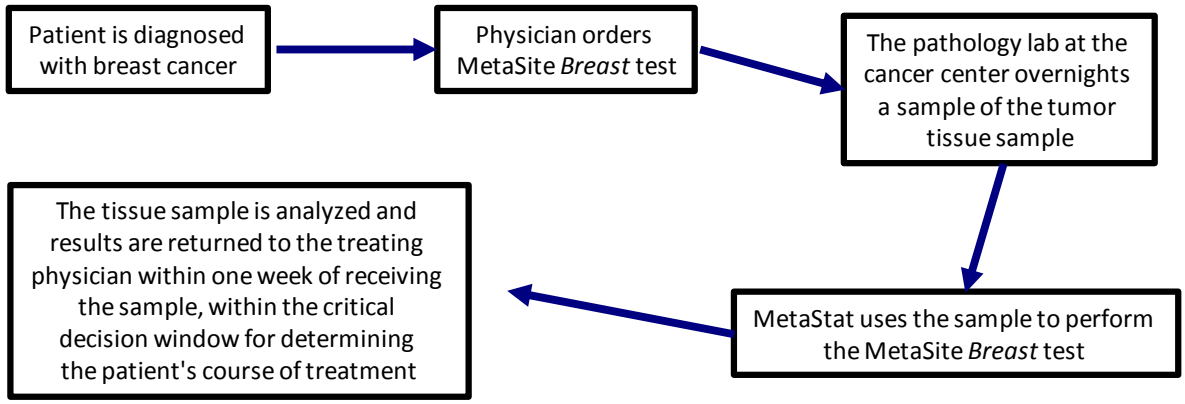
MetaStat may be able to further reduce cancer treatment costs by providing physicians/patients with critical information before the course of action for treatment is finalized. When metastasis risks are not properly assessed, patients may undergo toxic and costly chemotherapy regimens that were unnecessary or, conversely, may receive insufficient treatment, allowing the cancer to progress. An eight-week regimen of chemotherapy can cost as much as $30,000 (Source: Livestrong.com, "The Average Cost for Cancer Chemotherapy Treatment," March 31, 2011). As well, if a high-risk breast cancer patient is misclassified as low risk, the individual may miss the opportunity to treat the cancer in the early stages, when it is most treatable, potentially necessitating future treatment that may be more costly and may not yield the most beneficial results if the cancer spreads.

Compatible with the Current Diagnostic Paradigm

Figure 23 summarizes how MetaSite *Breast* can be incorporated into the traditional treatment decision process without requiring physicians to perform additional procedures on the patient or purchase any new equipment.

Figure 23

METASITE *BREAST* INCORPORATED INTO THE CONVENTIONAL TREATMENT DECISION PROCESS



Source: MetaStat, Inc.

An advantage of MetaSite *Breast* is that it employs widely available immunohistochemical tissue staining techniques and allows operators to view different cell types on a single slide using different dye colors. Initially, the Company expects one Leica BOND-III immunohistochemical staining cabinet (shown in Figure 24) to be sufficient to process MetaSite *Breast* tests. As such, MetaStat believes that this approach may be more cost effective than marketed products that use genomic-based techniques.

Figure 24

INITIAL LABORATORY EQUIPMENT AND PERSONNEL NECESSARY FOR METASITE *BREAST* TEST ANALYSIS

Initially, MetaStat can support processing of the the MetaSite Breast test with one Leica BOND-III immunohistochemical staining cabinet.

Sources: Leica Microsystems Inc. and MetaStat, Inc.

Additionally, based on current regulations, MetaStat does not expect to have to pursue FDA approval for MetaSite *Breast*. Rather, the Company must establish a clinical reference laboratory for administering the assay that meets Clinical Laboratory Improvement Amendments (CLIA) and Good Laboratory Practice (GLP) standards.

INITIAL MARKETING PROGRAM

MetaStat aims to commence marketing at major cancer centers in eight target cities during 2015. In each of these cities, the Company has enlisted the support of key opinion leaders at major academic cancer centers. The Company's management team and Scientific Advisory Boards have pre-existing relationships with several key cancer centers, which are likely to represent early targets for the initial marketing program.

In each city, the Company aims to establish relationships at a minimum of one academic medical center and potentially at community-based cancer centers as well. MetaStat anticipates a national marketing effort approximately three to four years after generating sales in its initial target cities—enabling a sales force expansion driven by the early revenues.

MetaStat aims to grow these relationships and nurture new relationships with other cancer centers to spread awareness of its novel diagnostics for breast cancer. To date, the Company has showcased its technologies at major cancer centers, such as the M.D. Anderson Cancer Center and Montefiore Medical Center. Based on discussions with heads of the breast medical oncology departments at these organizations, the Company has reported that its tests could expand the cancer diagnostics market while helping payors lower costs through customized cancer therapy.

MenaCalc Diagnostic Platform

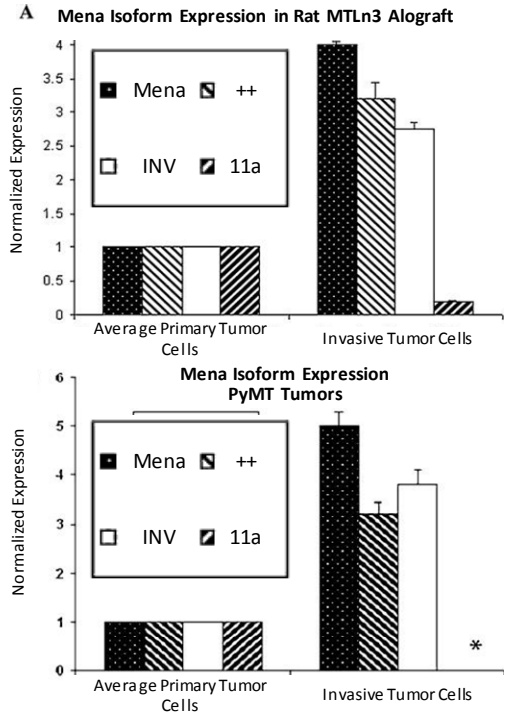
The MenaCalc technology platform is based on research conducted by academic institutions that have found that patients' potential for cancer metastasis could likely be determined by measuring their relative amounts of Mena isoforms, offering clinicians a more complete picture upon which to base a patient's treatment strategy. Further, a patient's Mena isoform profile can be documented over time, which could identify trends and detect stability or progression of the disease as well as detect the efficacy of various therapies in real time.

The MenaCalc platform targets breast, prostate, lung, and colorectal cancers, including the roughly 40% of breast cancer patients who have HER2-positive or **triple-negative cancer (TNC)** tumors and for whom there are not many viable prognostic options on the market today.

Clinical studies have occurred at MIT, Cornell, and Yale University, with two trials recently completed at Yale. Results of one of these trials were published in *Breast Cancer Research* during 2012 in an article entitled "Quantitative Assessment of Invasive Mena Isoforms (MenaCalc) as an Independent Prognostic Marker in Breast Cancer" (Source: Agarwal et al., *Breast Cancer Research* 2012; 14[5]:R124). Most recently, research confirming the utility of the MenaCalc platform was presented at the 2014 Annual Meeting of the U.S. and Canadian Academy of Pathology. Collectively, preclinical and clinical research has supported MetaStat's belief that MenaCalc measures tumor invasiveness and has the potential to predict metastatic risk and consequently change the treatment paradigm in multiple tumor types.

PLATFORM TECHNOLOGY

Figure 25
MENA ISOFORM EXPRESSION MAY INDICATE METASTATIC POTENTIAL OF INVASIVE TUMOR



Source: MetaStat, Inc.

MenaCalc is a platform technology intended to enable a rapid and cost-effective expansion into new oncology indications since expression of the Mena protein is linked to multiple epithelial cancer types.

As published in *Clinical & Experimental Metastasis* in 2011 in an article entitled, "Mena invasive ($Mena^{INV}$) and Mena 11a isoforms play distinct roles in breast cancer cell cohesion and association with TMEM," $Mena^{INV}$ is associated with greater tumor metastasis than noninvasive Mena (Source: Roussos et al., *Clinical & Experimental Metastasis* 28(6):515-27). Researchers have found that Mena occurs differently in invasive tumor cells versus "non-motile resident" (i.e., noninvasive) tumor cells, whereby invasive tumor cells express relatively higher levels of $Mena^{INV}$ and lower levels of the Mena 11a isoform, and noninvasive tumor cells express relatively lower levels of $Mena^{INV}$ but express relatively higher levels of Mena 11a. This effect is illustrated in Figure 25.

The overexpression of $Mena^{INV}$ and downregulation of Mena 11a in tumor cells have shown to be precursors to the formation of MetaSites—the unique three-celled structures that enable metastatic tumor cells to enter into the bloodstream and spread through to other organs in the body (details on pages 30-32). This technology has shown to be associated with disease survival, and can be used to stratify patients into low- and high-risk for metastasis and poor outcomes.

Figure 26

KEY ADVANTAGES OF THE MENACALC DIAGNOSTIC PLATFORM

- Requires very little tissue (core or fine needle biopsy) and readily fits into current diagnostic paradigm
- Ability to aid in surgical decision-making in addition to informing decision-making regarding radiation therapy and chemotherapy
- Mena expression linked to several cancers, including breast, prostate, lung, and colorectal
- Platform technology enables rapid and cost-effective expansion into newer indications

Source: MetaStat, Inc.

MenaCalc *Breast*

MenaCalc *Breast* is an individualized tissue-based test that can be performed on disassociated, discontinuous cells obtained by a needle biopsy of breast cancer tissue (in a standard biopsy procedure known as "**fine needle aspiration**"). After extraction from the patient, the cells are evaluated for the presence and ratio of the noninvasive Mena 11a isoform to the invasive Mena^{INV} isoform.

Using the MenaCalc diagnostic platform, MetaStat can determine each patient's individual expression levels of the isoforms of the Mena protein in cancer tissue. The relationship between Mena 11a and Mena^{INV} is used to compute a "MenaCalc *Breast* Metastasis Score," which may predict a breast cancer patient's individual metastatic profile (i.e., determining whose cancer is likely to continue to spread and whose may stay localized) as early on in disease progression as possible. Because it is designed to be effective on disassociated cells, the MenaCalc *Breast* test may eventually represent a valuable tool for determining prognosis before the patient undergoes surgery to remove the tumor(s).

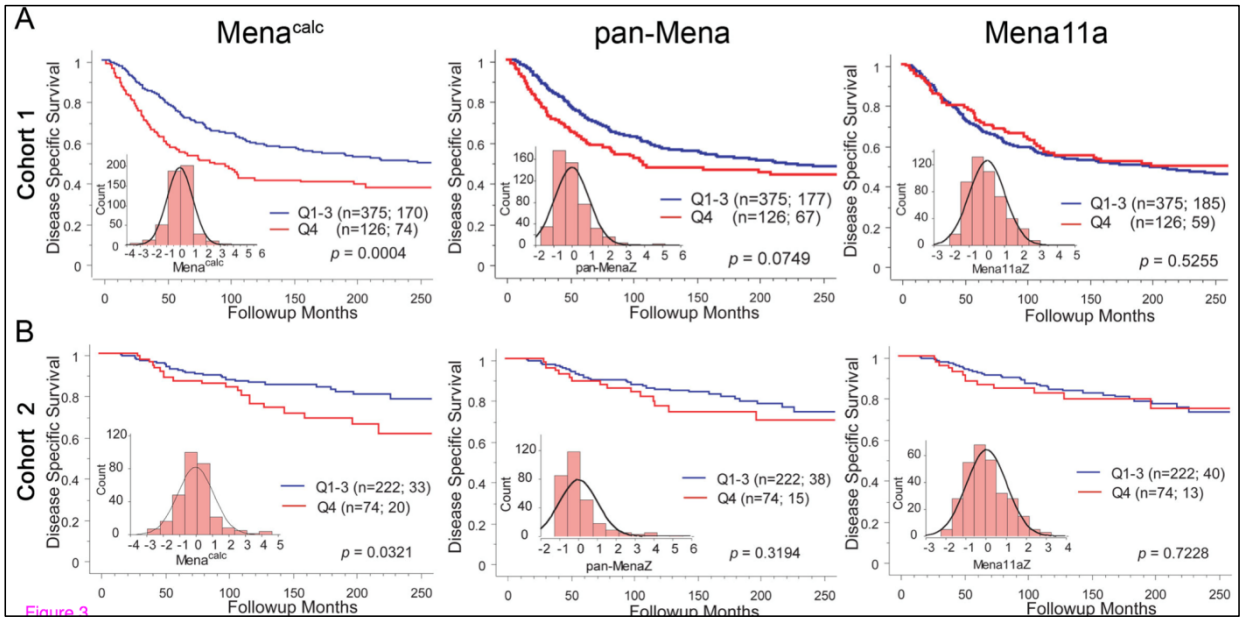
Trial at Yale University School of Medicine

MetaStat reports that research to date has illustrated a correlation between the MenaCalc *Breast* Metastasis Score and the MetaSite *Breast* Metastasis Score. The Company conducted a roughly 800-patient study at Yale confirming the predictive value of the MenaCalc *Breast* Metastasis Scores. Data from this trial was published in *Breast Cancer Research* during 2012 in an article, entitled "Quantitative Assessment of Invasive Mena Isoforms (MenaCalc) as an Independent Prognostic Marker in Breast Cancer" (Source: Agarwal et al., *Breast Cancer Research* 2012; 14[5]:R124).

In this study, the prognostic value of MenaCalc was assessed in hundreds of patient tissue samples which had 20 to 30 years of follow-up data. Cohort 1 in Figure 27 (page 42) shows data from 501 patients with 20 years of follow-up. In this cohort, MenaCalc was associated with poor outcome (log rank P-value = 0.0004). MenaCalc was also prognostic in Cohort 2, which had 296 tissues samples with accompanying medical histories from 1976 to 2005. In cohort 2, MenaCalc was also associated with poor outcome (log rank P-value = 0.0321). On their own, Pan-Mena and Mena 11a (the anti-invasive isoform) were not found to stratify patients for poor outcome in either cohort.

Figure 27

MENACALC APPEARS TO BE ASSOCIATED WITH DISEASE SURVIVAL

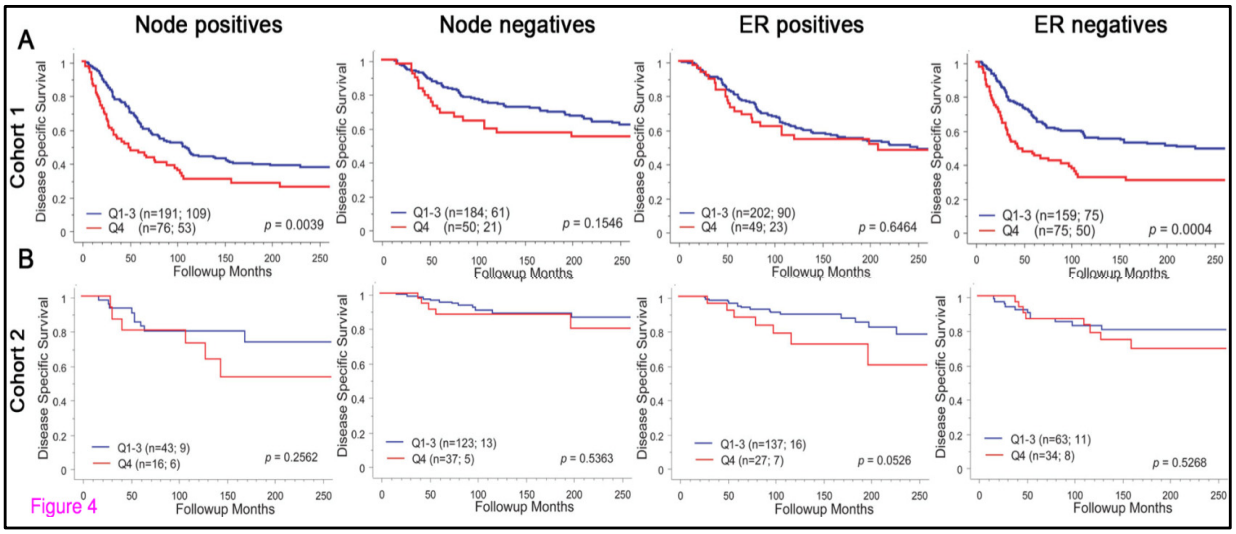


Source: Argarwal, et al., Breast Cancer Research, 2012.

Additional data indicates that MenaCalc may be prognostic for node-positive (cancer that has spread to the lymph nodes) and ER-negative (or triple-negative cancer [TNC]) molecular subtypes of breast cancer that are not addressed with current prognostic tools such as Onco*type* DX®, etcetera. In cohort 1 in Figure 28, MenaCalc was prognostic for poor outcomes in the node-positive subset (log rank P-value = 0.0039) and outcomes in the ER-negative subset (log rank P-value = 0.0004). Cohort 2 was less well-powered with fewer samples/events and did not show statistical significance.

Figure 28

MENACALC IS PROGNOSTIC FOR NODE-POSITIVE AND ER-NEGATIVE TUMOR SUBSETS



Source: Argarwal, et al., Breast Cancer Research, 2012.

MetaStat believes that further validation studies are needed in order to begin initial marketing of the MenaCalc assay for breast cancer. During 2014, the Company plans to perform a large-scale, 550- to 1,000-patient validation study in breast cancer with metastatic risk as primary endpoint for the MenaCalc *Breast* test.

Third-party Study Confirms MenaCalc's Prognostic Potential

An abstract presented at the 2014 Annual Meeting of the U.S. and Canadian Academy of Pathology further confirmed the potential of the MenaCalc platform. Researchers from the University of Toronto, Yale, MIT, and Albert Einstein College of Medicine sought to build upon the research from Argarwal, et. al. (2012) to determine if this technology could be used as an independent prognostic marker for axillary node-negative (ANN) breast cancer. The team of 10 investigators, which included Dr. John Condeelis, Dr. Frank Gertler, and Dr. Thomas Rohan who together comprise MetaStat's Scientific Advisory Board for Diagnostics (biographies on page 15), applied MenaCalc to 406 breast cancer tissue samples from individuals who had been patients at eight Toronto hospitals between 1987 and 1996.

The tissue samples were then stratified into four groups based on their MenaCalc score, and analyzed to determine an association between MenaCalc score and overall survival, accounting for the impact of other factors such as HER2 status, patient age, tumor size, and so on. The results showed that high MenaCalc levels were associated with decreased overall survival in axillary node-negative breast cancer patients independent of other factors, indicating that MenaCalc may be an effective prognostic biomarker for this patient population (Source: *Mena^{calc}, a Quantitative Method of Metastasis Assessment, as a Prognostic Marker for Axillary Node-Negative Breast Cancer*, 2014).

MenaCalc *Lung*, *Prostate*, and *Colorectal* Indications

In addition to MenaCalc *Breast*, MetaStat believes the MenaCalc technology may be suitable as a new diagnostic tool for an array of epithelial-based cancers. The Mena protein is thought to be a key factor contributing to the progression of metastasis in multiple epithelial-based solid tumors, potentially including pancreas, prostate, colon, brain, liver, lung, head, and neck tumors in addition to prior findings for breast cancer. As these cancers represent many of the world's most common tumor types, diagnostic and prognostic tests based on the MenaCalc platform may ultimately address millions of patients globally.

To this effect, MetaStat is evaluating MenaCalc's efficacy in lung, prostate, and colorectal cancers. Data collected to date using MenaCalc in lung adenocarcinoma demonstrates high predictive accuracy in distant metastasis. In early 2011, the Company completed a pilot study at MIT that produced promising preliminary data supporting the ability of a MenaCalc *Prostate* test for predicting prostate cancer metastasis. Consequently, MetaStat now plans to conduct a larger, confirmatory trial for the MenaCalc *Prostate* product candidate. The development of a prognostic test, such as MetaStat's for prostate cancer, could be particularly beneficial for patients and physicians alike, as it would allow patients to make an informed decision regarding treatment based on the knowledge of whether or not their tumor will metastasize. Prostate tumors are by nature slow growing, and many men die of old age before they are affected by their prostate cancer (Source: National Cancer Institute). By knowing their risk of the tumor spreading, prostate cancer patients can decide between active surveillance and surgery or radiation therapy, thereby allowing patients with favorable metastasis profiles to avoid the risks associated with high-impact cancer treatments.

The Company has also completed a 70-patient study at Yale that was designed to evaluate the ability of MenaCalc *Lung* to predict metastasis of lung adenocarcinomas. The study confirms the ability of MenaCalc *Lung* to predict survival. MetaStat plans to execute a large-scale proof-of-concept study in adenocarcinoma of the lung for MenaCalc *Lung* in 2015/2016.

MenaBloc Therapeutic Program

Preventing or reducing metastasis is a critical component of successfully treating cancer and decreasing the number of cancer-related deaths (Source: *European Journal of Cancer* 2010; 46:1177-1180). Yet to MetaStat's knowledge, there is no therapeutic approved specifically to target metastasis. Currently, treatment for metastatic disease is similar to, but more aggressive, than that used to target primary tumors, and includes chemotherapy, biological therapy, targeted therapy, hormonal therapy, local therapy, surgery (although more rarely for metastatic disease), radiation therapy, or a combination thereof. Treatment depends on the type of cancer as well as the size, location, and number of metastatic tumors, the patient's age and health, and the therapies used previously. Radiation therapy remains a mainstay of treatment for metastatic tumors, and patients may also receive multiple chemotherapy courses. The primary goal of these treatments is to control the growth of the cancer or to relieve symptoms.

However, conventional methods for treating metastatic tumors are centered on the notion that metastases were essentially similar to primary tumors, and thus assumed that therapies to control primary tumor growth could also suppress metastatic growth. However, accumulating evidence has demonstrated that traditional growth control approaches are not only inadequate to combat metastatic disease, but may also be harmful in the long term (Source: *European Journal of Cancer* 2010; 46:1177-1180). Today, metastatic disease remains the chief reason why cancer patients succumb to their disease, accounting for approximately 90% of cancer fatalities (Source: CancerQuest, Emory University's cancer education and outreach program, October 2011).

Advancing a Drug Discovery Laboratory Specifically for Metastatic Cancer Therapeutics

Believing that there is a significant unmet need for therapies based on a solid understanding of the process of metastatic disease, including techniques to kill or stop the spread of metastatic cancer cells or to disrupt individual steps in the metastatic process, MetaStat's pipeline includes a therapeutic program designed to discover inhibitors of the Mena pathway. The Company opened a drug discovery laboratory in October 2013 staffed with highly qualified scientists who are experienced in molecular cancer research. The team at this laboratory is led by Dr. Elizabeth Buck, who works in close collaboration with MetaStat's Scientific Advisory Board for Therapeutics' chairman, Dr. David Epstein.

Prior to joining MetaStat, Drs. Buck and Epstein worked together at OSI Pharmaceuticals along with other research scientists who are also now with MetaStat. (Dr. Epstein is currently director of the Center for Technology and Development at the Duke-NUS Medical School in Singapore.) Among other initiatives, OSI focused on research into key drivers of cancer development and disease progression—efforts that were led by Dr. Epstein as OSI's chief scientific officer for oncology research. At OSI, these individuals produced a pipeline of oncology products comprising four small molecule kinase inhibitors, which are now in clinical development (Source: Duke-NUS). Prior to its 2010 acquisition by Astellas Pharmaceuticals, OSI was regarded for its expertise in **personalized cancer therapies** and for discovering and understanding the mechanisms of tumor cells' epithelial to mesenchymal transition (EMT), a factor in tumors' resistance to targeted treatments.

MetaStat aims to screen and develop cancer therapeutics targeting Mena in multiple epithelial-based tumor types, with the objective of entering Phase I clinical trials in 2017.

MIT Licenses

In December 2013, MetaStat licensed a collection of alternatively spliced therapeutic targets that have a role in the epithelial to mesenchymal transition (EMT) of tumor cells. As described on page 24, EMT is an early event in the metastatic process and contributes to therapeutic resistance in breast and other cancers. Scientific research has shown that EMT–dependent splicing changes occur in human breast cancer tumors, and have the ability to impact tumor progression and resistance (Source: Shapiro et. al., *PLoS Genetics*, August 2011, Volume 7, Issue 8). This discovery is key to MetaStat's current therapeutic program, which combines the Company's understanding of alternatively spliced Mena isoforms with technology and patents recently licensed from MIT, the Koch Institute for Integrative Cancer Research, the Albert Einstein College of Medicine, and the Montefiore Medical Center relating to the use of alternatively spliced mRNA and protein isoform markers in the diagnosis, prognosis, and treatment of metastatic, epithelial-based solid tumors.

MetaStat believes that targeting the alternative splicing events implicated in EMT (and thus metastasis and tumor resistance) presents a novel opportunity for new cancer therapeutics linked to a diagnostic platform—enabling detection of patients' unique tumor biomarkers and offering a treatment approach if needed to delay patients' tumor progression and decrease metastatic spread.

Additional Preclinical Research Supporting the Development of a Mena-inhibitor Therapeutic

Scientists from MIT, the Albert Einstein College of Medicine, Cornell, and IFO-Regina have previously created a mouse that was unable to produce Mena protein. These mice are known as "Mena null" mice. Once developed, researchers cross bred the Mena null mice with PyMT mice, which are known to be genetically predisposed to highly metastatic breast cancer. The resulting mice were afflicted with metastatic breast cancer but did not have the Mena protein. The engineered Mena null, PyMT mice were then compared to typical PyMT mice, which formed the control group for these studies. Without the Mena null cross-breeding, typical PyMT mice are commonly used as a model for human breast cancer tumors. These mice are predisposed to develop metastatic breast cancer that goes through distinct stages of tumor progression and metastasis comparable to the progression of human breast disease.

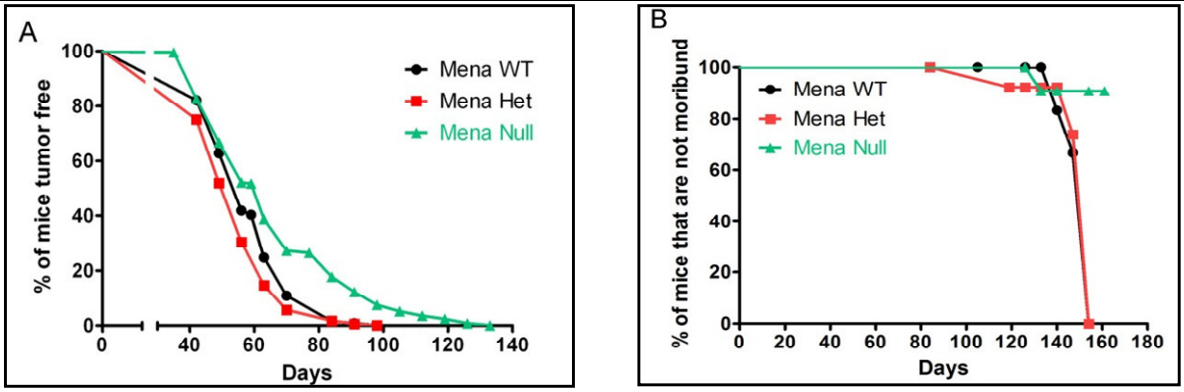
Result Highlights

Researchers found that while both groups of mice developed breast tumors, only control mice showed metastatic tumors. Due to the rapid spread of disease in the control mice, all of these subjects were killed by their tumors. In contrast, cancer in the Mena null, PyMT mice remained localized and these subjects showed a significant survival advantage. Most Mena null, PyMT mice were able to die of old age (Source: "Mena deficiency delays tumor progression and decreases metastasis in **polyoma middle-T** transgenic mouse mammary tumors," *Breast Cancer Research*, 2010). Figure 29 (page 46) illustrates a selection of key data from this research, noting that mice were considered "not moribund"—mice that have not yet developed tumors or that have small tumors that have not immobilized them—until their death. Mice with excessive tumor burden causing illness or immobilization were euthanized.

Figure 29

A DEFICIENCY OF MENA INCREASES PyMT TUMOR LATENCY AND SURVIVAL IN MICE

Percent of mice without palpable tumors at the given age	Kaplan Meir curve measuring % of mice that are not moribund (mice that have not yet developed tumors or that have small tumors that have not immobilized them)
N >100 mice/genotype	N = 12 to 25 mice/genotype



Mena WT = Wild Type (control group; PyMT mice where Mena is unaffected)

Mena Het = Heterozygote (Mena isoforms are reduced in heterozygote mice versus completely eliminated in Mena null mice)

Mena Null = Mice that lack the Mena protein or its isoforms

Source: Roussos et al. Breast Cancer Research 2010 12:R101 <http://breast-cancer-research.com/content/12/6/R101/>.

Product Development

As expected, the discovery of a link between the Mena protein and tumor metastasis could lead to the development of new cancer treatments that block the Mena protein in humans in order to slow or stop the spread of patients' breast cancer. Localized disease is considerably easier to treat through surgery and other existing methods, and is consequently associated with higher survival rates than advanced, metastatic disease. To this end, MetaStat has commenced the design and development of new drug treatments targeted at disabling the action of the Mena protein. Product development on this front includes high-throughput screening of potential molecules that could be used to inhibit Mena.

Figure 30

KEY ADVANTAGES OF MENABLOC

- Use of MenaCalc in multiple tumor types conditions the market toward a therapeutic (MenaBloc) to downregulate Mena expression
- Real opportunities may exist in preventing metastasis among high-risk patients by giving MenaBloc as a maintenance therapy after surgery or in conjunction with chemotherapy and targeted therapies

Source: MetaStat, Inc.

Market Opportunities

Whether the goal is to prevent cancer cells from spreading throughout the body or, for metastatic tumors, to cease further growth and spread, metastasis is a significant challenge in cancer treatment and a leading cause of cancer-related deaths. Over 1.6 million new cancer cases were diagnosed in the U.S. in 2013, with as many as 1,440 people dying *each day* from a metastatic cancer.

Figure 31 contains data for the numbers of new cases and deaths in 2013 alone for each of MetaStat's four tumor targets: breast, lung, prostate, and colorectal cancers.

Figure 31

NEW CASES AND MORTALITY FOR METASTAT'S TARGET ONCOLOGY INDICATIONS (2013 data)

	Estimated New Cases			Estimated Deaths		
	Both Sexes	Male	Female	Both Sexes	Male	Female
Breast	234,580	2,240	232,340	40,030	410	39,620
Lung and Bronchus	228,190	118,080	110,110	159,480	87,260	72,220
Prostate	238,590	238,590		29,720	29,720	
Colon†	102,480	50,090	52,390	50,830	26,300	24,530
Rectum	40,340	23,590	16,750			

† Estimated deaths for colon and rectal cancers are combined.

Source: the American Cancer Society, Inc.'s "Cancer Facts & Figures," 2013.

Excluding skin cancers, breast cancer is the most frequently diagnosed cancer for females (Source: the American Cancer Society's *Cancer Facts & Figures*, 2013). In 2013, an estimated 232,340 women were diagnosed with invasive breast cancer in the U.S. and 64,640 women were diagnosed with in situ (non-invasive) breast cancer. These rates have remained relatively stable over the past few years.

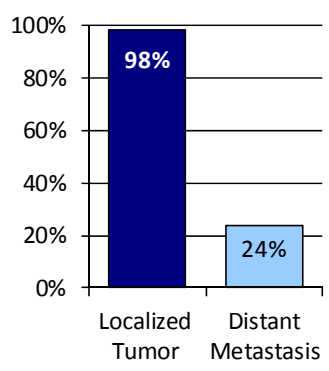
In men, prostate cancer is the most frequently diagnosed cancer type (excluding skin cancers). The lungs and colorectal region are the second and third most common tumor sites for both sexes, and all four of the cancers represent the top three cancer-related killers for everyone in the U.S. They are major markets with hundreds of thousands of patients and an ever-present need for next-generation diagnostics and therapeutics built on new understandings of the pathologies of metastatic tumors.

For all cancer types, a patient's likelihood of survival is highest when the tumor is caught and treated at its earliest stages, before it has spread to secondary sites in the body. For example, a Stage 1 breast cancer patient who has only a localized tumor that has not spread has a 5-year relative survival rate of 98%. Once the tumor metastasizes to distant lymph nodes or other organs, the survival rate drops to 24%.

MetaStat estimates that as many as 225,000 patients a year are diagnosed with Stages 0 to 3 breast tumors. For many of these patients, their metastatic risk is inferred from a variety of clinical and pathological assessments, and for approximately 35% of these individuals, a whole genome assay based on proliferation genes is performed to look for any upregulated markers of cancer cell proliferation. However, upregulation of proliferation genes alone does not always adequately separate, or stratify, the high-risk patients from the low-risk in terms of their likelihood for developing metastatic disease or having a poor disease outcome.

Figure 32

BREAST CANCER SURVIVAL RATES



Source: Cancer Facts & Figures, 2013.

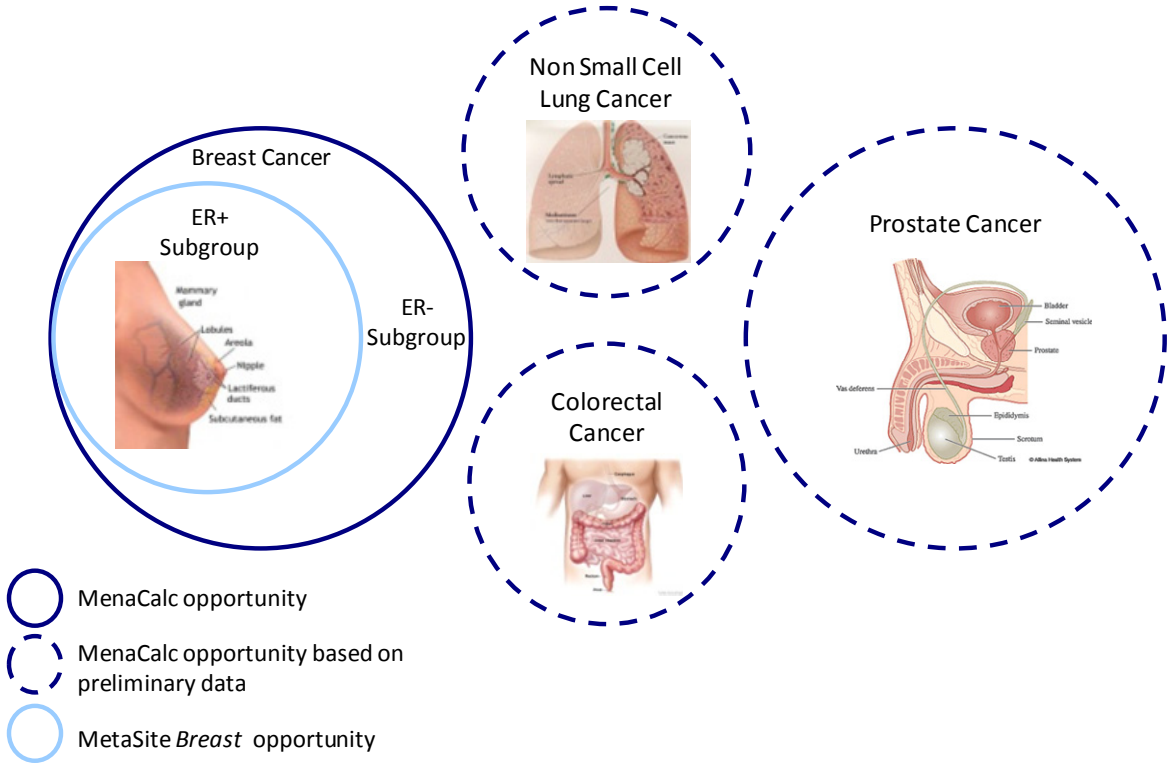
Due to existing methods of assigning patients to risk subgroups based on their cancer's pathology, physicians and patients may not know whether treatment decisions based on the grouping is actually appropriate for the patient's individual risk. Consequently, many patients have to tolerate the toxicity and morbidity of aggressive treatment when their real metastatic risk is low while others miss the small window of time when early and aggressive treatment could have made a real difference.

MetaStat is not the only entity that believes there can be a better way to accurately distinguish which patients are likely to develop systemic metastatic disease and which are not. Experts from the National Institutes of Health (NIH) recently concluded that a primary goal for future research should be to accurately define patient risk categories with the objective of being able to administer the level of treatment required for a successful outcome (Source: *Cancer Facts & Figures*, 2013).

Figure 33 illustrates how MetaStat views its primary market opportunities for providing improved metastatic risk stratification, noting that its MetaSite *Breast* and MenaCalc *Breast* diagnostic assays could complement each other in order to provide metastatic risk coverage to the entire breast cancer market (multiple molecular subgroups). This is unlike current assays in this market which generally target only ER+ tumor types. Prostate cancer could also become a major market for the Company, as 93% of all prostate cancers are in the local or regional stages at diagnosis—representing over 220,000 people each year (as of 2013) who could benefit from an improved measure of their risk of developing metastatic prostate cancer.

Figure 33

OPPORTUNITIES FOR METASITE *BREAST* AND MENACALC TO MEET UNMET NEEDS FOR BETTER RISK STRATIFICATION



Source: MetaStat, Inc.

Competition

Competition for MetaStat includes existing diagnostic products as well as new diagnostics that may be introduced over the coming years as the Company completes development of its products. It can be difficult to change existing diagnostic approaches, as they have become routine procedures used by physicians, pathologists, oncologists, laboratories, and others in the medical community. Although MetaStat is focused on driving development and adoption of its tests for use outside the laboratory, the Company will likely also have to address competition from the kits and reagents used by local pathology labs.

Due to the number of firms and academic or government institutions researching new diagnostic techniques and new cancer therapies, MetaStat may compete with a variety of entities for funding, resources, and market share. These include other developers of diagnostic tests as well as companies that focus on gene profiling and gene or protein expression and commercial laboratories that have strong distribution networks for diagnostic tests. MetaStat believes that its technology and scientific expertise offer multiple competitive advantages, as detailed throughout the Core Story on pages 20-48.

The companies listed below are not intended to be an exhaustive collection of MetaStat's possible competition, yet is believed to be representative of the type of competitors that the Company may encounter going forward.

Competitors that Develop Diagnostic Tests

Agendia, Inc. (www.agendia.com)
Genomic Health, Inc. (www.genomichealth.com)
Genoptix Medical Laboratory, part of the Novartis Pharmaceuticals Division (www.genoptix.com)
Roche Diagnostics, a division of Roche Holdings (www.roche.com/diagnostics)
Siemens Healthcare Diagnostics, part of Siemens AG (www.siemens.com/about/en/businesses/healthcare/diagnostics.htm)
Veridex LLC, a Johnson & Johnson company (www.veridex.com)

Companies that Focus on Gene Profiling and Gene or Protein Expression

Celera Corp., a subsidiary of Quest Diagnostics Inc. (www.celera.com)
GE Healthcare Ltd., the global healthcare unit of General Electric Co. (www.gehealthcare.com)
Hologic, Inc. (www.hologic.com)
Myriad Genetics, Inc. (www.myriad.com)
Novartis AG (www.novartisdiagnostics.com)
Qiagen N.V. (www.qiagen.com)
Response Genetics, Inc. (www.responsegenetics.com)

Commercial Laboratories with Strong Distribution Networks for Diagnostic Tests

Laboratory Corporation of America Holdings (www.labcorp.com)
Quest Diagnostics Inc. (www.questdiagnostics.com)

Figure 34
TICKER SYMBOLS AND MARKET CAPITALIZATIONS
(as of April 22, 2014)

Company	Ticker	Market Cap.
Agendia, Inc.	*private*	—
Genomic Health, Inc.	GHDX-NASDAQ	$874 M
Genoptix Medical Laboratory	*part of Novartis*	—
Roche Diagnostics	RHHBY-OTC	$123 B
Siemens Healthcare Diagnostics	SI-NYSE	$115 B
Veridex LLC	*part of JNJ*	—
Celera Corp.	*part of Quest*	—
GE Healthcare Ltd.	GE-NYSE	$267 B
Hologic, Inc.	HOLX-NASDAQ	$5.7 B
Myriad Genetics, Inc.	MYGN-NASDAQ	$2.9 B
Novartis AG	NVS-NYSE	$210 B
Qiagen N.V.	QGEN-NASDAQ	$4.9 B
Response Genetics, Inc.	RGDX-NASDAQ	$49 M
Laboratory Corp. of America Holdings	LH-NYSE	$8.6 B
Quest Diagnostics Inc.	DGX-NYSE	$8.4 B

Sources: MetaStat Inc. and Yahoo! Finance.

Key Points

- MetaStat, Inc. is developing next-generation diagnostic and therapeutic products for metastatic cancer, which is the cause of up to 90% of solid tumor cancer-related deaths. The Company's pipeline is based on over 15 years of research and collaboration by the Massachusetts Institute of Technology (MIT), Albert Einstein College of Medicine of Yeshiva University, Cornell University, and Italy's IFO-Regina Elena Cancer Institute.

- MetaStat's platform technologies may improve diagnosis and treatment for up to 80% of all solid tumor cancers, including breast, prostate, lung, bowel, pancreatic, brain, liver, and head and neck cancers.

- MetaStat's technology focuses on predicting the risk of cancer metastasis based on its underlying mechanisms, which entails identifying the structural and behavioral mechanisms that allow cancer cells to move and determining how this information can be used in prognosis. To MetaStat's knowledge, its technology is the only technique to focus on such mechanistic markers.

- MetaStat's initial product opportunity is MetaSite *Breast*, a candidate designed to predict the risk of breast cancer progression. MetaSite *Breast* has been validated in clinical studies and the Company expects it to have a low-cost and rapid path to market as early as 2015.

    - MetaStat's products are designed to overcome limitations of gene-based diagnostic tests in order to improve the quality of treatment decisions. Clinical data suggests that MetaSite *Breast* enables a higher degree of precision in stratifying risk than competitive products.

    - Breast cancer is one of the most commonly diagnosed cancers for women. In 2013, more than 232,000 women were thought to have been diagnosed with invasive breast cancer in the U.S., adding to the millions of women already living with this diagnosis (Source: *Breast Cancer Facts & Figures 2013*).

- The Company is also developing multiple products based on its MenaCalc diagnostic platform: (1) MenaCalc *Breast*; (2) MenaCalc *Lung*; (3) MenaCalc *Prostate*; and (4) MenaCalc *Colorectal*.

    - A correlation between the MenaCalc *Breast* Metastasis Score and the MetaSite *Breast* Metastasis Score has been confirmed in a 797-patient study. In addition, the Company believes its MenaCalc *Breast* test can provide diagnostic and prognostic information for women with all sub-types of breast cancer, which has the potential to separate MetaStat from the competition.

    - Unpublished data collected in lung adenocarcinoma and prostate tumors have shown the ability of MenaCalc to predict cancer spread in these cancer types.

- In October 2013, MetaStat opened a drug discovery laboratory in affiliation with Stony Brook University in order to advance its MenaBloc program for identifying potential therapeutic targets for metastatic cancer. The Company has recruited well-known cancer researchers from OSI Pharmaceuticals to head up its drug discovery efforts.

- MetaStat holds rights to three issued U.S. patents and 10 pending patent applications globally.

- MetaStat's leadership is experienced in biotechnology/life sciences research and management, including at major global firms such as Roche. The Company possesses highly skilled Scientific and Clinical Advisory Boards, with individuals from MIT, the Albert Einstein College of Medicine, and Cornell who were instrumental in pioneering the academic research supporting MetaStat's product development.

- As of November 30, 2013, MetaStat had $488,108 in cash. The Company seeks to raise $5 million to $7 million to reach important milestones.

Historical Financial Results

The following Figures 35, 36, and 37 provide a summary of MetaStat's key historical financial statements: its Statements of Operations, Balance Sheets, and Statements of Cash Flows, as presented in the Company's Form 10-Q filed with the U.S. Securities and Exchange Commission (SEC) on January 14, 2014.

Figure 35
UNAUDITED CONSOLIDATED STATEMENT OF EXPENSES

	Three Months ended November 30, 2013	Three Months ended November 30, 2012	Nine Months ended November 30, 2013	Nine Months ended November 30, 2012	Period from Inception (July 22, 2009) to November 30, 2013
Revenue					
Interest income	$ 20	$ 94	$ 81	$ 442	$ 677
Total Revenue	20	94	81	442	677
OPERATING EXPENSES					
General & administrative	479,116	364,110	1,406,419	1,237,808	4,071,603
Research & development	90,261	45,000	234,976	378,517	1,776,179
Depreciation	3,538	2,800	10,342	8,213	22,738
Accretion - discount	196,190	–	559,496	–	560,996
Warrant Expense	–	149,995	–	149,995	378,688
Stock-based compensation	648,087	(5,806)	3,268,253	5,259	4,032,357
Total Operating Expenses	1,417,192	556,099	5,479,486	1,779,792	10,842,561
NET LOSS	$ (1,417,172)	$ (556,005)	$ (5,479,405)	$ (1,779,350)	$ (10,841,884)
Basic & Diluted Net Loss Per Share	$ (0.07)	$ (0.03)	$ (0.26)	$ (0.09)	
Weighted shares outstanding	21,469,435	21,054,418	21,413,084	20,825,840	

Source: MetaStat, Inc.

Figure 36

UNAUDITED CONSOLIDATED BALANCE SHEETS

	November 30, 2013	February 28, 2013
ASSETS		
CURRENT ASSETS		
Cash	$ 488,108	$ 969,188
Prepaid Insurance	42,654	–
Total Current Assets	530,762	969,188
PROPERTY AND EQUIPMENT		
EQUIPMENT (net of accumulated depreciation of $28,710 and $1,271, respectively)	208,393	53,326
OTHER ASSETS		
Refundable deposit	4,667	–
TOTAL ASSETS	$ 743,822	$ 1,022,514
LIABILITIES AND STOCKHOLDERS' EQUITY		
LIABILITIES		
Accounts payable and accrued liabilities	$ 240,896	$ 168,005
Short-term note payable	10,536	–
Convertible debentures - net of discount of $139,879	1,847,121	716,957
Accrued interest payable	91,477	1,940
TOTAL LIABILITIES	2,190,030	886,902
STOCKHOLDERS' EQUITY		
Preferred stock, 10,000,000 shares authorized; no shares issued and outstanding	–	–
Common stock, $0.0001 par value; 150,000,000 shares authorized; 21,469,435 and 21,054,418 shares issued and outstanding, respectively	2,147	2,106
Paid-in-capital	9,393,529	5,495,985
Accumulated deficit as a development-stage company	(10,841,884)	(5,362,479)
Total equity	(1,446,208)	135,612
TOTAL LIABILITIES AND STOCKHOLDERS' EQUITY	$ 743,822	$ 1,022,514

Source: MetaStat, Inc.

Figure 37
UNAUDITED CONSOLIDATED STATEMENT OF CASH FLOWS

	Nine Months ended November 30, 2013	Nine Months ended November 30, 2012	Period from Inception (July 22, 2009) to November 30, 2013
CASH FLOWS FROM OPERATING ACTIVITIES			
Net loss	$ (5,479,405)	$ (1,779,350)	$ (10,841,884)
Adjustments to reconcile net loss to net cash used in operating activities:			
Interest payable	89,537	–	91,477
Depreciation expense	10,342	8,213	22,738
Warrant expense	–	149,995	378,688
Option expense	1,944,261	–	1,944,261
Common stock issued for services	1,323,992	5,259	2,088,097
Accretion of discount	559,497		560,997
Changes in assets and liabilities:			
Prepaid expenses and other current assets	(42,654)	–	(42,654)
Refundable deposit	(4,668)	–	(4,668)
Accounts payable	72,891	(250,034)	240,896
NET CASH USED IN OPERATING ACTIVITIES	(1,526,207)	(1,865,917)	(5,562,052)
CASH FLOWS FROM INVESTING ACTIVITIES			
Cash paid for purchase of fixed assets	(165,409)	(290,507)	(231,131)
NET CASH USED IN INVESTING ACTIVITIES	(165,409)	(290,507)	(229,002)
CASH FLOWS FROM FINANCING ACTIVITIES			
Proceeds from sale of stock	–	880,000	3,418,755
Proceeds from subscription receivables	–	865,000	865,000
Payments on short-term debt	(62,641)	–	(62,641)
Borrowings on short-term debt	73,177	–	73,177
Borrowings on convertible debentures	1,200,000	–	1,987,000
NET CASH PROVIDED BY FINANCING ACTIVITIES	1,210,536	1,745,000	6,281,291
NET INCREASE (DECREASE) IN CASH	(481,080)	(411,424)	488,108
Cash at the beginning of the year	969,188	878,340	–
Cash at the end of the year	$ 488,108	$ 466,916	$ 488,108
SUPPLEMENTAL DISCLOSURES:			
Interest Paid	$ –	$ –	$ –
Income taxes paid	$ –	$ –	$ –
NON-CASH TRANSACTIONS			
Recapitalization of PVSO shareholders	$ –	$ –	$ 8
Debt discount	$ 629,332	$ –	$ 700,875

Source: MetaStat, Inc.

Risks and Disclosures

This Executive Informational Overview® (EIO) has been prepared by MetaStat, Inc. ("MetaStat" or "the Company") with the assistance of Crystal Research Associates, LLC ("CRA") based upon information provided by the Company. CRA has not independently verified such information. Some of the information in this EIO relates to future events or future business and financial performance. Such statements constitute forward-looking information within the meaning of the Private Securities Litigation Act of 1995. Such statements can only be predictions and the actual events or results may differ from those discussed due to the risks described in MetaStat's statements on Forms 10-K, 10-Q, and 8-K, as well as other forms filed from time to time.

The content of this report with respect to MetaStat has been compiled primarily from information available to the public released by the Company through news releases, Annual Reports, and U.S. Securities and Exchange Commission (SEC) filings. MetaStat is solely responsible for the accuracy of this information. Information as to other companies has been prepared from publicly available information and has not been independently verified by MetaStat or CRA. Certain summaries of activities and outcomes have been condensed to aid the reader in gaining a general understanding. CRA assumes no responsibility to update the information contained in this report. In addition, CRA has been compensated by the Company in cash of sixty-five thousand U.S. dollars and one hundred and fifty thousand warrants for its services in creating this report and for updates. For more complete information about the risks involved in an investment in MetaStat, please see the Company's Form 10-K filed with the SEC on May 28, 2013, and available at the following link: www.sec.gov/Archives/edgar/data/1404943/000141588913001075/mtst10k_feb282013.htm.

Investors should carefully consider the risks and information about MetaStat's business, as fully described in the Company's Form 10-K filed with the SEC on May 28, 2013, and overviewed below. Investors should not interpret the order in which considerations are presented in this or other filings as an indication of their relative importance. The risks and uncertainties overviewed in MetaStat's Form 10-K and below are not the only risks that the Company faces. Additional risks and uncertainties not presently known to MetaStat or that it currently believes to be immaterial may also adversely affect the Company's business. If any of such risks and uncertainties develops into an actual event, MetaStat's business, financial condition, and results of operations could be materially and adversely affected, and the trading price of the Company's shares could decline.

This report is published solely for information purposes and is not to be construed as an offer to sell or the solicitation of an offer to buy any security in any state. Past performance does not guarantee future performance. Additional information about MetaStat and its public filings, as well as copies of this report, can be obtained by calling (832) 758-7488.

RISKS RELATING TO METASTAT'S BUSINESS

If MetaStat is unable to continue as a going concern, its securities will have little or no value.

The report of the Company's independent registered public accounting firm that accompanies the Company's audited consolidated financial statements for the years ended February 28, 2013, and February 29, 2012, contains a going concern qualification in which such firm expressed substantial doubt about the Company's ability to continue as a going concern. As of November 30, 2013, the Company had an accumulated deficit of $10,841,884. MetaStat currently anticipates that its cash and cash equivalents will be sufficient to fund its operations through May 2014, without raising additional capital. The continuation of the Company as a going concern is dependent upon continued financial support from its shareholders, the ability of the Company to obtain necessary equity and/or debt financing to continue operations, and the attainment of profitable operations. These factors raise substantial doubt regarding the Company's ability to continue as a going concern. MetaStat cannot make any assurances that additional financings will be available to it and, if available, completed on a timely basis, on acceptable terms, or at all. If the Company is unable to complete a debt or equity offering, or otherwise obtain sufficient financing when and if needed, it would negatively impact its business and operations, which would likely cause the price of its common stock to decline. It could also lead to the reduction or suspension of the Company's operations and ultimately force the Company to go out of business.

The Company is at an early stage of development as a company and does not have, and may never have, any products that generate revenues.

The Company is a development-stage life sciences company and has incurred substantial net losses since inception. To date, the Company has not achieved, and may never achieve, revenues sufficient to offset expenses. At this time, MetaStat does not have any commercial products or laboratory services that generate revenues. MetaStat's existing diagnostic offerings will require additional clinical evaluation, regulatory review, significant marketing efforts, and substantial investment before they could provide any revenues. Given MetaStat's stage of development, the Company expects to be able to begin initial marketing as early as 2015 for the MetaSite *Breast* test and commence implementation of a sales and marketing strategy as early as 2015. If the Company is unable to develop, receive approval for, or successfully commercialize any of its diagnostic candidates, it will be unable to generate significant revenues or any revenues at all. If MetaStat's development programs are delayed, the Company may have to raise additional capital or reduce or cease operations.

The Company expects to continue to incur significant research and development (R&D) expenses, which may make it difficult to achieve profitability.

In recent years, the Company has incurred significant costs in connection with the development of the MetaSite *Breast* test, the MenaCalc platform of diagnostics assays for breast, prostate, and lung cancers, as well as initial work on the MenaBloc therapeutic. MetaStat's R&D expenses were $516,798 and $854,550 for the fiscal years ended February 28, 2013, and February 29, 2012, respectively. The Company expects R&D expense levels to remain high for the foreseeable future as it seeks to expand the clinical utility of the MetaSite *Breast* test and develop additional diagnostics in its product portfolio. As a result, the Company will need to generate significant revenues in order to achieve profitability. MetaStat's failure to achieve profitability in the future could cause the market price of its common stock to decline.

The Company does not have its own research facilities and will be dependent on third parties for product development.

The Company does not have its own R&D facilities and may engage consultants and independent contract research organizations to design and conduct clinical trials in connection with the development of MetaStat's products. As a result, these important aspects of a product's development will be outside of MetaStat's direct control. In addition, there can be no assurance that such third parties will perform all of their obligations under arrangements with the Company or will perform those obligations satisfactorily.

If the Company fails to obtain additional financing, it will be unable to complete the development and commercialization of product candidates or continue R&D programs.

In addition to the funds raised in recent private placements, the Company may be required to raise additional capital to complete the development and commercialization of current and future product candidates. If the Company is unable to raise additional capital when required or on acceptable terms, it may have to significantly delay, scale back, or discontinue one or more clinical trials and the commercialization of its diagnostic tests.

If third-party payors, including managed care organizations and Medicare, do not provide reimbursement for MetaStat's products, the products' commercial success could be compromised.

The MetaSite *Breast* test has an anticipated list price of $2,595. Physicians and patients may decide not to order the MetaSite *Breast* test unless third-party payors, such as managed care organizations and government payors such as Medicare and Medicaid, pay a substantial portion or all of the test's price. There is significant uncertainty concerning third-party reimbursement of any test incorporating new technology, including the MetaSite *Breast* test and any of MetaStat's future diagnostics and therapies. Reimbursement by a third-party payor may depend on a number of factors, including a payor's determination that tests using MetaStat's technologies are not experimental or investigational, medically necessary, appropriate for the specific patient, cost-effective, and supported by peer-reviewed publications.

Since each payor makes its own decision as to whether to establish a policy to reimburse, seeking these approvals is a time-consuming and costly process. To date, the Company has not secured policy-level reimbursement approval from any third-party payors and has no approvals for state Medicaid programs. The Company cannot be certain that coverage for MetaStat's products will be provided in the future by any third-party payors.

Several entities conduct technology assessments of new medical tests and devices and provide the results of their assessments for informational purposes to other parties. These assessments may be used by third-party payors and healthcare providers such as Blue Cross and Blue Shield plans, which collectively provide healthcare coverage for nearly one-third of all Americans, as grounds to deny coverage for a test or procedure. These assessments have not yet been carried for the MetaSite *Breast* test. The Company can offer no assurance that these evaluations will ever be conducted, and if conducted, will result in a positive conclusion resulting in third-party reimbursement.

Insurers, including managed care organizations and government payors such as Medicare, have increased efforts to control the cost, utilization, and delivery of healthcare services. From time to time, the U.S. Congress has considered and implemented changes in the Medicare fee schedules in conjunction with budgetary legislation. Further reductions of reimbursement for Medicare services may be implemented from time to time. Reductions in the reimbursement rates of other third-party payors have occurred and may occur in the future. These measures have resulted in reduced prices, added costs, and decreased test utilization for the clinical laboratory industry.

If the Company is unable to obtain reimbursement approval from private payors and Medicare and Medicaid programs for its diagnostic tests, or if the amount reimbursed is inadequate, MetaStat's ability to generate revenues could be limited. Even if the Company is being reimbursed, insurers may withdraw their coverage policies or cancel their contracts at any time or stop paying for MetaStat's tests, which would reduce MetaStat's revenue.

The Company may experience delays in clinical trials that could adversely affect its financial position and commercial prospects.

Any delays in completing clinical trials for the MetaSite *Breast* test and MenaCalc platform of diagnostics assays may delay MetaStat's ability to raise additional capital or to generate revenue, and the Company may have insufficient capital resources to support operations. Even if the Company has sufficient capital resources, the ability to become profitable will be delayed if there are problems with the timing or completion of clinical trials. Moreover, if the Company were required to conduct additional trials prior to marketing its diagnostic tests, those trials could lead to delays or failure to obtain necessary regulatory approvals and harm MetaStat's ability to become profitable as well.

Adverse events in MetaStat's clinical trials may force the Company to stop development of its product candidates or prevent regulatory approval, if needed, of its product candidates.

MetaStat's technology platform may provide the Company the opportunity to develop therapeutic candidates to preemptively suppress or eliminate metastasis. The eventual testing of MetaStat's product candidates in human clinical trials may produce serious adverse events. These adverse events could interrupt, delay, or halt clinical trials of product candidates and could result in the FDA or other regulatory authorities denying approval of MetaStat's product candidates for any or all targeted indications. An independent data safety monitoring board, the FDA, other regulatory authorities, or the Company may suspend or terminate clinical trials at any time. The Company cannot assure anyone that any of its product candidates will be safe for human use.

If MetaStat's product candidates do not meet safety or efficacy endpoints in clinical evaluations, they will not receive regulatory approval and the Company will be unable to market them.

There are various federal and foreign statutes and regulations that govern or influence the manufacturing, safety, labeling, storage, record keeping and marketing of pharmaceutical products. The process of obtaining these approvals and the subsequent compliance with appropriate U.S. and foreign statutes and regulations are time-consuming and require the expenditure of substantial resources. In addition, these requirements and processes vary widely from country to country. The regulatory approval process is expensive and takes many years, and the timing or costs of any approval cannot be accurately predicted.

If the Company fails to obtain regulatory approval for current or future product candidates, it will be unable to market and sell such products and therefore may never be profitable. The FDA and other foreign regulatory agencies can delay, limit, or deny approval for many reasons, including if a product candidate may not be safe or effective; the manufacturing processes or facilities the Company selected may not meet the applicable requirements; and changes in the FDA's approval policies or adoption of new regulations may require additional work. Any delay in, or failure to receive or maintain, regulatory approval for any of MetaStat's products could prevent the Company from ever generating meaningful revenues or achieving profitability.

Even if the Company receives regulatory approvals, MetaStat's product candidates may later exhibit adverse effects that limit or prevent their widespread use or that force the Company to withdraw those candidates from the market. A marketed product continues to be subject to strict regulation after approval. Any unforeseen problems with an approved product or any violation of regulations could result in product restrictions, including MetaStat's withdrawal from the market. Any delay in, or failure to receive or maintain regulatory approval for, any products could prevent the Company from ever generating meaningful revenues or achieving profitability.

Healthcare policy changes, including recently enacted legislation reforming the U.S. healthcare system, may have a material adverse effect on MetaStat's financial condition and results of operations.

Changes in healthcare policy, such as the creation of broad test utilization limits for diagnostic products in general or requirements that Medicare patients pay for portions of clinical laboratory tests or services received, could substantially impact the sales of MetaStat's tests, decrease revenues, increase costs, and divert management's attention from MetaStat's business. Such impacts could derive from the Patient Protection and Affordable Care Act, as amended by the Health Care and Education Affordability Reconciliation Act, collectively, the PPACA, which makes changes that are expected to significantly impact the pharmaceutical and medical device industries and clinical laboratories; the Middle Class Tax Relief and Job Creation Act of 2012, which in part reduced the potential future cost-based increases to the Medicare Clinical Laboratory Fee Schedule by 2%; and ongoing discussions by the Centers for Medicare and Medicaid Services (CMS) regarding its payment policy for Multi-Analyte codes with Algorithmic Analyses (MAAAs). A greater explanation of legislative and policy initiatives that could impact MetaStat as it seeks to develop and commercialize its product candidates are provided in the Company's Risk Factors presented in its Form 10-K filed with the SEC on May 28, 2013.

The Company cannot predict whether future healthcare initiatives will be implemented at the federal or state level or in countries outside of the U.S. in which the Company may do business, or the effect any future legislation or regulation will have on the Company. The taxes imposed by new federal legislation, cost reduction measures, and the expansion in government's role in the U.S. healthcare industry may result in decreased profits to the Company, lower reimbursements by payors for MetaStat's products, or reduced medical procedure volumes, all of which may adversely affect MetaStat's business, financial condition, and results of operations. In addition, sales of MetaStat's tests outside of the U.S. make the Company subject to foreign regulatory requirements and cost-reduction measures, which may also change over time.

If the FDA were to begin regulating the MetaSite *Breast* test, the Company could experience delays in commercializing the test, be forced to stop sales, experience delays in commercializing future products, incur substantial costs and time delays associated with meeting requirements for pre-market clearance or approval, as well as experience decreased demand for its products and demand for reimbursement of its products.

Clinical laboratory tests like the MetaSite *Breast* test are regulated under the Clinical Laboratory Improvement Amendments of 1988 (CLIA), as administered through the CMS, as well as by applicable state laws. Diagnostic kits that are sold and distributed through interstate commerce are regulated as medical devices by the FDA. Clinical laboratory tests that are developed and validated by a laboratory for its own use are called laboratory development tests (LDTs). Most LDTs are not currently subject to FDA regulation, although reagents or software provided by third parties and used to perform LDTs may be subject to regulation.

The Company believes that the MetaSite *Breast* test is not a diagnostic kit and also believes that it is an LDT. As a result, the Company believes the MetaSite *Breast* test should not be subject to regulation under established FDA policies. The FDA may decide at any time at its sole discretion to modify these rules, or the U.S. Congress may enact new legislation, resulting in the need for the Company to conduct further trials in order to qualify the MetaSite *Breast* test for marketing approval. This may reduce or eliminate any potential revenue from sales of the MetaSite *Breast* test and may necessitate further round(s) of fundraising resulting in substantial dilution to investors.

Complying with numerous regulations pertaining to MetaStat's business is an expensive and time-consuming process, and any failure to comply could result in substantial penalties.

The Company is subject to CLIA, a federal law that regulates clinical laboratories that perform testing on specimens derived from humans for the purpose of providing information for the diagnosis, prevention, or treatment of disease. CLIA is intended to ensure the quality and reliability of clinical laboratories in the U.S. by mandating specific standards in the areas of personnel qualifications, administration, and participation in proficiency testing, patient test management, quality control, quality assurance, and inspections. The Company plans to obtain a certificate of accreditation under CLIA to perform testing. To renew the certificate of accreditation, the Company will be subject to survey and inspection every two years. Moreover, CLIA inspectors may make random inspections of MetaStat's laboratory. Currently, CLIA regulations do not include specific standards for a genetic specialty.

If the Company were to lose its CLIA accreditation or appropriate state license(s), whether due to revocation, suspension, or limitation, the Company would no longer be able to sell the MetaSite *Breast* test or other diagnostic tests, which would significantly harm business. If the Company were to lose its license in other states where it is required to hold licenses, it would not be able to test specimens from those states. The Company is subject to other regulations by both the federal government and states in which it conducts business, including the following:

- Medicare billing and payment regulations applicable to clinical laboratories;
- the federal Medicare and Medicaid Anti-kickback Law and state anti-kickback prohibitions;
- the federal physician self-referral prohibition, commonly known as the Stark Law, and the state equivalents;
- the federal Health Insurance Portability and Accountability Act of 1996;
- the Medicare civil money penalty and exclusion requirements; and
- the federal civil and criminal False Claims Act.

The Company has and will continue to adopt policies and procedures designed to comply with these laws, including policies and procedures relating to financial arrangements between the Company and physicians who refer it patients. In the ordinary course of business, the Company conducts internal reviews of its compliance with these laws. MetaStat's compliance is also subject to governmental review. The growth of MetaStat's business and sales organization may increase the potential of violating these laws or MetaStat's internal policies and procedures. The risk of being found in violation of these laws and regulations is further increased by the fact that many of them have not been fully interpreted by the regulatory authorities or the courts, and their provisions are open to a variety of interpretations. Any action brought against the Company for violation of these laws or regulations, even if the Company successfully defends against it, could create significant legal expenses and divert management's attention from the operation of its business. If MetaStat's operations are found to be in violation of any of these laws and regulations, the Company may be subject to any applicable penalty associated with the violation, including civil and criminal penalties, damages, and fines, and the Company could be required to refund payments received, and could be required to curtail or cease operations. Any of the foregoing consequences could seriously harm MetaStat's business and financial results.

Initially, MetaStat's financial results will depend on sales of one test, the MetaSite *Breast* test, and the Company will need to generate sufficient revenues from this and other diagnostics or therapies to run its business.

For the foreseeable future, the Company expects to derive substantially all of its revenues from sales of one test, the MetaSite *Breast* test. The Company anticipates commencing implementation of its sales and marketing strategy as early as 2015 in conjunction with the anticipated publication of the results of the large-population validation study. The Company is in various stages of R&D for other function-based diagnostic assays that it may offer as well as for enhancements to its existing test. The Company does not currently expect to commercialize these additional tests for additional cancer indications until at least 2015, and is not able to estimate when it may be able to commercialize therapeutics for cancer metastasis or whether it will be successful in doing so. Also, if the Company is unable to develop products to keep pace with rapid technological, medical, and scientific change, its operating results and competitive position would be harmed. MetaStat's tests could become obsolete unless the Company continually innovates and expands products to demonstrate recurrence and treatment benefit in patients treated with new therapies introduced to the oncology market. If the Company is unable to demonstrate the applicability of its test to new treatments, sales of MetaStat's test could decline, which would harm revenues.

If the Company is unable to increase sales of the MetaSite *Breast* test or to successfully develop and commercialize other competitive diagnostic tests, enhancements, or therapeutics, revenues and MetaStat's ability to achieve profitability would be impaired, and the market price of MetaStat's common stock could decline.

In addition, the Company may experience limits on revenues if physicians decide not to order its tests, or if patients decide not to use the tests.

If medical practitioners do not order the MetaSite *Breast* test or any future tests developed by the Company, the Company will likely not be able to create demand for its products in sufficient volume for it to become profitable. To generate demand, the Company will need to continue to make oncologists, surgeons, and pathologists aware of the benefits of the MetaSite *Breast* test and any products the Company may develop in the future through published papers, presentations at scientific conferences, and one-on-one education by MetaStat's sales force. Some physicians or patients may decide not to order MetaStat's test due to its price, part or all of which may be payable directly by the patient if the applicable payor denies reimbursement in full or in part. Even if patients recommend that their physicians use MetaStat's test, physicians may still decide not to use the MetaSite *Breast* test, either because they have not been made aware of its utility or they wish to pursue a particular course of therapy regardless of test results. Likewise, even if recommended by the practitioner, patients may decide not to use the test if they do not want to be made aware of the likelihood of metastasis or for other reasons. If only a small portion of the physician or patient population decides to use MetaStat's test, the Company will experience limits on revenues and its ability to achieve profitability. In addition, the Company will need to demonstrate its ability to obtain adequate reimbursement coverage from third-party payors.

If the Company becomes subject to product liability claims, damages may exceed insurance coverage levels.

The Company will obtain liability insurance for its product candidates as each is entered into large-population validation studies and/or any other studies where such liability insurance is needed. The Company cannot predict all of the possible harms or side effects that may result from the use of its products. Therefore, the amount of insurance coverage the Company currently holds, or that it or its collaborators may obtain, may not be adequate to protect from any claims arising from the use of MetaStat's products that are beyond the limit of MetaStat's insurance coverage. If the Company cannot protect against potential liability claims, the Company or its collaborators may find it difficult or impossible to commercialize MetaStat's products, and the Company may not be able to renew or increase MetaStat's insurance coverage on reasonable terms, if at all.

If MetaStat is unable to develop adequate sales, marketing, or distribution capabilities or enter into agreements with third parties to perform some of these functions, it will not be able to commercialize products effectively.

MetaStat may have a limited infrastructure in sales, marketing, and distribution. To directly market and distribute any products, the Company must effectively build a sales and marketing organization with appropriate technical expertise and distribution capabilities. The Company may not be able to establish such capabilities on its own or enter into such arrangements with third parties in a timely manner or on acceptable terms.

If the Company does not find development and commercialization collaborators for its product candidates, it may have to reduce or delay its rate of product development and commercialization and increase expenditures.

The Company may enter into relationships with selected biotechnology companies to help develop and commercialize MetaStat's product candidates, especially in the field of therapeutics. If the Company is not able to establish such collaborative arrangements, the Company may have to reduce or delay further development of some of its programs, increase planned expenditures, and undertake development and commercialization activities at its own expense. If the Company enters into development or commercialization collaborations with biotechnology companies, these relationships will be subject to a number of risks, including the following: (1) collaborators may not pursue further development and commercialization of products resulting from collaborations or may elect not to renew R&D programs; (2) collaborators may delay clinical trials, underfund a clinical trial program, stop a clinical trial or abandon a product candidate, repeat or conduct new clinical trials or require the development of a new formulation of a product candidate for clinical testing; (3) a collaborator with marketing and distribution rights to one or more of MetaStat's products may not commit enough resources to the marketing and distribution of such products, limiting MetaStat's potential revenues from the commercialization of these products; and (4) disputes may arise delaying or terminating the R&D or commercialization of MetaStat's product candidates, or may result in significant legal proceedings.

The Company relies on a limited number of suppliers or, in some cases, a sole supplier, for some of its laboratory instruments and materials and may not be able to find replacements in the event MetaStat's supplier no longer supplies that equipment.

The Company expects to rely on Leica Microsystems GmbH, a German company owned by Danaher Corp. (DHR-NYSE), to supply laboratory equipment on which the Company performs its tests. The Company will periodically forecast MetaStat's needs for laboratory equipment and enter into standard purchase orders or leasing arrangements with Leica based on these forecasts. The Company believes that there are relatively few equipment manufacturers other than Leica that are currently capable of supplying the equipment necessary for the MetaSite *Breast* test. Even if the Company were to identify other suppliers, there can be no assurance that the Company will be able to enter into agreements with such suppliers on a timely basis on acceptable terms, if at all. If the Company should encounter delays or difficulties in securing from Leica the quality and quantity of equipment required for the MetaSite *Breast* test, it may need to reconfigure MetaStat's test process, which would result in delays in commercialization or an interruption in sales. If any of these events occur, MetaStat's business and operating results could be harmed. Additionally, if Leica deems the Company to have become uncreditworthy, it has the right to require alternative payment terms, including payment in advance. The Company may also be required to indemnify Leica against any damages caused by any legal action or proceeding brought by a third party against Leica for damages caused by MetaStat's failure to obtain required approval with any regulatory agency.

The Company may also rely on several sole suppliers for laboratory materials such as reagents used to perform its tests. Although MetaStat believes that it will be able to develop alternate sourcing strategies for these materials, it cannot be certain that these strategies will be effective. If the Company should encounter delays or difficulties in securing these laboratory materials, delays in commercialization or an interruption in sales could occur.

MetaStat's success depends on retention of key personnel.

The Company is dependent on its management team members, including Dr. Oscar L. Bronsther, chief executive officer (CEO) and chief medical officer (CMO). MetaStat's future success will also depend in large part on its continued ability to attract and retain other highly qualified scientific, technical, and management personnel, as well as personnel with expertise in sales and marketing, clinical testing, and government regulation. The Company faces competition for personnel from other companies, universities, public and private research institutions, government entities, and other organizations. If the Company is unsuccessful in recruitment and retention efforts, its business will be harmed.

MetaStat's corporate compliance program cannot guarantee that the Company is in compliance with all potentially applicable regulations.

The development, manufacturing, pricing, sales, and reimbursement of MetaStat's products, together with MetaStat's general operations, are subject to extensive regulation by federal, state and other authorities within and outside of the U.S. While the Company has developed and instituted a corporate compliance program based on what it believes are the current best practices, the Company cannot assure anyone that it is or will be in compliance with all potentially applicable regulations. If the Company fails to comply with any of these regulations, it could be subject to a range of regulatory actions, including suspension or termination of clinical trials, failure to approve a product candidate, restrictions on products or manufacturing processes, withdrawal of products from the market, significant fines, or other sanctions or litigation.

MetaStat's operations may involve hazardous materials, and compliance with environmental laws and regulations is expensive.

MetaStat's future R&D activities may involve the controlled use of hazardous materials, including chemicals that cause cancer, volatile solvents, radioactive materials, and biological materials including human tissue samples that have the potential to transmit diseases. MetaStat's operations may also produce hazardous waste products. The Company is subject to a variety of federal, state, and local regulations relating to the use, handling, and disposal of these materials. The Company generally may contract with third parties for the disposal of such substances and may store certain low-level radioactive waste at MetaStat's facility until the materials are no longer considered radioactive. While the Company believes that it will comply with then current regulatory requirements, the Company cannot eliminate the risk of accidental contamination or injury from these materials. If the Company uses biological and hazardous materials in a manner that causes injury, it could be liable for damages. The Company may be required to incur substantial costs to comply with current or future environmental and safety regulations. If an accident or contamination occurred, MetaStat would likely incur significant costs associated with civil penalties or criminal fines and in complying with environmental laws and regulations.

RISKS RELATED TO INTELLECTUAL PROPERTY

If the Company is unable to protect intellectual property, it may not be able to compete effectively.

MetaStat's success will depend in part on its ability to obtain or license patents and enforce patent protection of its products and licensed technologies, as well as the ability of the Licensors to enforce patent protection covering the patents which the Company licenses pursuant to the License Agreement, Second License Agreement, and Third License Agreement both in the U.S. and other countries to prevent MetaStat's competitors from developing, manufacturing, and marketing products based on MetaStat's technology. The patent positions of biotechnology companies, such as MetaStat, are generally uncertain and involve complex legal and factual questions. The Company will be able to protect its licensed intellectual property rights from unauthorized use by third parties only to the extent that its licensed technologies are covered by any valid and enforceable patents or are effectively maintained as trade secrets. The Company could incur substantial costs in seeking enforcement of any eventual patent rights against infringement, and cannot guarantee that patents that it obtains or in-licenses will successfully preclude others from using technology that the Company relies upon. The Company has applied and intends to apply for patents in the U.S. and other countries as and when it deems appropriate. However, these applications may be challenged or may fail to result in issued patents.

The Company cannot predict the breadth of claims that may be allowed and issued in patents related to biotechnology applications. The laws of some foreign countries do not protect intellectual property rights to the same extent as the laws of the U.S., and many companies have encountered significant problems in protecting and defending such rights in foreign jurisdictions. For example, methods of treating humans are not patentable in many countries outside of the U.S.

The Company does not know whether any of the pending or future patent applications will result in the issuance of patents. Any patents the Company or the Licensors obtain may not be sufficiently broad to prevent others from using MetaStat's technologies or from developing competing therapeutic products based on MetaStat's technology or proprietary therapies. Once any such patents have issued, the Company cannot predict how the claims will be construed or enforced. To the extent patents may be issued, the Company does not know whether these patents will be subject to further proceedings that may limit their scope, provide significant proprietary protection or competitive advantage, or cause them to be circumvented or invalidated. Furthermore, patents that may issue on MetaStat's or the Licensors pending applications may become subject to dispute, including interference, reissue, or reexamination proceedings in the U.S., or opposition proceedings in foreign countries. Any of these proceedings could result in the limitation or loss of rights.

The Company may rely on trade secret protection for confidential and proprietary information, and has taken measures to protect its proprietary information and trade secrets though these measures may not provide adequate protection. While the Company seeks to protect proprietary information by entering into confidentiality agreements with employees, collaborators, and consultants, it cannot ensure that proprietary information will not be disclosed, or that it can meaningfully protect trade secrets. In addition, competitors may independently develop or may have already developed substantially equivalent proprietary information or may otherwise gain access to MetaStat's trade secrets.

Litigation or third-party claims of intellectual property infringement could impair MetaStat's ability to develop and commercialize products successfully.

MetaStat's success will depend in part on its ability to avoid infringing patents and proprietary rights of third parties, and not breaching any licenses that it has entered into with regard to its technologies. A number of pharmaceutical companies, biotechnology companies, independent researchers, universities, and research institutions may have filed patent applications or may have been granted patents that cover technologies similar to the technologies owned by or licensed to the Company. For instance, a number of patents may have issued and may issue in the future on tests and technologies that the Company has developed or intends to develop. If patents covering technologies required by MetaStat's operations are issued to others, the Company may have to rely on licenses from third parties, which may not be available on commercially reasonable terms, or at all.

In addition, if a patent holder believes that one of MetaStat's product candidates infringes on its patent, it may sue the Company even if the Company has received patent protection for its technology. Third parties may claim that the Company is employing proprietary technology without authorization. In addition, third parties may obtain patents that relate to MetaStat's technologies and claim that use of such technologies infringes these patents. Regardless of their merit, such claims could require the Company to incur substantial costs, including the diversion of management and technical personnel, in defending against any such claims or enforcing patents. In the event that a successful claim of infringement is brought against the Company, the Company may be required to pay damages and obtain one or more licenses from third parties. The Company may not be able to obtain these licenses at a reasonable cost, or at all. Defense of any lawsuit or failure to obtain any of these licenses could adversely affect MetaStat's ability to develop and commercialize MetaStat's products.

MetaStat's rights to use technologies licensed from third parties are not within its control, and the Company may not be able to sell its products if it loses its existing rights or cannot obtain new rights on reasonable terms.

For example, the Company licenses technology from MIT, Einstein, Cornell, and IFO-Regina (Rome, Italy) used to analyze tissue samples in its tests, in sponsored research to develop additional tests, and to develop anti-metastasis therapeutics. In return for the use of a third party's technology, the Company has agreed to pay the licensors royalties based on sales of its products. The Company may need to license other technology to commercialize future products. MetaStat's business may suffer if these licenses terminate, if the licensors fail to abide by the terms of the license or fail to prevent infringement by third parties, if the licensed patents or other rights are found to be invalid, or if the Company is unable to enter into necessary licenses on acceptable terms.

RISKS RELATED TO METASTAT'S SECURITIES

Insiders have substantial control over the Company, and they could delay or prevent a change in corporate control even if MetaStat's other stockholders wanted it to occur.

MetaStat's executive officers, directors, and principal stockholders hold approximately a large majority of MetaStat's outstanding common stock. Accordingly, these stockholders are able to control all matters requiring stockholder approval, including the election of directors and approval of significant corporate transactions. This could delay or prevent an outside party from acquiring or merging with the Company even if MetaStat's other stockholders wanted it to occur.

The Company cannot assure anyone that the common stock will become liquid, that it will be listed on a securities exchange, or that it will not be subject to continued volatility. In addition, there may not be sufficient liquidity in the market for MetaStat's securities in order for investors to sell their securities.

Currently, the Company is quoted on an OTC exchange, where an investor may find it difficult to obtain accurate quotations as to the market value of MetaStat's common stock. In addition, the market price of MetaStat's common stock has been and will likely continue to be highly volatile, as is the stock market in general, and the market for OTC quoted stocks in particular. Further, if the Company fails to meet the criteria set forth in SEC regulations, by law, various requirements would be imposed on broker-dealers who sell its securities to persons other than established customers and accredited investors. Consequently, such regulations may deter broker-dealers from recommending or selling MetaStat's common stock, which may further affect its liquidity. In addition, there is currently only a limited public market for MetaStat's common stock and there can be no assurance that a trading market will develop further or be maintained in the future.

In order to raise sufficient funds to expand operations, the Company may have to issue additional securities at prices, which may result in substantial dilution to MetaStat's shareholders.

If the Company raises additional funds through the sale of equity or convertible debt, MetaStat's current stockholders' percentage ownership will be reduced. In addition, these transactions may dilute the value of MetaStat's outstanding securities. The Company may have to issue securities that may have rights, preferences, and privileges senior to MetaStat's common stock. The Company cannot provide assurance that it will be able to raise additional funds on terms acceptable to the Company, if at all. If future financing is not available or is not available on acceptable terms, the Company may not be able to fund future needs, which would have a material adverse effect on business plans, prospects, results of operations, and financial condition.

If the Company fails to maintain an effective system of internal control over financial reporting, it may not be able to accurately report financial results. As a result, current and potential investors could lose confidence in MetaStat's financial reporting, which could harm business and have an adverse effect on stock price.

Pursuant to Section 404 of the Sarbanes-Oxley Act of 2002, the Company is required to annually furnish a report by MetaStat's management on its internal control over financial reporting. Such report must contain, among other matters, an assessment by MetaStat's principal executive officer and principal financial officer on the effectiveness of MetaStat's internal control over financial reporting, including a statement as to whether or not internal control over financial reporting is effective as of the end of the fiscal year. This assessment must include disclosure of any material weakness in MetaStat's internal control over financial reporting identified by management. In addition, under current SEC rules, the Company may be required to obtain an attestation from its independent registered public accounting firm as to its internal control over financial reporting for its annual report on Form 10-K covering MetaStat's next fiscal year. Performing the system and process documentation and evaluation needed to comply with Section 404 is both costly and challenging. During the course of MetaStat's testing, the Company may identify deficiencies which it may not be able to remediate in time to meet the deadline imposed by the Sarbanes-Oxley Act of 2002 for compliance with the requirements of Section 404. In addition, if the Company fails to maintain the adequacy of its internal controls, as such standards are modified, supplemented, or amended from time to time, it may not be able to ensure that it can conclude on an ongoing basis that it has effective internal controls over financial reporting in accordance with Section 404 of the Sarbanes-Oxley Act of 2002. Failure to achieve and maintain an effective internal control environment could cause investors to lose confidence in the Company's reported financial information, which could have a material adverse effect on the price of its common stock.

MetaStat's common stock is considered "penny stock."

The SEC has adopted regulations, which generally define penny stock to be an equity security that has a market price of less than $5.00 per share, subject to specific exemptions. The market price of MetaStat's common stock is currently less than $5.00 per share and therefore may be a penny stock. Brokers and dealers effecting transactions in penny stock must disclose certain information concerning the transaction, obtain a written agreement from the purchaser, and determine that the purchaser is reasonably suitable to purchase the securities. These rules may restrict the ability of brokers or dealers to sell the common stock and may affect investors' ability to sell shares.

In addition, the market for penny stocks has experienced numerous frauds and abuses, which could adversely impact investors in MetaStat's stock. OTC securities are frequent targets of fraud or market manipulation, both because of their generally low prices and because OTC reporting requirements are less stringent than those of the stock exchanges or NASDAQ.

Appendix

Figure 38

SCIENTIFIC PUBLICATIONS SUPPORTING METASTAT'S TECHNOLOGY PLATFORM

Core Technology Publications	Article Title Date - *Journal Title* - Author(s)

A Mena Invasion Isoform Potentiates EGF-Induced Carcinoma Cell Invasion and Metastasis
2008 - *Developmental Cell* - Philippar et al.

Tumor Microenvironment of Metastasis in Human Breast Carcinoma: A Potential Prognostic Marker Linked to Hematogenous Dissemination
2009 - *Clinical Cancer Research* - Robinson et al.

Mena deficiency delays tumor progression and decreases metastasis in polyoma middle-T transgenic mouse mammary tumors
2010 - *Breast Cancer Research* - Roussos et al.

Quantitative assessment of invasive mena isoforms (MenaCalc) as an independent prognostic marker in breast cancer
2012 - *Breast Cancer Research* - Argawal et al.

Selective gene-expression profiling of migratory tumor cells in vivo predicts clinical outcome in breast cancer patients
2012 - *Breast Cancer Research* - Patsialou et al.

An EMT-Driven Alternative Splicing Program Occurs in Human Breast Cancer and Modulates Cellular Phenotype
2011 - *Plos Genetics* - Shapiro et al.

Supporting Publications from Team Members

Gene expression analysis on small numbers of invasive cells collected by chemotaxis from primary mammary tumors of the mouse
2003 - *BMC Biotechnology* - Wang et al.

Intravital Imaging of Cell Movement in Tumors
2003 - *Nature Reviews Cancer* - Condeelis & Segall

Identification and Testing of a Gene Expression Signature of Invasive Carcinoma Cells within Primary Mammary Tumors
2004 - *Cancer Research* - Wang et al.

A Paracrine Loop between Tumor Cells and Macrophages Is Required for Tumor Cell Migration in Mammary Tumors
2004 - *Cancer Research* - Wychoff et al.

Human Mena Protein, a Serex Defined Antigen Overexpressed in Breast Cancer Eliciting Both Humoral and CD8 T-Cell Immune Response
2004 - *International Journal of Cancer* - Di Modungo et al.

The Great Escape: When Cancer Cells Hijack the Genes for Chemotaxis and Motility
2005 - *Annu Rev Cell Dev Biol* - Condeelis, Singer & Segall

Tumor cells caught in the act of invading: their strategy for enhanced cell motility
2005 - *Trends in Cell Biology* - Wang et al.

Macrophages: Obligate Partners for Tumor Cell Migration, Invasion, and Metastasis
2006 - *Cell* - Condeelis & Pollard

Molecular Cloning of hMena (ENAH) and Its Splice Variant hMena+11a: Epidermal Growth Factor Increases Their Expression and Stimulates hMena+11a Phosphorylation in Breast Cancer Cell Lines
2007 - *Cancer Research* - Di Modungo et al.

Coordinated Regulation of Pathways for Enhanced Cell Motility and Chemotaxis Is Conserved in Rat and Mouse Mammary Tumors
2007 - *Cancer Research* - Wang et al.

Direct Visualization of Macrophage-Assisted Tumor Cell Intravasation in Mammary Tumors
2007 - *Cancer Research* - Wychoff et al.

Intravital imaging of metastatic behavior through a mammary imaging window
2008 - *Nature Methods* - Kedrin et al.

Identification of invasion specific splice variants of the cytoskeletal protein Mena in mammary tumor cells during invasion in vivo
2009 - *Clinical Experimental Metastasis* - Goswami et al.

In Vivo Assay for Tumor Cell Invasion
2009 - *Methods in Molecular Biology* - Hernandez et al.

Mena invasive (MenaINV) and Mena11a isoforms play distinct roles in breast cancer cell cohesion and association with TMEM
2011 - *Clinical Experimental Metastasis* - Roussos et al.

Mena invasive (MenaINV) promotes multicellular streaming motility and transendothelial migration in a mouse model of breast cancer
2011 - *Journal of Cell Science* - Roussos et al.

Metastasis: tumor cells becoming MENAcing
2011 - *Trends in Cell Biology* - Condeelis & Gertler

Correlated Immunohistochemical and Cytological Assays for the Prediction of Hematogenous Dissemination of Breast Cancer
2012 - *Journal of Histochemistry & Cytochemistry* - Oktay et al.

Source: MetaStat, Inc.

Glossary

Actin—An important structural molecule for the cytoskeletons of many eukaryotic cells.

Adenocarcinoma—A malignant tumor formed from glandular structures in epithelial tissue.

Alternatively Spliced mRNA—A process by which multiple forms of messenger RNA (mRNA) and protein isoforms are generated from the same gene. Alternative splicing is a regulatory mechanism by which variations in the incorporation of the exons, or coding regions, into mRNA leads to the production of more than one related protein, or isoform. As many genes associated with cancer go through alternative splicing (to produce "alternatively spliced isoforms"), researchers theorize that this (alternative splicing) may be a pathway to regulate cancer onset and progression (Source: *PLoS Genetics*' "An EMT–Driven Alternative Splicing Program Occurs in Human Breast Cancer and Modulates Cellular Phenotype," August 2011).

Aptamers—Small single-stranded nucleic acids that fold into a well-defined three-dimensional structure.

Assay—Analysis to determine the presence, absence, or quantity of one or more components; Also, a test used in this analysis.

Chemoattractant—A chemotactic agent that induces an organism or a cell to migrate toward it.

Chemotactic—Pertaining to a tendency of cells to migrate toward or away from certain chemical stimuli.

Chemotherapy—Medications given to kill or slow the growth of cancer cells. Chemotherapy is often used with surgery or radiation to treat cancer when the cancer has spread, when it has come back (recurred), or when there is a strong chance that it could recur.

Ductal Carcinoma—The most common type of breast cancer. It begins in the cells that line the milk ducts in the breast.

Epidermal Growth Factor (EGF)—A protein that stimulates normal cell growth, cancerous cell growth, and wound healing. Significant elevations of EGF have been implicated in the development and progression of solid tumors, including those of the lung, breast, prostate, colon, ovary, head, and neck.

Epithelial—Part of the epithelium, a thin layer of tissue that covers organs, glands, and other structures within the body.

Exon—A segment of a DNA or RNA molecule containing information coding for a protein or peptide sequence.

Extracellular Matrix—A term used to describe the surrounding substance or environment of a cell.

Fine Needle Aspiration—The use of a thin needle to withdraw material from the body for analysis. The aspirated material is examined under the microscope by a pathologist.

Hematogenous—Concerned with the production of blood or of one or more of its constituents, or taking place or spreading by way of the blood.

HER2-negative—Cancer that is negative for the HER2 protein, which is a protein that indicates aggressive cancer.

HER2-positive—Breast cancer that tests positive for a protein called human epidermal growth factor receptor 2 (HER2), which promotes the growth of cancer cells. HER2-positive breast cancers tend to be more aggressive than other types of breast cancer.

Immunostaining—The staining of a specific substance by using an antibody against it that is complexed with a staining medium.

Invadopodia—Protrusions in the cell membrane of some cells that are rich in actin and extend into the extracellular matrix.

Isoform—Any of several different forms of the same protein.

Lymphovascular Invasion—The invasion of cancer cells into blood vessels and/or the lymphatic system.

Macrophage—A white blood cell that helps the body defend itself against disease by surrounding and destroying foreign organisms.

Mechanisms—The natural or established processes by which cancer develops.

Mena—A protein that has a role in regulating cell movement, shape, and adhesion. The Mena protein is found in excessive amounts in tumors and is known to help cancer cells move away from a tumor and spread around the body to form secondary cancers—one of the main obstacles in treating cancer.

Metastasis—The spread of cancer to another part of the body, where it can form a secondary tumor.

Metastatic—Cancer that has spread to other parts of the body from the original tumor site.

Motility—The ability to move spontaneously and independently.

Oncogene—A gene that in certain circumstances can transform a cell into a tumor cell.

Perivascular—Situated or occurring around a blood vessel.

Personalized Cancer Therapies—Treatments tailored to a patient's specific needs based on genetic abnormalities found in the individual's tumor.

Polyoma Middle T (PyMT)—A mouse line that has been engineered to be genetically predisposed to highly metastatic breast cancer.

Prognostic—Predicting the likely outcome of a disease; of or relating to a prognosis.

Protrusion—A bulge or projection from an object.

Radiation—A cancer treatment in which radiation energy is focused onto a specific area of the body to eradicate cancer cells and shrink tumors.

RNA—Ribonucleic acid, a nucleic acid present in all living cells. Its principal role is to act as a messenger carrying instructions from DNA for controlling the synthesis of proteins.

Sample—Used to isolate and process DNA, RNA, and proteins from biological samples, such as blood or tissue.

Systemic—A disease or symptom that affects many different parts of the body.

Triple-negative Cancer (TNC)—The absence of staining for estrogen receptor, progesterone receptor, and HER2. TNC is insensitive to some of the most effective therapies available for breast cancer treatment, including HER2-directed therapy, such as trastuzumab, and endocrine therapies, such as tamoxifen or the aromatase inhibitors.

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