Please click here and advance to page 10 to read about nimotuzumab

YM BIOSCIENCES, INC. (NYSE AMEX: YMI)

"JAK – IN-THE-BOX!"

YM BioSciences, Inc. (NYSE AMEX: YMI, TSX: YM) is a life-sciences product development company. YMI's most advanced product in development is nimotuzumab, an EGFR-targeting monoclonal antibody for which YMI holds the license for most major international pharmaceutical markets. It is currently in numerous trials globally for the treatment of glioma, head and neck, gastric, cervical, and non-small-cell lung (NSCLC) cancers. The product is approved for marketing in 25 secondary market countries by developers unrelated financially to YMI. CYT387 is a potent, selective, oral JAK1/JAK2 inhibitor designed to suppress the over activity of the tyrosine kinases, including a mutant form of JAK2 enzyme, JAK2V617F. Clinical development of CYT387 is underway in the U.S. initially in patients with myelofibrosis, a type of myeloproliferative neoplasm (MPN). CYT997, YMI's novel, oral, vascular disrupting agent (VDA), is also in the clinic in a trial in glioma in Australia and the UK.

Share Price (6/22/10)	$1.17
52-Week Price Low / High	$0.48 – $2.24
Mkt. Capitalization (issued)	$88.3 MM
Shares Outstanding (issued)	75.47 MM
12-month Target Price	$5.50
Cash & Equiv. (3/31/10)	$46.7 MM
Fiscal Year Ends	June 30th
Website	ymbiosciences.com

All currency figures in USD$, unless otherwise noted.

12-Month Price Chart

Source: BigCharts.com

Selected nimotuzumab late stage trials:

- Phase III Trial (Singapore & worldwide) (National Cancer Center of Singapore): Adjuvant Head & Neck Cancer
- Phase III Trial (Western Europe) (Oncoscience AG): First-Line Pediatric Glioma
- Phase III Trial (Western Europe) (Oncoscience AG): First-Line Adult Glioma
- Phase II Trial (U.S./Canada) (YMI): Diffuse Intrinsic Pontine Glioma
- Phase II/III (Western Europe) (Oncoscience AG): Pancreatic Frontline
- Phase II Trial (Japan) (Daiichi-Sankyo): First-Line NSCLC
- Phase II Trial (Japan) (Daiichi-Sankyo/Kuhnil Pharma): Advanced/Recurrent Gastric Cancer
- Phase II Trial (U.S. & Canada) (YMI): Palliative NSCLC
- Phase II Trial (U.S. & Canada) (YMI): Brain Metastases from NSCLC
- Phase II Trial (Singapore) (Innogene Kalbiotech/Kalbe Farma): Cervical Cancer
- Phase II Trial (Singapore) (Innogene Kalbiotech/Kalbe Farma): Locally-Advanced Head & Neck Cancer

CYT387 JAK Program

- Phase I/II Trial (US) (YMI) led by Tefferi at Mayo Clinic in patients with myelofibrosis, a type of myeloproliferative neoplasms (MPN).

CYT997 VDA Program

- Phase I/II clinical trial in combination with chemotherapy in patients with relapsed glioblastoma multiforme.

We are reiterating our BUY rating on YM BioSciences, Inc. (NYSE AMEX: YMI, TSX: YM) and our 12-month price target of $5.50 for YMI shares.

- **YMI's JAK1/2 inhibitor, CYT387: Looks like a winner.** YMI's JAK1/2 clinical program led by Dr. Ayalew Tefferi, a Key Opinion Leader in the field of JAK, at the Mayo Clinic. The selectivity of CYT387 as compared to **Incyte Corp.'s (NasdaqGM: INCY)** JAK1/2 inhibitor INCB18424 may result in a superior therapeutic window and broader applicability for CYT387 in benign indications. Given that Incyte licensed ex-US rights to INCB18424 to **Novartis AG (NYSE: NVS)** recently for $150 million up-front with a total potential deal size of $1 billon and INCB28050 to **Eli Lilly & Co. (NYSE: LLY)** for an upfront of $90 million and prospective milestones of $665 million for inflammatory diseases, YMI's JAK franchise could be a tremendous value driver as it progresses through its clinical trials. In addition to a Phase I/II trial in patients with myelofibrosis, a type of myeloproliferative neoplasm (MPN), preclinical work on CYT387 is also currently underway in indications other than MPNs, including liquid and solid tumors, inflammatory diseases and transplantation.

- **Nimotuzumab data expected from Daiichi-Sankyo Phase II trials in gastric cancer and NSCLC in Q4 CY'10 is expected to set the stage for pivotal Phase III trials.**

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HIGHLIGHTS

- **YMI'S JAK1/2 CLINICAL PROGRAM, ORIGINALLY PLANNED AS A SEQUENTIAL PHASE I/II, WAS ADVANCED AHEAD OF SCHEDULE IN MAY 2010 IN ORDER TO TARGET PHASE II COMPLETION IN THIS CURRENT CALENDAR YEAR.** Abnormal JAK1/2 activity is observed in several indications, including myeloproliferative neoplasms (MPNs), inflammatory conditions, and cancer indications, which points to a broad applicability of CYT387. YMI's compound is differentiated from other JAK1/2 inhibitors in development based on its superior selectivity for JAK1/2 enzymes. On April 21, 2010, YM BioSciences, Inc. announced data for CYT387 in the premier hematology journal *Blood* that indicated that YM's JAK1/2 has an exceptional profile.[1] The paper discusses work conducted in the laboratory of Dr. Michael Deininger at Oregon Health Sciences University Knight Cancer Institute, Portland, Oregon, which demonstrated that orally-administered CYT387 normalizes the common MPN features of elevated blood cell counts and enlarged spleen size in an *in vivo* model of the disease (see diagrams on page 6 of this report). Clinical development of CYT387 is underway in the U.S. initially in patients with myelofibrosis, a disease that affects approximately 20,000 patients in North America with no approved treatments with market estimates in excess of $750 million. Preliminary efficacy data from the Phase II portion of the trial may be available in late 2010, instead of 2H CY2011 as previously announced. Rapid progression into a NDA registration trial in myelofibrosis is expected by the 2H CY2011.

- **GLOBAL PARTNER DAIICHI-SANKYO EXPECTED TO RELEASE NIMOTUZUMAB PHASE II DATA IN GASTRIC CANCER AND NSCLC.** Data from a randomized Phase II gastric cancer trial is expected in Q3 CY2010, and data from the NSCLC trial is expected in Q4 CY2010. The gastric cancer trial has completed enrollment of 80 patients with advanced/recurrent disease. The primary endpoint of the trial is progression-free survival (PFS). The NSCLC trial is expected to enroll approximately 39 patients and has a primary endpoint of completion of treatment by patients being treated with concurrent chemoradiation plus nimotuzumab. The data from both of these trials will determine plans for registration Phase III trials in both indications with Phase III decisions expected around the end of CY2010.

- **DATA AT ASCO SHOW PROMISE OF NIMOTUZUMAB FOR LIFE THREATENING DISEASES LIKE HEAD AND NECK CANCER AND GLIOMA.** Posters and an abstract featuring nimotuzumab were presented during the 46th meeting of the Annual Society of Clinical Oncology (ASCO), June 4 to 8, 2010 in Chicago. The topics presented at the ASCO 2010 Annual Meeting included: (i) a summary of four-year survival results in head & neck cancer, (ii) results from a pilot study using nimotuzumab in combination with chemoradiation in head and neck cancer, and (iii) a summary of a study combining nimotuzumab with temozolomide and radiotherapy in patients with glioma. Notably, 4-year long-term survival data from a randomized Phase IIb study of nimotuzumab in squamous cell carcinoma of head and neck cancer (SCCHN) in a poster entitled "An open-label, randomized, study of h-R3mAb (nimotuzumab) in patients with advanced (stage III or IVa) squamous cell carcinoma of head and neck (SCCHN)" further demonstrated the clinical benefit with survival advantage for patients when combined with radiotherapy or chemoradiotherapy. These results compare favorably to the benefit shown in previous studies with other EGFR inhibitors in this indication, but the poster reports that nimotuzumab continues to demonstrate efficacy without any of the elevated toxicities common with the other agents. Data from YMI's licensee in Europe (Oncoscience AG) from a Phase III trial in adult glioma that had been anticipated to be presented at ASCO 2010 was unavailable. YMI was advised that, with last patient enrolled in March 2010, data collection and analysis had not been able to be completed in time.

[1] Jeffrey W. Tyner, Michael W. Deininger et al.; "CYT387, a novel JAK2 inhibitor, induces hematologic responses and normalizes inflammatory cytokines in murine myeloproliferative neoplasms"; Blood, First Edition, 2010.

- **YMI COMMENCES US PHASE II TRIALS FOR NIMOTUZUMAB IN NON-SMALL-CELL LUNG CANCER PATIENTS.** On May 27, 2010, YMI announced the enrollment of its first U.S. patient for two ongoing Phase II clinical trials in patients receiving palliative treatment for non-small-cell lung cancer (NSCLC) and patients with brain metastases from NSCLC.[2] The trials are expected to enroll 128 and 88 patients, respectively, at clinical sites in the U.S., Canada, Europe, Korea, Singapore, and India. YMI expects overall global enrollment to be nearing completion by the end of CY2010. YMI first received approval in 2008 to use nimotuzumab but only in a Phase II trial in the U.S. in children with inoperable, recurrent brain cancer. This limitation was listed in 2009 so that YMI is now free to use nimotuzumab in any clinical setting and this new expanded Phase II clearance in NSCLC is a very important first step toward finding a viable treatment and extending the life of patients with this devastating disease, as well as for introducing nimotuzumab to U.S. oncologists and eventually making it commercially available in the U.S.
- **WE REITERATE OUR BUY RATING ON YMI SHARES AND OUR 12-MONTH PRICE TARGET OF $5.50 FOR YMI SHARES.** Our price target for YMI shares is $5.50/share based on our DCF valuation model. We project royalties from sales of nimotuzumab, sales of CYT387 in myeloproliferative disorders (MPDs), and sales of CYT997 in glioblastoma multiforme. Given that nimotuzumab will prospectively be an important competitor to both Erbitux® and Vectibix®, CYT387 and YMI's JAK program is growing in attractiveness, and CYT997, which by being orally available, holds the promise of being able to maintain anti-vascular activity by continuous low-dosing, we belive YMI shares hold upside potential as each program advances. Firming up the balance sheet, equity financings completed in March and June raised a total of USD$20.7 million, bringing cash & equivalents to approximately USD$49.9 million.

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[2] YM Biosciences Inc. press release, "YM BioSciences announces FDA clearance for two ongoing Phase II nimotuzumab trials into USA." January 26, 2010.

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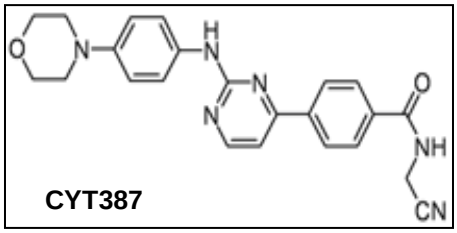
CYT387 OVERVIEW

CYT387 is a potent, selective, oral JAK1/JAK2 inhibitor designed to suppress the over-activity of the tyrosine kinases, including a mutant form of JAK2 enzyme, JAK2V617F. Abnormal JAK1/2 activity is observed in several indications, including myeloproliferative neoplasms (MPNs), inflammatory conditions, and cancer indications, which points to a broad applicability of CYT387. The compound is differentiated from other JAK1/2 inhibitors in development based on its superior selectivity for JAK1/2 enzymes. Clinical development of CYT387 is underway in the U.S. initially in patients with myelofibrosis, a type of MPN.



CYT387

The CYT387 Phase I/II study commenced in 2009 and is led by Dr. Ayalew Tefferi, Professor of Medicine at the Mayo Clinic and a Key Opinion Leader in the field of JAK1/2 inhibitors. Preliminary data from the Phase I portion of the study is expected in middle of this year. The Phase II component of the study was initiated sooner than expected due to promising safety and activity observations, and interim efficacy results from the study are expected to be presented at the annual meeting of the American Society of Hematology in December 2010.

CYT387, YMI's novel and highly selective oral JAK1/2 inhibitor, was discovered under the leadership of Dr. Andrew Wilks, who is credited with the seminal discovery of the JAK1 and JAK2 kinases. Under normal circumstances, the activation of JAK2 stimulates blood cell production. Genetic mutations in the JAK2 enzyme result in up-regulated activity and are implicated in MPNs, a family of conditions characterized by abnormal production of blood cells in the bone marrow. JAK1 is an important regulator of inflammation and becomes overactive in MPNs as well as inflammatory and cancer conditions. Consequently, CYT387, which potently and selectively blocks JAK1/2 signaling pathways, has potential utility in the treatment of MPNs, solid and liquid tumors, rheumatoid arthritis, and psoriasis.

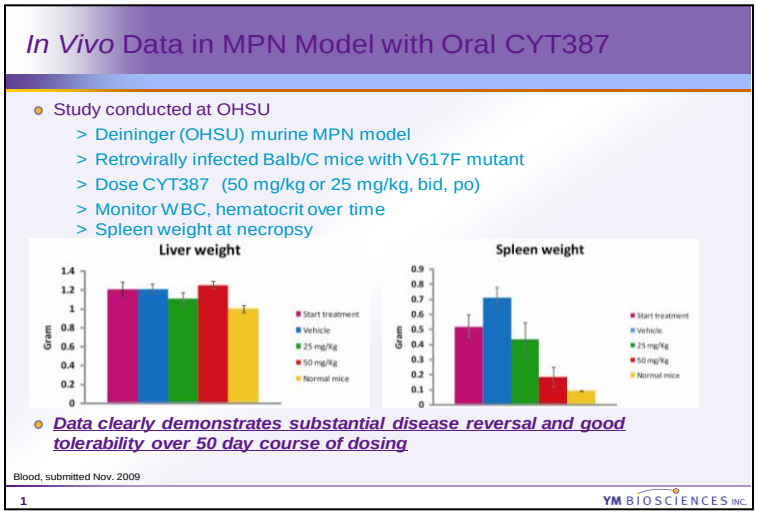
JAK2 PROGRAM: CYT387 PROFILE	
Description:	• Synthetic, small molecule • Novel: described in international (PCT) patent application
Kinase Profile:	• Potent JAK2 Inhibitor • ATP competitive: equi-active against JAK2 (V617F) • Equipotent JAK1; >10X selectivity over JAK3, TYK2 • Minimal off-target activity in kinase panels (>150 screened)
Cellular Activity:	• Potently blocks JAK2 activity in cells • Blocks mutant activity in MPN patient-derived cells • Limited cytotoxicity
ADMET:	• Orally active, half life indicates once-a-day dosing • Favorable *in vitro* safety profile • Predicted human oral activity • "Clean" across broad counterscreen panel

Source: Cytopia, Ltd. Corporate Presentation October 2009

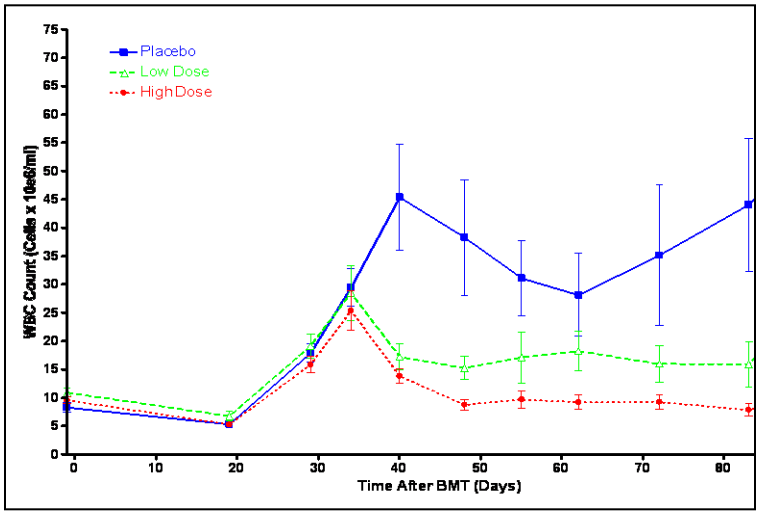
In preclinical profiling, CYT387 demonstrates excellent selectivity and safety profile with minimal off-target activities as well as favorable pharmacokinetic properties. Preliminary data using samples derived from MPN patients have shown *activity in suppressing* the over-activity caused by the JAK2V617F mutant enzyme.

On April 21, 2010, YM BioSciences Inc. announced the pre-publication of data for CYT387 in the hematology journal *Blood* that indicated that YM's JAK1/2 has an exceptional profile.[3] The paper discusses work conducted in the laboratory of Dr. Michael Deininger at Oregon Health Sciences University Knight Cancer Institute, Portland, Oregon, which demonstrated that orally-administered CYT387 normalizes the common MPN features of elevated blood cell counts and enlarged spleen size (depicted below) in an *in vivo* model of the disease.

CYT387 Normalizes Spleen Weight and White Blood Cell Count in MPN Patients



Notably, In the figure displayed to the left, spleen weight (a common characteristic of MPNs) is normalized with the treatment of CYT387. *Blood, Tyner and Deininger, April 2010.*



In the figure to the left, rapid white blood cell (WBC) count normalization is seen for both doses of CYT387 (low=25mg/kg bid po and high = 50 mg/kg bid po). *Blood, Tyner and Deininger, April 2010.*

Source: YM BioSciences, Inc.

The data also indicated that CYT387 significantly reduces circulating levels of inflammatory cytokines, such as IL-6 and TNF-alpha, which are common in patients with MPNs. Importantly, blood cell production was shown to return to the bone marrow with drug treatment.

In separate work, CYT387 was demonstrated to have improved selectivity over JAK3 and TYK2 compared to **Incyte Corp.'s (NasdaqGM: INCY)** JAK1/2 inhibitor, INCB18424. Incyte licensed ex-US rights to INCB18424 to **Novartis AG (NYSE: NVS)** for $150 million up front and a total potential deal size of $1 billon. The improved selectivity of CYT387 as compared to INCB18424 may result in a superior therapeutic window for CYT387[4] and broader applicability for CYT387 in benign indications.

[3] Jeffrey W. Tyner, Michael W. Deininger et al.; "CYT387, a novel JAK2 inhibitor, induces hematologic responses and normalizes inflammatory cytokines in murine myeloproliferative neoplasms"; Blood, First Edition, 2010.
[4] YM Biosciences, Inc. Press Release, "YM Biosciences announces pivotal preclinical efficacy data for the JAK1/2 inhibitor CYT387 published in Blood, the Journal of the American Society of Hematology". April 29, 2010.

CYT387 Current Phase I/II Clinical Trial

A Phase I/II myelofibrosis (MF) study commenced in 2009, led by Dr. Ayalew Tefferi, a world expert in JAK1/2 inhibitors, at the Mayo Clinic. MF is a serious neoplastic condition characterized by varying degrees of bone marrow failure, splenic enlargement, and debilitating constitutional symptoms resulting in a significant loss in quality of life and reduced life-span. There are currently no approved treatments for patients with MF.

Phase I/II Study Details

Estimated Enrollment: 60 patients.

Dosing: Oral, once-daily, self-administration dosing; patients assigned to dose levels in successive cohorts starting with a dose in the first cohort of 100 mg/day, dose escalation at a 1.5-fold increment.

Endpoints:

Primary:
- To determine the safety and tolerability, dose-limiting toxicities and maximum tolerated dose of orally-administered CYT387 in patients with PMF or post-ET/PV MF.
- Objective Response Rate, as measured by complete response rate, partial response rate and clinical improvement rate according to IWG-MRT consensus criteria.
- To determine the pharmacokinetics of CYT387 in patients with PMF or post-ET/PV MF.

Secondary:
- To determine the effect of CYT387 on cytogenetic findings in patients with PMF or post-ET/PV MF.
- To determine the effect of CYT387 on granulocyte JAK2V617F allele burden in patients with PMF or post-ET/PV MF.
- To determine the effect of CYT387 on endogenous myeloid colony formation in patients with PMF or post-ET/PV MF.
- To determine the effect of CYT387 on plasma levels of inflammatory, fibrogenic and angiogenic cytokines in patients with PMF or post-ET/PV MF.
- To evaluate pharmacodynamic correlates of CYT387 activity in patients with PMF or post-ET/PV MF who are receiving treatment with CYT387.

Source: ClinicalTrials.gov

YMI announced in March 2010 that the Company received approval to expand enrollment in the Phase I/II trial earlier than anticipated based on favorable safety and biological activity data collected to date.[5] As a result, preliminary efficacy data from the Phase II portion of the trial may be available in late 2010, instead of 2H CY2011 as previously announced. The Company hopes to use this trial to progress into a registration Phase III clinical trial in myelofibrosis.

CYT387 Key Events

Q3 CY2010 – Preliminary data from the Phase I portion of the CYT387 clinical trial in myelofibrosis.

Q4 CY2010 – Preliminary efficacy data from the Phase II portion of the CYT387 clinical trial.

Q2 CY2011 – Final efficacy data from the Phase II portion of the trial

Q4 CY2011 – Initiate Phase III registration trial of CYT387 in myelofibrosis.

[5] YM Biosciences, Inc. Press Release, "YM Biosciences announces early expansion of ongoing CYT387 Phase I/II clinical study based on favorable safety and activity data". March 30, 2010.

JAK 1/2 Inhibitor Competitive Landscape

Company	Development Status	Compound	Indication
Incyte Corp. (NasdaqGM: INCY)	Phase III	INCB18424	myelofibrosis
YM BioSciences (NYSE Amex: YMI)	Phase I/II	CYT387	myelofibrosis
S*BIO Pte Ltd.	Phase I/II	SB1518	myelofibrosis
TargeGen Inc.	Phase I (concluded)	TG101348	myelofibrosis
AstraZeneca (NYSE: AZN)	Phase I	AZD1480	myelofibrosis

Source: Griffin Securities, Inc.

Nimotuzumab At ASCO 2010

Posters and an abstract featuring nimotuzumab were presented during the 46th meeting of the Annual Society of Clinical Oncology (ASCO), June 4 to 8, 2010 in Chicago. The topics presented at the ASCO 2010 Annual Meeting included: (i) a summary of four-year survival results in head & neck cancer, (ii) results from a pilot study using nimotuzumab in combination with chemoradiation in head and neck cancer, and (iii) a summary of a study combining nimotuzumab with temozolomide and radiotherapy in patients with glioma. Data from YMI's licensee in Europe (Oncoscience AG) from a Phase III trial in adult glioma that had been anticipated to be presented at ASCO 2010 was unavailable. YMI was advised that, with last patient enrolled in March 2010, data collection and analysis had not been able to be completed in time.

HEAD AND NECK – Phase IIb 4-Year Survival Data

Long-term survival data from a randomized nimotuzumab squamous cell carcinoma of head and neck cancer (SCCHN) study previously presented demonstrate the clinical benefit with survival advantage for patients when combined with radiotherapy or chemoradiotherapy. These results compare favorably to the benefit shown in previous studies with other EGFR inhibitors but do not show the toxicities reported in those trials. Updated 4-year survival data was presented at ASCO, poster entitled "An open-label, randomized, study of h-R3mAb (nimotuzumab) in patients with advanced (stage III or IVa) squamous cell carcinoma of head and neck (SCCHN) Four-year survival data results from a Phase IIb study" (abstract #5530), Friday June 4, 2010.

The addition of nimotuzumab to radiotherapy (RT) and chemoradiotherapy (CRT) has an important effect on overall survival

- **Background:** Phase IIb, 4-arm, open-label, randomized, multi-center study to assess the safety and efficacy of radiation/chemoradiation therapy in combination with the novel anti-EGFR monoclonal antibody h-R3 in patients with advanced (stage III or IVa) inoperable head and neck cancer. A total of 92 patients were enrolled in the trial, of which 76 were considered evaluable. At 48 months, 41% of the patients in the nimotuzumab+CRT ARM were alive compared to 21% in CRT-alone arm; and 34.7% in the nimotuzumab+RT arm were alive compared to 13% in the RT-alone arm. The difference at 48 months between CRT and nimotuzumab+CRT arms reached statistical significance (p=0.0149). Kaplan-Meier curves for survival maintained a consisten separation at the 48-month update, demonstrating that the benefit of adding a fixed course of nimotuzumab to RT and CRT persists for an extended period.
- **Conclusions:** Notably, 4-year long-term survival data from a randomized Phase IIb study of nimotuzumab in squamous cell carcinoma of head and neck cancer (SCCHN) in a poster entitled "An open-label, randomized, study of h-R3mAb (nimotuzumab) in patients with advanced (stage III or IVa) squamous cell carcinoma of head and neck (SCCHN)" further demonstrated the clinical benefit with survival advantage for patients when combined with radiotherapy or chemoradiotherapy. These results compare favorably to the benefit shown in previous studies with other EGFR inhibitors in this

indication, but the poster reports that nimotuzumab continues to demonstrate efficacy without any of the elevated toxicities common with the other agents. The randomized Phase IIb ("BEST") clinical trial demonstrated that the efficacy of nimotuzumab compares favorably to results reported for Erbitux® and showed a statistically significant difference in overall survival (p=0.0018) between the arms. Significantly, this randomized trial supports the mechanism data that demonstrates that patients showed essentially equivalent clinical benefit from nimotuzumab in high-EGFR expressing cells (SCCHN >80%) without the numerous severe toxicities of Erbitux®.[6] Importantly, the addition of nimotuzumab did not add to the severe toxicities of either regimen, with no Grade 3-4 skin toxicities observed. Updated 4-year survival data will be presented at ASCO, poster entitled "An open-label, randomized, study of h-R3mAb (nimotuzumab) in patients with advanced (stage III or IVa) squamous cell carcinoma of head and neck (SCCHN) four-year survival data results from a Phase IIb study" (abstract #5530), Friday June 4, 2010.

HEAD & NECK - RADIOTHERAPY +/- CHEMOTHERAPY +/- NIMOTUZUMAB

A second poster on nimotuzumab in head & neck cancer, entitled "Results from a pilot study of nimotuzumab with concurrent chemoradiation in patients with locally advanced squamous cell carcinoma of head and neck (abstract # 5565)" summarized a study that had previously indicated improved chemoradiation outcomes with the addition of nimotuzumab. The data is also consistent with randomized data generated in the same trial setting. The study had enrollment of 92 patients.

ADULT GLIOMA - NIMOTUZUMAB + TEMOZOLOMIDE + RADIOTHERAPY

An abstract published in conjuction with the ASCO meeting entitled "Feasibility and safety of combining nimotuzumab with temozolomide and radiotherapy in adult patients with glioblastoma: An Indian clinical experience (abstract # e12509)" featured data from a study that suggests combining radiotherapy and temozolomide with nimotuzumab may lead to improved survival outcomes and is safe in patients. The trial is ongoing with enrollment of 56 patients and a primary endpoint of overall survival.

ADULT GLIOMA - TEMOZOLOMIDE + RADIOTHERAPY +/- NIMOTUZUMAB

Oncoscience AG, YMI's regional licensee in Western Europe for the development of nimotuzumab was expected to announce preliminary study results from its Phase III trial in which nimotuzumab and radiotherapy with concomitant and adjuvant temozolomide will be compared to the current standard of care in patients with glioblastoma multiforme (GBM). This Phase III study recruited 148 patients (74 patients in each arm). The primary endpoint for this trial is Progression Free Survival (PFS) at 6, 12, and 18 month intervals with response rate and symptom control among the secondary endpoints. Data had been anticipated to be presented at ASCO 2010. YMI, however, was advised that, with last patient enrolled in March 2010, data collection and analysis had not been able to be completed in time.

[6] Reddy BK, *et al*. A phase IIb 4-arm open-label randomized study to assess the safety and efficacy of h-R3 monoclonal antibody against EGFR in combination with chemoradiation therapy or radiation therapy in patients with advanced (stage III or IVA) inoperable head and neck cancer. American Society of Clinical Oncology (ASCO) Annual Meeting, 2009 (abstr 6041) and 2010, (abstract 5530).

NIMOTUZUMAB OVERVIEW

Nimotuzumab is YMI's humanized monoclonal antibody (mAb) targeting the epidermal growth factor receptor (EGFR) currently being studied in pediatric and adult gliomas, non-small cell lung (NSCLC), gastric, cervical, pancreatic, head & neck cancers, and various other solid tumors. Importantly, nimotuzumab has demonstrated activity in numerous clinical trials in over 10 indications without the presence of the severe side effects, including follicular (skin) rash associated with other EGFR receptor-targeting agents, such as cetuximab (Erbitux®), panitumumab (Vectibix®), and erlotinib (Tarceva®). YMI's license to nimotuzumab includes most of the major world markets, including the U.S. and Canada, Europe, Japan, and the Pacific Rim countries, excluding the People's Republic of China. In addition to YMI's extensive global consortium of licensees working to develop and commercialize nimotuzumab, CIMAB, the licensor, has numerous other licensees in emerging pharmaceutical markets. The table below lists YMI's regional licensees:

Major Partner	Region
Daiichi-Sankyo	**Japan**
Oncoscience AG	**Western Europe**
Kuhnil Pharma Co.	**Korea**
Innogene Kalbiotech/Kalbe Farma	**Singapore**

Source: YM Bioscience, Inc.

Nimotuzumab is already approved for sale in Argentina, Brazil, and Mexico in the Americas, and India, China, Indonesia, the Philippines and 19 other developing countries elsewhere.[7]

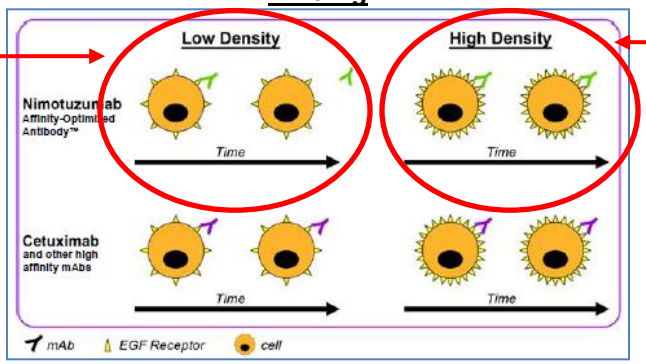
NIMOTUZMAB'S BIVALENT BINDING MECHANISM

On April 20, 2009, YMI presented a poster at the 100th AACR annual meeting entitled, "Binding properties of the anti-EGFR monoclonal antibody nimotuzumab limit its interaction with the EGFR in renal and epidermal cells."[8,9] Importantly, the results of the study demonstrated that nimotuzumab has a unique mechanistic difference compared to cetuximab and panitumumab that allows it to achieve statistically equivalent anti-tumor activity without causing the numerous serious side effects of the class, including follicular (skin-related) rash. Nimomtuzmab requires bivalent binding (requiring both "arms" of the molecule to link with EGF receptors) for efficient attachment to EGFR on the cellular surface. The strength of this binding is described as avidity, which is approximately affinity (monovalent binding) squared. Since nimotuzumab requires both "arms" of the molecule to attach to create a stable bond, it primarily binds in environments with high EGFR density, such as tumors, where EGF receptors are in close enough proximity for each arm to reach a different receptor. Conversely, cetuximab and panitumumab bind in both low and high EGFR density environments indiscriminately. Trastuzumab (Herceptin®), a well-known monoclonal antibody that targets the HER2 receptor approved to treat breast cancer, also depends on bivalent binding.[10] Nimotuzumab's avidity (bivalent binding) is illustrated in the diagram:

[7] YM Biosciences, Inc. website: "Consortium" http://www.ymbiosciences.com/products/nimotuzumab/codevelopment.php. 2009.
[8] YM Biosciences Inc. press release, "YM Biosciences Announces Nimotuzumab Presentations to be made at the 100th Annual Meting of the American Association for Cancer Research and European Society for Medical Oncology." March 13, 2009.
[9] Garrido G, *et al*. Binding properties of the anti-EGFR monoclonal antibody, nimotuzumab, limit interaction with the EGFR in renal and epidermal cells. American Association for Cancer Research (AACR) 100th Annual Meeting, 2009
[10] Steffen, A, et al. *In Vitro* Characterization of a Bivalent Anti-HER-2 Affibody with Potential for Radionuclide-Based Diagnostics. *Cancer Biotherapy and Radiopharmaceuticals* 2005; 20(3): 239-248.

Nimotuzumab: Attaches to EGF Receptors in High Expressing Tumor Cells through Bivalent Binding

Nimotuzumab demonstrates little binding activity in low-expressing cells, thus avoiding serious side effects, including severe skin rash.



Nimotuzumab binds in high-expressing tumor cells, such as in head and neck, glioma, cervical, and gastric cancers, and radiation treated cancers.

Source: YM Biosciences, Inc., Griffin Securities, Inc.

EGF receptors have many important biological functions and are expressed on the surface of healthy cells.[11] EGF receptors are amplified on cells in certain cancers and also in response to certain therapies – particularly radiation. Because the activity of nimotuzumab is necessarily concentrated in tumors that overexpress EGFR, it is a specifically active anti-cancer drug with a much improved safety profile over previous generations of higher affinity antibodies. Similar to higher affinity antibodies, nimotuzumab binds to EGF receptors with both antibody arms, thus blocking receptor activation and cancer cell growth in high EGFR density environments. In contrast to cancer cells, normal cells have low numbers of EGF receptors; therefore, nimotuzumab demonstrates transient binding activity, which causes it to avoid serious side effects, including severe skin rash. However, single arm binding affinity of higher affinity antibodies, including cetuximab and panitumumab, also occurs in low EGF density environments which if dosed for an extended period of time, can causes toxicity which can be severe. These higher affinity antibodies are unable by virtue of their higher affinity to discriminate between diseased and healthy cells.

High EGFR-Expressing Cancers
Head & Neck Cancer
Glioma
Cervical Cancer
Gastric Cancer
Non-Small-Cell Lung Cancer

Source: Griffin Securities, Inc.

Thus, by binding indiscriminately, cetuximab and panitumumab cause a range of side effects, including skin rash, which can be very serious. As shown in the following table, certain side effects attributed to EGFR inhibitors, such as Grades III & IV skin rash are high with Erbitux® and Vectibix® and very rare ("VR") with nimotuzumab. This result was confirmed in the *Journal of Clinical Oncology.*[12]

Treatment-Related Side Effects of HER1/EGFR Inhibition

	Erbitux™ plus Radiation (n=208)	Nimotuzumab plus Radiation (n=54)	Vectibix™ plus BSC (n=229)	BSC Alone (n=234)
Rash (All Grades) Antibody Related	87% (Acneiform)	6% (All types)	90%	9%
Rash (Grades 3 & 4)	14%	VR	14%	0%
Hypomagnesemia - Total	50%	VR	39%	2%
Nail	--	VR	29%	0%
Nausea	49%	56%	23%	16%
Diarrhea	19%	9%	21%	11%
Constipation	35%	4%	--	--
Vomiting	29%	15%	19%	12%
Eye	--	VR	15%	2%

Source: YM BioSciences, Inc.

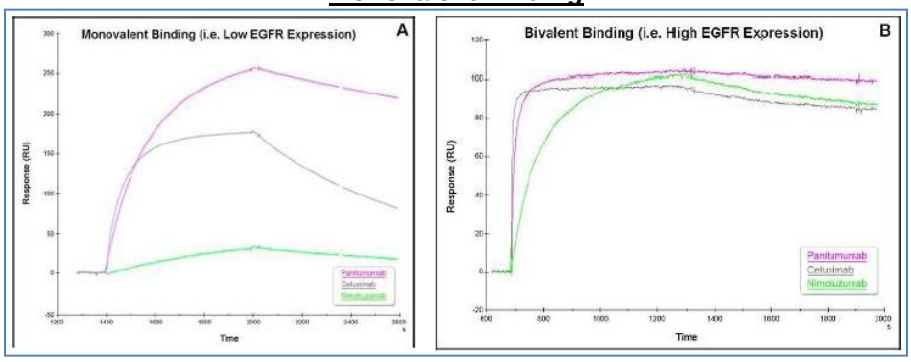
[11] Herbst, RS. Review of epidermal growth factor receptor biology. *Int J Radiat Oncol Biol Phys* 2004; 59(2 Suppl): 21-6.

[12] (JCO), Vol 22, No. 9, May 1, 2004. In clinical trials, potentially fatal infusion reactions were reported. Grade 1 and 2 infusion reactions, including chills, fever, and dyspnea usually occurring on the first day of initial dosing, were observed in 16% of patients receiving Erbitux plus irinotecan and 23% of patients receiving Erbitux monotherapy. Severe infusion reactions occurred with the administration of Erbitux in approximately 3% (17/633) of patients. Acneform rash was reported in 88% (560/633) of all treated patients and was severe (Grade 3 or 4) in 12% (79/633). Subsequent to the development of severe dermatologic toxicities, complications including S. aureus sepsis and abscesses requiring incision and drainage were reported. Product Monogram, Erbitux, February 2004.

Some of the cutaneous (skin-related) side effects caused by these agents are very distressing, partly because they are chronic due to the long duration of treatment. Therefore, patients need early and appropriate dermatological management.[13] A 2007 study published in the *Radiotherapy and Oncology* found that 28% of patients treated with cetuximab and radiation therapy developed grade IV skin rash.[14] A second 2007 study published in the *International Journal of Biological Markers* found that 90% of patients developed grade III or IV skin rash while being treated with cetuximab, radiation therapy and chemotherapy.[15]

In order to attempt to validate the proposed bivalent binding mechanism of nimotuzumab, YMI completed several studies. The first study compared the binding affinity of nimotuzumab, cetuximab, and panitumumab in high and low EGFR expression environments *in vitro* and demonstrated that cetuximab and panitumumab bind in the presence of low as well as high EGFR-expressing cells, while nimotuzumab only demonstrates activity in high EGFR expression locations.[16] This result is presented in the graphs that follow. The green line is nimotuzumab, the grey line is cetuximab, and the purple line is panitumumab. The first graph ("A") illustrates the binding response over time in a low EGFR expression environment. Nimotuzumab demonstrates very little binding activity, while panitumumab and cetuximab illicit a high signal and definitive binding activity. In the high EGFR environment, depicted in the second graph ("B"), all three antibodies display equivalent signals indicating binding activity.

Mechanistic Differences of Nimotuzumab, Cetuximab, and Panitumumab: Bivalent Versus Monovalent Binding

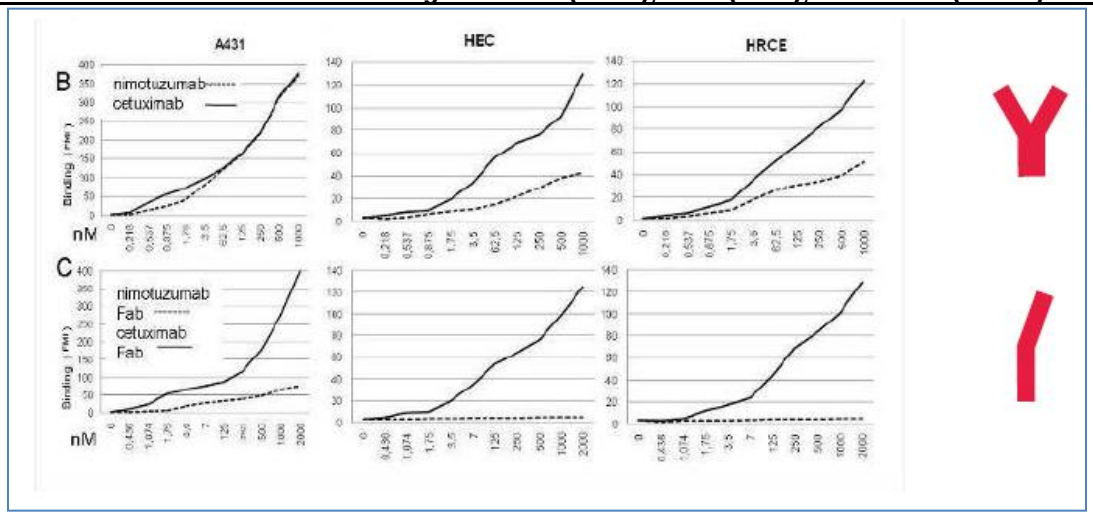


Source: YM Biosciences, Inc., Garrido et al. 2009

To prove that other monoclonal antibodies bind with only one arm – confirming monovalent binding affinity – a whole antibody and a fragment with only one arm were administered to human cells. If an antibody can bind having only one arm, it should establish the concept. As predicted from the experimental data above, nimotuzumab showed the same degree of binding activity in the tumor cell model (A431) as cetuximab when introduced as a complete molecule. In skin and renal cells (HEC and HRCE, respectively), nimotuzumab showed 60% less binding affinity than cetuximab. This is important because cetuximab's tendency to bind to EGFR in non-tumor containing cells is the reason for the severe side effects. In the bottom row of graphs, nimotuzumab showed very little binding activity when employed as a Fab (i.e. when the ability to form bivalent bonds was removed). Notably, cetuximab's binding affinity remained the same in all scenarios, including in the non-tumor skin and renal cells. Because nimotuzumab only binds in areas that contain high EGFR density, it preferentially targets tumors and avoids binding to skin cells and other low EGFR density areas. This targeted activity allows it to act with the same efficacy as cetuximab but without the serious side effect profile and makes it unique in targeting tumor while avoiding normal tissue.

13 Lancet Oncology 2005; 6: 491–500; Cutaneous side effects of kinase inhibitors and blocking antibodies; Codex, France.
[14] Giro, C. et al. High rate of severe radiation dermatitis during radiation therapy with concurrent cetuximab in head and neck cancer: Results of a survey in EORTC institutes. *Radiotherapy and Oncology* 2008; 90: 166-171.
[15] Monti, M. and S. Motta. Clinical management of cutaneous toxicity of anti-EGFR agents. *Int J Biol Markers* 2007; 22: 53-61.
[16] Garrido G, *et al*. Binding properties of the anti-EGFR monoclonal antibody, nimotuzumab, limit interaction with the EGFR in renal and epidermal cells. American Association for Cancer Research (AACR) 100th Annual Meeting, 2009

Monovalent Versus Bivalent Binding in Tumor (A431), Skin (HEC), and Renal (HRCE) Cells

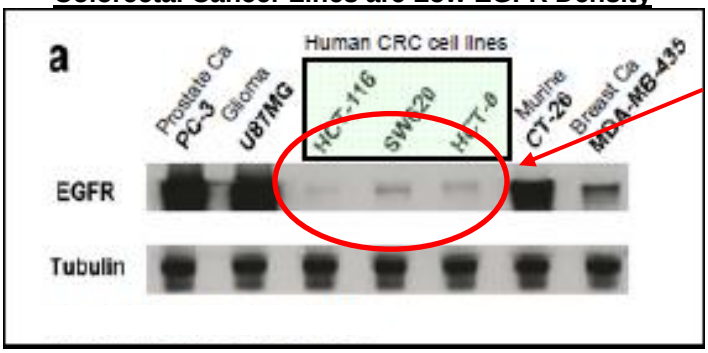


Source: YM Biosciences, Inc, Tikhomirov I et al. 2009

BIVALENCY SUPPORTED BY CLINICAL EVIDENCE

In August 2008, YM BioSciences presented data from a Phase II clinical trial in metastatic colorectal cancer (mCRC).[17] At the time, the bivalency of nimotuzumab had not been considered, so it was the belief of the Company that in order to successfully bring an EGFR-targeting antibody to market, they would have to replicate ImClone's BOND 1 study results of cetuximab in colorectal cancer, an indication believed to be promising for EGFR inhibitors. In fact, because of nimotuzumab's requirement for bivalent binding for efficacy, colorectal cancer is one of the least amenable indications to target (e.g., low-expressing tumor cell tumors are not targets for nimotuzumab) and refractory colorectal cancer would be less favorable because the initial treatment of the primary tumor with irinotecan and the established effect of irinotecan in specifically targeting high-EGFR cells leaving the patient with low EGFR-expressing cells. Numerous colorectal cancer cells have, in any event, low-EGFR density. As demonstrated above, nimotuzumab targets high EGFR density areas; it does not bind definitively to low EGFR density cells, which would include lower-expressing colorectal tumor cells. The image below shows EGFR expression in various cancer cell lines. From the left of the diagram, prostate cancer and glioma display very high levels of EGFR expression. Colorectal cancer, shown in the middle three cell lines, has very little EGFR expression. Breast cancer cell lines, the last displayed below, have somewhat higher EGFR expression.

Colorectal Cancer Lines are Low EGFR Density



EGFR expression is low in colorectal cancer cell lines.

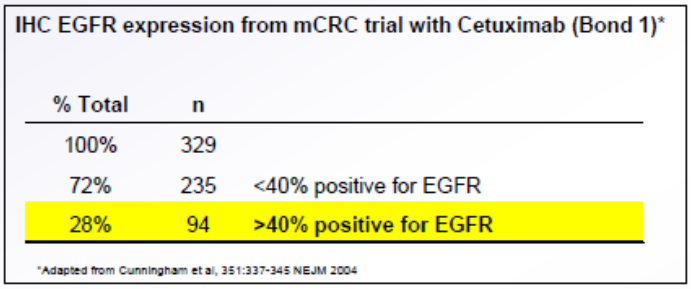
Source: YM BioSciences, Inc.

Because nimotuzumab does not bind definitively in low EGFR-expression environments, the efficacy of nimotuzumab in the Phase II trial was approximately ¼ that of cetuximab's response rate, a result that would be entirely predictable from the monovalent versus bivalent binding data depicted on the previous

[17] YM Biosciences Inc. press release, "YM Biosciences Reports Phase II Data for Nimotuzumab in Metastatic Colorectal Cancer." August 4, 2008.

page where nimotuzumab showed the same degree of binding to tumor cells vis-à-vis cetuximab but approximately 60% less binding in healthy tissue cells (e.g., skin and renal cells, minimizing attachment in low EGFR density areas. With hindsight, the mCRC trial results support the finding of nimotuzumab's unique bivalent mechanistic property because mCRC patients generally express low EGFR levels. In fact, in the Bond 1 mCRC trial with cetuximab, only 28% (94 out of 329) of the patients expressed high (>40%) EGFR levels, while the other 72% of patients expressed low EGFR levels (<40%). This is illustrated below:

Distribution of EGFR Expression Levels in mCRC Patients



Source: YM BioSciences, Inc.

NIMOTUZUMAB KEY EVENTS

Q3 2010 – Phase I preliminary safety and activity data on CYT387 in MF.

Q3 2010 – Preliminary Phase II data from Daiichi Sankyo's clinical trial in gastric cancer.

2H 2010 – Phase III decision expected from Daiichi Sankyo in gastric cancer.

Q4 2010 – Preliminary Phase II data from YMI USA's clinical trial in pediatric glioma.

Q4 2010 – Preliminary Phase II data from Daiichi Sankyo's clinical trial in NSCLC.

Q4 2010 – Recruitment completion targeted in Phase II clinical trial in brain metastases from NSCLC.

Q4 2010 – Recruitment completion targeted in Phase II clinical trial in palliative NSCLC.

Q4 2010 – Phase III decision expected from Daiichi Sankyo in NSCLC.

Q4 2010 – Phase II preliminary safety and activity data on CYT387 in MF.

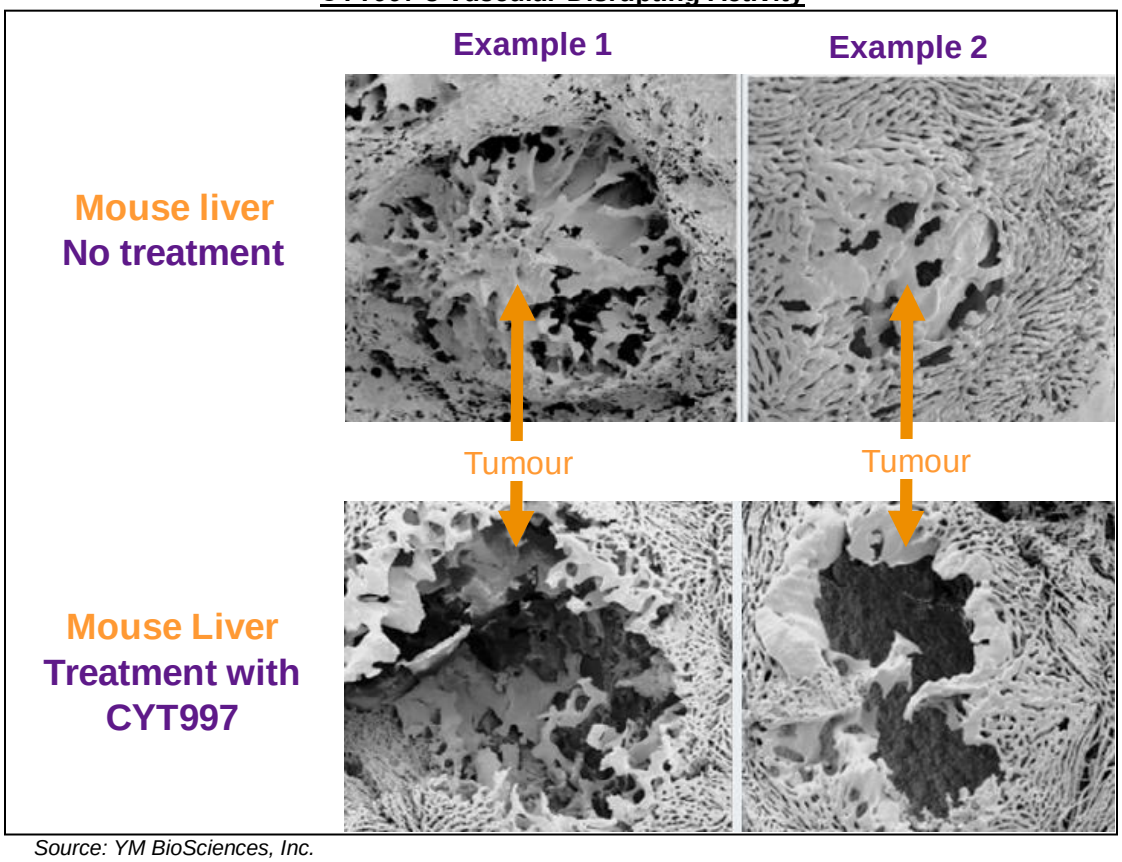
CYT997 OVERVIEW

CYT997 is an orally bioavailable, small-molecule, vascular-disrupting agent (VDA). CYT997 selectively disrupts tumor blood flow by blocking tubulin polymerization in tumor blood vessel endothelial cells. The selective disruption of the tumor vasculature leads to necrosis of the tumor. CYT997 also possesses direct cytotoxic activity against cancer cells. Oral and IV presentations of CYT997 have completed Phase I studies.[18,19]

CYT997 is differentiated from other agents in this class by being one of very few "orally" bioavailable VDA's in clinical development (the only one we could identify). Potential advantages of oral administration of CYT997 include patient convenience and even more importantly, potentially improved efficacy and safety. The vast majority of current VDA's in development are administered intravenously which positions them to be administered infrequently and at high-doses. Such schedules result in rapid tumor revascularization (within days of treatment) and toxicities. High-doses of VDA's are also likely cause spikes in circulating pro-angiogenic endothelial precursor cells (CEPs) that restore tumor vasculature.[20] In contrast, oral administration of CYT997 at low, minimally toxic, doses at frequent intervals may lead to a more sustained disruption of tumor vasculature, reduced induction CEP-mediated resistance pathway, and improved safety.

Use of VDAs encompasses majority of solid tumors and CYT997 demonstrated activity against a range of cancer cells. CYT997 is currently being studied in a Phase I/II clinical trial in combination with carboplatin in patients with relapsed glioblastoma multiforme (glioma).

CYT997's Vascular-Disrupting Activity



Source: YM BioSciences, Inc.

[18] ASCO 2008 Presentation. Lickliter et al. Phase I evaluation of CYT997, a novel cytotoxic and vascular-disrupting agent, in patients with advanced cancer. Royal Brisbane and Women's Hospital.
[19] ASCO 2009 Presentation. Francesconi et al. Phase I evaluation of orally-administered CYT997, a novel cytotoxic vascular disrupting agent, in patients with advanced cancer. Queensland Government Queensland Health.
[20] Shaked et al. Rapid Chemotherapy-Induced Acute Endothelial Progenitor Cell Mobilization: Implications for Antiangiogenic rugs as Chemosensitizing Agents. *Cancer Cell* 2008;14(3): 263-73.

CYT997 is a wholly synthetic compound that possesses highly potent cytotoxic activity *in vitro* through inhibition of microtubule polymerization. CYT997 blocks the cell cycle at the G2-M boundary, and Western blot analysis indicates an increase in phosphorylated Bcl-2, along with increased expression of cyclin B1. Caspase-3 activation is also observed in cells treated with CYT997 along with the generation of poly (ADP-ribose) polymerase. The compound possesses favorable pharmacokinetic properties, is orally bioavailable, and is efficacious in a range of *in vivo* cancer models, including some refractory to paclitaxel treatment. CYT997 exhibits vascular disrupting activity as measured *in vitro* by effects on the permeability of human umbilical vein endothelial cell monolayers, and *in vivo* by effects on tumor blood flow. CYT997 possesses a useful combination of pharmacologic and pharmacokinetic properties and has considerable potential as a novel anticancer agent.[21]

CYT997 KEY EVENTS

2H 2010 – Preliminary data from Phase I/II clinical trial of CYT997 + carboplatin in glioblastoma multiforme (GBM).

(Intentionally left blank)

[21] Molecular Cancer Therapeutics. 2009;8(11):3036–45 2009. CYT997: a novel orally active tubulin polymerization inhibitor with potent cytotoxic and vascular disrupting activity in vitro and in vivo. Christopher J. Burns, Emmanuelle Fantino, Ian D. Phillips, Stephen Su, Michael F. Harte, Patricia E. Bukczynska, Mark Frazzetto, Max Joffe, Irma Kruszelnicki, Bing Wang, Yue Wang, Neil Wilson, Rodney J. Dilley, Soo S. Wan, Susan A. Charman, David M. Shackleford, Rosa Fida, Cathy Malcontenti-Wilson and Andrew F. Wilks.

INVESTMENT CONCERNS AND RISKS

For a complete description of risks and uncertainties related to YM BioSciences, Inc.'s business, see the "Risk Factors" section in YM BioSciences' SEC filings, which can be accessed directly from the SEC Edgar filings at www.sec.gov. Potential risks include:

- **Stock risk and market risk:** There is a limited trading market for the Company's common stock. There can be no assurance that an active and liquid trading market will develop or, if developed, that it will be sustained, which could limit one's ability to buy or sell the Company's common stock at a desired price. Investors should also consider technical risks common to many small-cap or micro-cap stock investments, such as small float, risk of dilution, dependence upon key personnel, and the strength of competitors that may be larger and better capitalized.
- **New and rapidly changing field:** The pharmaceutical and biotechnological markets are rapidly evolving, and research and development are expected to continue at an accelerated pace with increased frequency. Other companies are also actively engaged in the development of therapies to directly or indirectly treat those disorders being pursued by YM BioSciences. These companies may have substantially greater research and development capabilities, as well as significantly greater marketing, financial, and human resources abilities than YM BioSciences.
- **Products still in development phases:** Although the Company intends to continue with clinical development of nimotuzumab for the treatment of pediatric and adult glioma, non-small cell lung (NSCLC), gastric, cervical cancers, and various other solid tumors, CYT387 for myelofibrosis and other myeloproliferative neoplasms (MPNs), CYT997 for advanced cancers, AeroLEF™ for break through pain, and other future pipeline candidates in various indications, the successful development of the Company's product candidates is uncertain. Product development costs and timelines can vary significantly for each product candidate and are difficult to accurately predict. In addition, products in development that appear to be promising may not reach commercialization for various reasons, including failure to achieve regulatory approvals, safety concerns, and/or the inability to be manufactured at a reasonable cost.
- **Funding requirements:** It is difficult to predict the Company's future capital requirements. The Company may need additional financing to continue funding the research and development of its products and to expand its business. There is no guarantee that it can secure the desired future capital or, if sufficient capital is secured, that current shareholders will not suffer significant dilution.
- **Regulatory risk:** Various statutes and regulations also govern or influence the manufacturing, safety, labeling, storage, record keeping and marketing of each product. The lengthy process of seeking approval and the subsequent compliance with applicable statutes and regulations require the expenditure of substantial resources. Any failure by us to obtain, or any delay in obtaining, regulatory approvals could materially adversely affect YM BioSciences' business. There is no guarantee that YM Biosciences' products will be approved by the U.S. Food and Drug Administration (FDA) or international regulatory bodies for marketing in the U.S. or abroad.
- **The Company may need to raise additional capital, which may not be available on terms acceptable to them, if at all:** As the Company continues to expand their research and development and sales and marketing activities, they may need to raise additional capital, which may not be available on terms acceptable to them, if at all. If the Company cannot raise necessary additional capital on acceptable terms, they may not be able to increase sales, develop or enhance their products and services, take advantage of future opportunities, or respond to competitive pressures or unanticipated requirements, any of which could cause their business to suffer.
- **Competitive risk:** The biotechnology industry is extremely competitive, mainly due to its large market potential. Many companies are developing products for the same therapeutic indications targeted by YM BioSciences. These companies may have substantially more resources than YM BioSciences, which could adversely affect the Company's position in the market place.

FINANCIAL FORECASTS & VALUATION

The following assumptions refer to YMI's revenue model, annual earnings model, and valuation analysis. The revenue estimates are for nimotuzumab royalties in the US, nimotuzumab royalties received from Oncoscience AG in Europe, Kuhnil Pharma Co. in Korea, Daiichi Sankyo Co. Ltd. in Japan, and Innogene Kalbiotech in Singapore, CYT997 royalties in the U.S., and CYT387 royalties in the U.S. We have not included potential upfront fees or milestone revenue from, nor expenses associated with, YMI's other product candidates.

HISTORICAL BALANCE SHEET

CAD$ in thousands

Fiscal Year ended June 30

ASSETS	**3/31/2010**	**6/30/2009**
Current Assets		
Cash & equivalents (1)	48,012	42,051
Accounts receivable	391	565
Prepaid expenses	130	353
Total Current Assets	$ 48,532	$ 42,968
Property & equipment	$ 96	$ 97
Intangible assets	14,982	3,005
Other	-	-
Total Assets	$ 63,610	$ 46,070
LIABILITIES		
Current Liabilities		
Accounts payable	$ 1,085	$ 431
Accrued liabilities	2,143	487
Deferred revenue, current portion	1,528	2,550
Total Current Liabilities	$ 4,755	$ 3,467
Deferred revenue, non-current portion	$ 2,032	$ 2,898
Total Liabilities	$ 6,787	$ 6,366
Shareholders Equity		
Share capital	$ 200,168	$ 172,921
Share purchase warrants	1,473	-
Contributed surplus	13,803	13,035
Deficit	(158,621)	(146,252)
Total Shareholders Equity	$ 56,823	$ 39,704
Total Liabilities & Equity	$ 63,610	$ 46,070

Notes:

(1) Cash and equivalents does not include approximately $3.2 million raised in June 2010.

REVENUE ASSUMPTIONS

Assumes Oncoscience AG will develop nimotuzumab for pediatric and adult glioma, and YMI will receive 15% of product sales in Europe.

Assumes Kuhnil Pharma Co. and Daiichi Sankyo Co. Ltd. will develop nimotuzumab for non-small cell lung cancer (NSCLC) and gastric cancer, and YMI will receive 15% of product sales in Korea and Japan.

Assumes Innogene Kalbiotech will develop nimotuzumab for cervical cancer, and YMI will receive 15% of product sales.

Assumes YMI will out-license nimotuzumab for pediatric and adult glioma, palliative NSCLC, brain metastases from NSCLC, gastric cancer, and cervical cancer for the U.S. market on approval, and the Company will receive an upfront payment of $100,000,000, recognition of which, as revenue, will be deferred and amortized to income over a 60-month period, and a royalty of 15% of total product sales in each indication. We also assume that YMI will receive milestone payments that add upside to our estimates.

We also expect Innogene Kalbiotech to develop nimotuzumab for cervical cancer in Indonesia, the Philippines, Malaysia, and South Africa, which we have not modeled at this time but could represent significant upside to our estimates. We expect Daiichi-Sankyo, the Japanese licensee of nimotuzumab, Kuhnil Pharma Co., the Korean licensee of nimotuzumab, and YMI's other partners to develop the drug for treatment of various solid tumors currently in clinical trials, but we have excluded potential revenue in our model at this time. We believe these opportunities could also add significant upside to our estimates.

Assumes YMI will develop CYT997 for glioblastoma multiforme and CYT387 for myeloproliferative diseases and out-license the drugs on approval for a 15% royalty on total product sales.

Other revenue assumptions include:

LICENSING & MILESTONES

Assumes Oncoscience AG will find a sub-licensee to aid distribution upon the commercial release of nimotuzumab in FY2011. Assumes an up-front payment of $75,000,000, 50% of which we believe the Company is eligible to receive, recognition of which, as revenue, will be deferred and amortized to income over a 48-month period. The projected revenue also includes existing licensing and milestone agreements.

DRUG SALES

All currency amounts expressed in the following section are in US Dollars. Where applicable, foreign exchange rate is USD$1.00 equals CAD$1.0259.

Nimotuzumab: Pediatric & Adult Glioma - EU

Year penetration starts	2011	Prevalence	45000
Starting penetration rate	5%	Percent addressable	80%
Years between penetration start and peak	5	Market growth rate	1%
Peak penetration	20%	Price per patient per year	$20,000
Duration of peak penetration in years	3	Treatment price growth	1%
Retention rate in decline years	90%	Royalty rate	15%
Stage of development	Phase III	Probability of commercialization	80%

- There are approximately 45,000 pediatric and adult glioma patients in Europe;[22]
- Approximately 80% of the patients will be eligible for nimotuzumab;
- Nimotuzumab penetrates the market beginning in FY2011 at a price of $20,000 per treatment cycle;

[22] The Globocan 2002 Database – web-page address http://www-dep.iarc.fr/globocan/database.htm International Agency for Research on Cancer (IARC), part of the World Health Organization (WHO).

- The price per prescription grows at an annual rate of 1%; and
- Nimotuzumab penetrates 5% of the addressable market in the FY2011 launch year and reaches a peak penetration of 20% of the addressable market in FY2016.

Nimotuzumab: Pediatric & Adult Glioma - U.S.

Year penetration starts	2012	Prevalence	115000
Starting penetration rate	5%	Percent addressable	80%
Years between penetration start and peak	5	Market growth rate	1%
Peak penetration	20%	Price per patient per year	$30,000
Duration of peak penetration in years	3	Treatment price growth	1%
Retention rate in decline years	90%	Royalty rate	15%
Stage of development	Phase III	Probability of commercialization	15%

- There are approximately 115,000 pediatric and adult glioma patients in the U.S.;[23]
- Approximately 80% of the patients will be eligible for nimotuzumab;
- Nimotuzumab penetrates the market beginning in FY2012 at a price of $30,000 per treatment cycle;
- The price per prescription grows at an annual rate of 1%; and
- Nimotuzumab penetrates 5% of the addressable market in the FY2012 launch year and reaches a peak penetration of 20% of the addressable market in FY2017.

Nimotuzumab: Non-Small-Cell Lung Cancer (NSCLC) - U.S.

Year penetration starts	2012	Prevalence	324000
Starting penetration rate	3%	Percent addressable	40%
Years between penetration start and peak	5	Market growth rate	1%
Peak penetration	20%	Price per patient per year	$30,000
Duration of peak penetration in years	3	Treatment price growth	1%
Retention rate in decline years	90%	Royalty rate	15%
Stage of development	Phase II	Probability of commercialization	15%

- There are approximately 324,000 NSCLC patients in the U.S.;[24]
- Approximately 40% of the patients will be eligible for nimotuzumab as the preferred treatment method;
- Nimotuzumab penetrates the market beginning in FY2012 at a price of $30,000 per treatment cycle;
- The price per prescription grows at an annual rate of 1%; and
- Nimotuzumab penetrates 3% of the addressable market in the FY2012 launch year and reaches a peak penetration of 20% of the addressable market in FY2017.

[23] SEER Cancer Statistics Review, 1973-2004, National Cancer Institute. Surveillance Epidemiology and End Results (SEER), 1973-2004.
[24] SEER Cancer Statistics Review, 1973-2004, National Cancer Institute. Surveillance Epidemiology and End Results (SEER), 1973-2004.

Nimotuzumab: Non-Small-Cell Lung Cancer (NSCLC) - Korea & Japan

Year penetration starts	2013	Prevalence	82000
Starting penetration rate	3%	Percent addressable	40%
Years between penetration start and peak	5	Market growth rate	1%
Peak penetration	20%	Price per patient per year	$25,000
Duration of peak penetration in years	3	Treatment price growth	1%
Retention rate in decline years	90%	Royalty rate	15%
Stage of development	Phase II	Probability of commercialization	30%

- There are approximately 82,000 NSCLC patients in Korea and Japan;[25]
- Approximately 40% of the patients will be eligible for nimotuzumab as the preferred treatment method over existing treatment options;
- Nimotuzumab penetrates the market beginning in FY2013 at a price of $25,000 per treatment cycle;
- The price per prescription grows at an annual rate of 1%; and
- Nimotuzumab penetrates 3% of the addressable market in the FY2013 launch year and reaches a peak penetration of 20% of the addressable market in FY2018.

Nimotuzumab: Gastric Cancer - Korea & Japan

Year penetration starts	2013	Prevalence	125000
Starting penetration rate	3%	Percent addressable	60%
Years between penetration start and peak	5	Market growth rate	1%
Peak penetration	20%	Price per patient per year	$25,000
Duration of peak penetration in years	3	Treatment price growth	1%
Retention rate in decline years	90%	Royalty rate	15%
Stage of development	Phase II	Probability of commercialization	35%

- There are approximately 125,000 gastric cancer patients in Korea and Japan;[26]
- Approximately 60% of the patients will be eligible for nimotuzumab as the preferred treatment method;
- Nimotuzumab penetrates the market beginning in FY2013 at a price of $25,000 per treatment cycle;
- The price per prescription grows at an annual rate of 1%; and
- Nimotuzumab penetrates 3% of the addressable market in the FY2013 launch year and reaches a peak penetration of 20% of the addressable market in FY2018.

[25] The Globocan 2002 Database – web-page address http://www-dep.iarc.fr/globocan/database.htm International Agency for Research on Cancer (IARC), part of the World Health Organization (WHO).
[26] The Globocan 2002 Database – web-page address http://www-dep.iarc.fr/globocan/database.htm International Agency for Research on Cancer (IARC), part of the World Health Organization (WHO).

Nimotuzumab: Gastric Cancer - EU

Year penetration starts	2013	Prevalence	220000
Starting penetration rate	3%	Percent addressable	60%
Years between penetration start and peak	5	Market growth rate	1%
Peak penetration	15%	Price per patient per year	$15,000
Duration of peak penetration in years	3	Treatment price growth	1%
Retention rate in decline years	90%	Royalty rate	15%
Stage of development	Phase II	Probability of commercialization	35%

- There are approximately 220,000 gastric cancer patients in Europe;[27]
- Approximately 60% of the patients will be eligible for nimotuzumab as the preferred treatment method;
- Nimotuzumab penetrates the market beginning in FY2013 at a price of $15,000 per treatment cycle;
- The price per prescription grows at an annual rate of 1%; and
- Nimotuzumab penetrates 3% of the addressable market in the FY2013 launch year and reaches a peak penetration of 15% of the addressable market in FY2018.

Nimotuzumab: Gastric Cancer - U.S.

Year penetration starts	2013	Prevalence	64000
Starting penetration rate	3%	Percent addressable	60%
Years between penetration start and peak	5	Market growth rate	1%
Peak penetration	20%	Price per patient per year	$30,000
Duration of peak penetration in years	3	Treatment price growth	1%
Retention rate in decline years	90%	Royalty rate	15%
Stage of development	Phase II	Probability of commercialization	15%

- There are approximately 64,000 gastric cancer patients in the U.S.;[28]
- Approximately 60% of the patients will be eligible for nimotuzumab as the preferred treatment method;
- Nimotuzumab penetrates the market beginning in FY2013 at a price of $30,000 per treatment cycle;
- The price per prescription grows at an annual rate of 1%; and
- Nimotuzumab penetrates 3% of the addressable market in the FY2013 launch year and reaches a peak penetration of 20% of the addressable market in FY2018.

[27] The Globocan 2002 Database – web-page address http://www-dep.iarc.fr/globocan/database.htm International Agency for Research on Cancer (IARC), part of the World Health Organization (WHO).
[28] SEER Cancer Statistics Review, 1973-2004, National Cancer Institute. Surveillance Epidemiology and End Results (SEER), 1973-2004.

Nimotuzumab: Cervical Cancer - U.S.

Year penetration starts	2013	Prevalence	250000
Starting penetration rate	3%	Percent addressable	50%
Years between penetration start and peak	5	Market growth rate	-2%
Peak penetration	10%	Price per patient per year	$30,000
Duration of peak penetration in years	3	Treatment price growth	1%
Retention rate in decline years	90%	Royalty rate	15%
Stage of development	Phase II	Probability of commercialization	15%

- There are approximately 250,000 cervical cancer patients in the U.S.;[29]
- Approximately 50% of the patients will be eligible for nimotuzumab as the preferred treatment method over existing treatment options;
- Nimotuzumab penetrates the market beginning in FY2013 at a price of $30,000 per treatment cycle;
- The price per prescription grows at an annual rate of 1%; and
- Nimotuzumab penetrates 3% of the addressable market in the FY2013 launch year and reaches a peak penetration of 10% of the addressable market in FY2018.

CYT997: Glioblastoma Multiforme - U.S.

Year penetration starts	2015	Prevalence	124000
Starting penetration rate	5%	Percent addressable	25%
Years between penetration start and peak	5	Market growth rate	1%
Peak penetration	20%	Price per patient per year	$30,000
Duration of peak penetration in years	3	Treatment price growth	1%
Retention rate in decline years	90%	Royalty rate	15%
Stage of development	Phase II	Probability of commercialization	15%

- The prevalence of CNS tumors in the U.S. is approximately 124,000, and about 25% of the cases are glioblastoma multiforme;[30]
- Approximately 80% of the patients will be eligible for CYT997;
- CYT997 penetrates the market beginning in FY2015 at a price of $30,000 per treatment cycle;
- The price per prescription grows at an annual rate of 1%; and
- CYT997 penetrates 5% of the addressable market in the FY2015 launch year and reaches a peak penetration of 20% of the addressable market in FY2020.

[29] SEER Cancer Statistics Review, 1973-2004, National Cancer Institute. Surveillance Epidemiology and End Results (SEER), 1973-2004.
[30] SEER Cancer Statistics Review, 1973-2004, National Cancer Institute. Surveillance Epidemiology and End Results (SEER), 1973-2004.

CYT387: Myeloproliferative Diseases - U.S.

Year penetration starts	2015	Prevalence	200000
Starting penetration rate	3%	Percent addressable	80%
Years between penetration start and peak	4	Market growth rate	1%
Peak penetration	20%	Price per patient per year	$30,000
Duration of peak penetration in years	4	Treatment price growth	1%
Retention rate in decline years	90%	Royalty rate	15%
Stage of development	Phase I	Probability of commercialization	35%

- There are approximately 200,000 patients with myeloproliferative diseases, including polycythemia vera (PV), essential thrombocythemia (ET), and idiopathic myelofibrosis (MF), in the U.S.;
- Approximately 80% of the patients will be eligible for CYT387;
- CYT387 penetrates the market beginning in FY2015 at a price of $30,000 per treatment cycle;
- The price per prescription grows at an annual rate of 1%; and
- CYT387 penetrates 3% of the addressable market in the FY2015 launch year and reaches a peak penetration of 20% of the addressable market in FY2019.

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INCOME STATEMENT

CAD$ in thousands, except per share data

FY ending June 30	2010	2011	2012	2013	2014
	2010	**2011**	**2012**	**2013**	**2014**
Total revenue	$ 4,500	$ 15,854	$ 68,982	$ 172,418	$ 249,627
COGS	-	-	-	-	-
Gross profit	$ 4,500	$ 15,854	$ 68,982	$ 172,418	$ 249,627
Operating expenses					
R&D	$ 14,500	$ 20,000	$ 15,000	$ 15,000	$ 10,000
Selling & marketing	-	-	-	-	-
General & administrative	6,250	6,500	6,750	7,000	7,250
Total expense	20,750	26,500	21,750	22,000	17,250
Operating profit	$ (16,250)	$ (10,646)	$ 47,232	$ 150,418	$ 232,377
Non-operating income/expense					
Interest expense	-	-	-	-	-
Interest income	-	-	-	-	-
Other	-	-	-	-	-
Total non-operating	-	-	-	-	-
Pretax profit	$ (16,250)	$ (10,646)	$ 47,232	$ 150,418	$ 232,377
Income tax	-	-	17,948	57,159	88,303
Net income	$ (16,250)	$ (10,646)	$ 29,284	$ 93,259	$ 144,074
Earnings (loss) per share	$ (0.17)	$ (0.11)	$ 0.30	$ 0.96	$ 1.47
Fully-diluted shares outstanding	96,794	97,000	97,250	97,500	97,750

Income Statement Assumptions:

- COGS of 0% of total sales as partners will assume these costs;
- Research and Development (R&D) expenses of $14.5 million in FY2010, $20 million in FY2011, $15 million in FY2012 and FY2013, and $10 million in FY2014;
- Zero Sales and Marketing (S&M) expense for nimotuzumab, CYT997, and CYT387;
- General and Administrative (G&A) expenses of $6.25 million in FY2010, $6.5 million in FY2011, $6.75 million in FY2012, $7.0 million in FY2013, and $7.25 million in FY2014;
- Income tax rate of 38%;
- The number of shares outstanding increases due to the exercise of stock options and warrants.

DISCOUNTED CASH FLOW (DCF) MODEL

Our DCF model, using a discount rate of 12.5%, suggests a value of USD$5.37 for YMI shares. Where applicable, we assume the foreign exchange rate is USD$1.00 equals CAD$1.0259.

CAD$ in thousands, except per share data	2010	2011	2012	2013	2014
	2010	**2011**	**2012**	**2013**	**2014**
Revenue	$ 4,500	$ 15,854	$ 68,982	$ 172,418	$ 249,627
Operating income	(16,250)	(10,646)	47,232	150,418	232,377
Net income	(16,250)	(10,646)	29,284	93,259	144,074
Depreciation/amortization	2,500	2,500	2,500	2,500	2,500
Stock-based compensation	1,000	1,000	1,500	2,000	2,000
Tax loss carryforwards	-	-	17,948	57,159	88,303
Capital expenditures	(100)	(125)	(125)	(150)	(150)
Asset acquisitions					
Other					
Total cash flow adjustments	3,400	3,375	21,823	61,509	92,653
Free cash flow	$ (12,850)	$ (7,271)	$ 51,107	$ 154,768	$ 236,727
Gross profit weighted probability	*100.0%*	*100.0%*	*36.1%*	*27.4%*	*26.4%*
Risk-adjusted free cash flow	$ (12,850)	$ (7,271)	$ 18,468	$ 42,395	$ 62,497

USD$ in thousands, except per share data

Exchange Rate (USD$/CAD$) 1.0259

Discount Rate	Discounted Cash Flows (2008 - 2023)	PV of Terminal Value at a Perpetual growth rate of rFCF 2.0%	3.0%	4.0%	Enterprise Value 2.0%	3.0%	4.0%
7.5%	$526,763	$ 484,634	$ 598,138	$ 776,501	$1,011,397	$1,124,901	$1,303,264
10.0%	$421,566	$ 236,006	$ 272,366	$ 320,845	$657,572	$693,932	$742,411
12.5%	$340,821	$ 128,359	$ 143,261	$ 161,670	$469,180	$484,082	$502,491
15.0%	$278,145	$ 74,558	$ 81,563	$ 89,841	$352,703	$359,708	$367,986
17.5%	$228,974	$ 45,290	$ 48,888	$ 53,019	$274,264	$277,862	$281,994

Discount Rate	Net Debt	Total Equity Value 2.0%	3.0%	4.0%	Value per Diluted Share 2.0%	3.0%	4.0%
7.5%	$ (41,225)	$1,052,622	$1,124,901	$1,344,489	$ 10.77	$ 11.51	$ 13.75
10.0%	(41,225)	$698,797	$735,156	$783,636	$ 7.15	$ 7.52	$ 8.02
12.5%	(41,225)	$510,405	$525,307	$543,716	$ 5.22	$ 5.37	$ 5.56
15.0%	(41,225)	$393,927	$400,933	$409,211	$ 4.03	$ 4.10	$ 4.19
17.5%	(41,225)	$315,489	$319,087	$323,218	$ 3.23	$ 3.26	$ 3.31

Discount Rate	Terminal Value as % Enterprise Value 2.0%	3.0%	4.0%	Implied EBITDA Multiple 2.0%	3.0%	4.0%
7.5%	47.9%	53.2%	59.6%	11.59	14.31	18.58
10.0%	35.9%	39.2%	43.2%	7.97	9.20	10.84
12.5%	27.4%	29.6%	32.2%	6.07	6.78	7.65
15.0%	21.1%	22.7%	24.4%	4.91	5.37	5.91
17.5%	16.5%	17.6%	18.8%	4.11	4.44	4.82

DISCLOSURES

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PRICE CHART

Source: BigCharts.com

11/15/2004 – Initiating Coverage: share price: $2.25; rating: BUY; 12-month price target: $5.40; **10/25/2005 –** Research Update: share price: $2.65; rating: BUY; 12-month price target: $6.00; **8/08/2007** – Research Update: share price: $1.45; rating: BUY; 12-month price target: $6.50; **8/24/2009** – Research Update: share price: $0.93; rating: BUY; 12-month price target: $5.00; **10/28/2009** – Research Update: share price: $1.07; rating: BUY; 12-month price target: $5.50. **6/23/2010** – Research Update: share price: $1.17; rating: BUY; 12-month price target: $5.50.

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