THE MORGAN WELCH INFLAMMATORY BREAST CANCER RESEARCH PROGRAM AND CLINIC

Annual Report

for Fiscal Year 2012

THE UNIVERSITY OF TEXAS
MD Anderson Cancer Center
Making Cancer History®

Executive and Core Leaders

Naoto T. Ueno, MD, PhD
Executive Director

Vicente Valero, MD
Clinical Research Director

Savitri Krishnamurthy, MD
Core Leader

Wei Yang, MBBS, FRCP
Core Leader

Danielle M. Walsh, MBA
Program Manager

Wendy Woodward, MD, PhD
Deputy Director

James Reuben, PhD
Executive Committee Member

Anthony Lucci, MD
Core Leader

Randa A. El-Zein, MD, PhD
Core Leader

Tom Buchholz, MD
Special Advisor

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RESEARCH PROGRAM AND CLINIC

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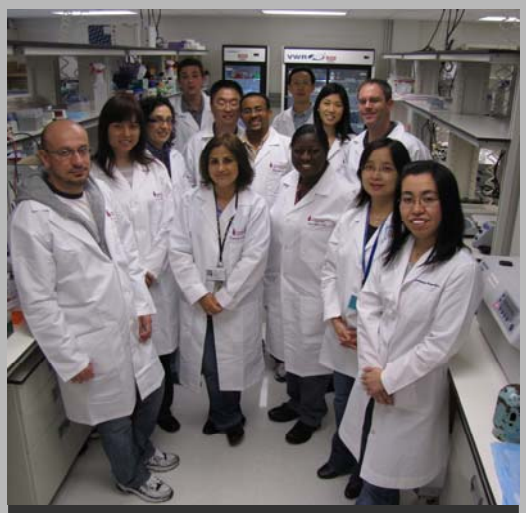
Overview

The University of Texas MD Anderson Cancer Center Morgan Welch Inflammatory Breast Cancer Research Program and Clinic is a unique model of multi-disciplinary care and research focused on understanding, preventing and treating inflammatory breast cancer (IBC). While IBC is considered rare, attributing to only 2-5% of all breast cancers, its aggressive nature makes it the deadliest, with only a 35-40% 5-year survival rate. In fact, death from IBC is disproportionate when compared to all breast cancers, resulting in up to 10% of all breast cancer deaths. Our multidisciplinary program is focused on developing tools for diagnosis, identifying therapeutic approaches specifically for treatment of IBC, understanding and preventing metastases, enhancing imaging approaches to assist in detecting the disease and evaluating the effectiveness of treatment for IBC patients. Our ultimate goal is to improve survival of IBC patients.

Over the past year, the Morgan Welch IBC Program has strived to progress into a period of strategic growth in patient care, basic and translational research, and in outreach. To accomplish this we have realigned our purpose and budget with clear mission, vision and defined goals for achievement.

Our mission is to reduce the suffering of IBC patients through translational research–driven, clinical medicine.

Our vision is to be a world premier IBC research group in disease prevention, in developing innovative molecular biomarkers and targeted therapy based on hypothesis-driven translational/clinical research, and in nurturing a new generation of oncology investigators.



OUR MISSION

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OUR VISION

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Based on the shared mission and vision, we defined our Program goals and aligned resources and effort to answer the following targeted questions over three years beginning in September 2011.

How can we distinguish IBC from non-IBC at the molecular level that will lead to **universal diagnostic criteria**?

What are the **IBC unique etiologies** that can be developed as IBC-specific targets?

What are the **risk factors** for a development of IBC?

Further, we identified the following innovations or changes as necessary to enhance our ability to answer the three questions above.

Development of molecular epidemiology

Development of a large clinical and biomarker database

Development of reliable IBC animal models

Well-defined collaboration strategies with a clear plan

Development of theme-focused research planning

Development of novel IBC clinical trials that include an extensive translational research component

Through a concerted effort by all members of the program and through new collaborations, the Morgan Welch IBC Program is committed to making breakthroughs in the understanding of IBC and how we can prevent, diagnose and treat new or metastatic disease. In June of 2012, members of the Program attended a team-building workshop to understand and evaluate our multi-disciplinary approach and to explore areas of opportunity for collaboration. From this workshop , we have identified 3 new working groups and topics of interest. Moreover, the workshop increased synergy between the disciplines and among the research staff and faculty. It was also a chance for the team to hold each other accountable to the mission, vision and goals of the Program.

Program Leadership

Leadership of the Morgan Welch IBC Program transitioned in early FY12 as Dr. Naoto T. Ueno was appointed Executive Director and Dr. Wendy Woodward as Deputy Director. Dr. Vicente Valero continues to serve as Clinical Research Director. Program Core Leaders represent the multiple disciplines that comprise the Morgan Welch Program and oversee the strategic interests of the Program while the Executive Committee, a sub-committee of Core Leaders, addresses daily operations. Together with the Executive Committee and Core Leaders of the Program, Drs. Ueno, Woodward and Valero are committed to building upon the success of the multi-disciplinary IBC clinic and laboratory.

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Financial Review

The Morgan Welch IBC Research Program and Clinic was formally established in September 2007 with a $4 million State appropriation entitled "Rare and Aggressive Breast Cancer Research". Monies from the State of Texas, renewed in 2009 and 2011, have allowed us to build the infrastructure to support a dedicated clinical and translational research team and have funded research projects in the laboratory and in the clinic, with direct impact to our patients.

FY12 has brought new challenges to the Program as we faced a reduction in appropriations via the State of Texas Rare and Aggressive Cancer program. We took this as an opportunity to better align resources towards our defined common goals as the 20% reduction in appropriations funding deemed it critical for us to become more efficient and effective in our use of resources. Our intentions with the allocation of funds were to alleviate the financial burden of projects and programs that did not support IBC goals and reallocate funds to new collaborations and seed projects. Moreover, we felt an urgent need to identify and secure other funding to support the Program so that we can leverage the state funds and other resources more effectively. The table below reflects expenditures of the Program in 2012, including clinical research and donor funds. These figures do not include individual principal investigator sponsored project funds.

	State Appropriations	Sponsored Projects	Donor Funds	Clinical Trials	Total
Personnel	$1,373,935.44	$2,842.49	$27,317.41	$184,433.06	$1,588,528.40
Non-Personnel	$239,759.18	$13,541.16	$34,577.57	$67,287.25	$355,165.16
Total Expenditures	$1,613,694.62	$16,383.65	$61,894.98	$251,720.31	$1,943,693.56

Philanthropy

We are grateful for the generous philanthropy bestowed upon the program in FY12. The Zeta Tau Houston Alpha Alumnae Association, in addition to their already established endowment fund, made a gift in direct support of IBC research. This year we have launched the First Annual Zeta Tau Alpha Houston Alumnae Association Fellowship in Inflammatory Breast Cancer Research. The call for abstracts, eligibility requirements and judging criteria were designed by the Morgan Welch IBC Program Executive Committee. The goal of the awards is to honor and recognize efforts with exceptional quality of IBC research and high impact (or potential impact) for our IBC patients. Further the monetary award will provide a means for the individual to travel to a national or international meeting to present their unique research and share their knowledge of IBC. The commitment from Zeta Tau Alpha Houston Alumnae Association has made this legacy award possible and we are ever grateful for the continued generosity and support.

ZETA TAU ALPHA HOUSTON ALUMNAE ASSOCIATION

The Zeta Tau Alpha Foundation has established an endowment through the Morgan Welch IBC Program to fund a fellowship in inflammatory breast cancer research . Winners of the First Annual Zeta Tau Alpha Houston Alumnae Association Fellowship in Inflammatory Breast Cancer Research announced on October 5, 2012 were Lara Carolina Alvarez de Lacerda, PhD and Gary Walker, MD, MPH.

In October 2011, Team Karen hosted their 3rd Annual Team Karen 10K trail run and Kids K Bike Race at Reveille Peak Ranch in Burnet, Texas. Mr. and Mrs. David and Karen Cottrell have long been supporters of IBC research through the Morgan Welch IBC Program and we are grateful for their endeavors to raise awareness of IBC in a fun and family-friendly way. The funds raised through the Karen Cottrell and Team Karen Inflammatory Breast Cancer Fund are being used as seed funding for new research projects. As such, a project led by Dr. Ricardo Alvarez, assistant professor in Breast Medical Oncology and physician in the Morgan Welch IBC Clinic, was identified to be the first such funded project. Under the mentorship of Dr. Naoto Ueno, Dr. Alvarez has begun the preclinical and cell culture analysis of the drug siltuximab, the first of two aims in his project entitled “Role of IL6 in IBC preclinical model”. The second aim of this study is to develop an animal model based on the preliminary results.

Throughout FY12, The IBC Network Foundation actively hosted “Hunt for Hope” events across the country to raise money for IBC research. The goal of the foundation is to directly fund important projects that may be unique and/or limited to types of metastatic disease and thus less likely to be supported through federal funding mechanisms. In

April of this year, the Foundation presented to Dr. Wendy Woodward, associate professor in Radiation Oncology and Deputy Director of the Morgan Welch IBC Program, a check to fund the Lori Grennan Pleural Effusion Study. This unique study will determine if we can reprogram cancer cells to stop dividing and reduce the symptoms of fluid in the lungs caused by metastatic breast cancer. This somewhat unusual outcome of breast cancer impacts many of our IBC patients and, to date, placing a tube to remove this fluid is the only specific treatment. Through our laboratory studies of these fluids we have learned they contain cancer stem cells that continually renew cancer cells as they are removed. We hope this trial of reprogramming cancer cells and halting this renewal with valproic acid will be a stepping stone to novel approaches to treat and cure metastatic disease in many organs. This trial would not have been possible without the generosity of the IBC Network Foundation and we are thankful to have the opportunity to bring this novel approach to our patients.

We would also like to extend appreciation to individuals who donated time or money to our Program. Your thoughtfulness and generosity foster support for inflammatory breast cancer research in numerous ways. We are eternally grateful and thank you for thinking of our cause.

Clinical Research and Operations

Patient care is the heart of our Program as we strive to reduce the suffering of patients with IBC. The lack of existing standard of therapy for this patient population and the disproportionate number of patients dying from this disease necessitates that we identify novel approaches to bring advancement in therapy options. For this reason, clinical trials are an integral part of our treatment approach for both newly diagnosed and metastatic disease. During FY12, the Program's clinical trial portfolio was comprised of 10 active trials, with 3 studies completing enrollment, 3 new trials activating and 4 new studies in development. Our landmark international IBC patient registry accrued a total of 59 new patients. This particular protocol is important as it is the foundation for our basic and translational research—providing the precious patient tissue and epidemiological data that will lead our understanding of the disease. In addition to the registry study, 41 patients participated in clinical studies targeted to IBC. Patient participation in these studies is important as we seek to define a new standard of treatment for our patients and improve their outcomes.

Protocol Number	Protocol Title	Principal Investigator	Activation Date	Target Accrual
2008-0372	Phase II study of Panitumumab, Nab-paclitaxel, and carboplatin for patients with primary IBC without HER2 -overexpression	Ueno	11/1/2010	40
NSABPFB-7	A Phase II randomized Clinical Trial Evaluating Neoadjuvant Therapy Regimens with Weekly Paclitaxel and Neratinib or Trastuzumab or Neratinib and Trastuzumab Followed by Doxorubicin and Cyclophosphamide with Postoperative Trastuzumab in Women with Locally Advanced HER2 Positive Breast Cancer	Valero	6/13/2011	10
2010-0696	A phase I/II study to evaluate the safety, tolerability, and preliminary efficacy of KW-2450 in combination with lapatinib and letrozole in subjects with advanced or metastatic breast cancer whose tumor over-express HER2	Ueno	6/13/2011	10
2010-0683	OAM4861g A randomized, phase II, multicenter, double-blind, placebo-controlled study evaluating the safety and efficacy of MATMAb in combination with Paclitaxel in patients with metastatic, triple-negative breast cancer	Valero	9/21/2011	15
2010-0296	A Phase II Study of TKI258 (Dovitinib Lactate) as Salvage Therapy in Patients with First Local or Distant Relapse HER2-negative Inflammatory Breast Cancer (IBC) (up to 2 prior therapy)	Alvarez	1/27/2012	33
2010-0842	A phase I/II Study of Entinostat and Lapatinib in Patients with HER2-Positive Metastatic Breast Cancer in Whom Trastuzumab has Failed	Ueno	1/10/2012	70
2006-1072	IBC International Patient Registry	Ueno	4/17/2007	500
LAB08-0199	Reactivation of Epstein-Barr Virus in Patients with Inflammatory Breast Cancer	Reuben		160
LAB04-0657	A Model of COX-2 Mediated bone Metastasis in Human Breast Cancer to include IBC cohort I	Lucci		800
LAB04-0698	Pilot Study: Correlation of Circulating tumor cells and COX-2 Expression in Primary Breast Cancer metastasis To include IBC cohort I	Lucci		800
2007-0766	A phase I-II study of R115777 (Tipifarnib, Zarnestra) plus sequential weekly paclitaxel followed by dose-sense doxorubicin and cyclophosphamide in patients with stage IIB-IIIC breast cancer Her2(-)	Valero	CNPE: 12/27/10	
2007-0818	A phase II study of neoadjuvant Lapatinib plus chemotherapy (sequential FEC75 and Paclitaxel) in women with inflammatory breast cancer whose tumors overexpress ErbB@ (Her2/neu)	Alvarez	CNPE: 12/30/2011	
2007-0448	A Randomized, Multicenter, Phase III Study Comparing the Combination of Pazopanib and Lapatinib versus Lapatinib Monotherapy in Patients with ErbB2 over-expressing Inflammatory Breast Cancer	Alvarez	Terminated: 11/2011	

The Morgan Welch Inflammatory Breast Clinic within the Nellie B. Connally Breast Center at MD Anderson was established based on our commitment to provide patient-centered team care incorporating all of the critical clinical team members from each discipline into one IBC team. This multi-disciplinary team medicine approach, combined with the broad experience and expertise that only comes from seeing and treating hundreds of patients with this rare disease, is critical to the care and outcomes for our patients. Our Board Certified clinical team consists of four breast medical oncologists, three radiation oncologists, three surgical oncologists, three pathologists, and two breast imaging radiologists. Further the clinic is supported by dedicated advanced practice nurses, registered nurses, research nurses, clinical studies coordinators and research technicians. We are not only committed to research and treatment of inflammatory breast cancer, we specialize in it. We work together to provide the best care to our patients and excel in providing the best experience for them.

In addition to our long history treating IBC at MD Anderson Cancer Center, our clinic has treated hundreds more IBC patients in just the last 5 years. Before the clinic opened, MD Anderson was seeing on average 10-15 IBC patients per year. This has since increased to 85-100 patients per year, with more than 750 patients being seen in the Morgan Welch Clinic since 2006.

Our IBC clinic is supported by world-class dedicated researchers who work together (team science) to bring new therapies to patients and to solve the mystery of this deadly disease. To aid in our understanding of this virulent disease and to improve collaboration and synergy among IBC investigators, we opened the first comprehensive research laboratory dedicated to IBC in late 2009. The team science model encourages constant communication and collaboration and allows our researchers to leverage shared resources. Moreover, the state-of-the-art facility provides a centralized core for the IBC biorepository, a collection of tissue and serum from our own IBC patients and partner centers from around the world.

The Morgan Welch IBC Research Program and Clinic is unique in its comprehensive approach to the disease. From bench to bedside, from early diagnosis to prevention of metastasis, we are working together to bring a new future to the women and men who face this disease.

Core Program Research Updates

In addition to patient care, the Morgan Welch IBC Program is committed to research at the basic and translational levels. We recognize that to be able to improve the lives of our patients through better prevention or diagnostic strategies and to be able to offer more effectives therapies, we need to study IBC and its environment at a molecular and genomic level. Moreover, by understanding the mechanisms for metastasis, we may better be able to inhibit disease progression, necessary for improving survivability. Following are updates from our key program leaders and other projects in IBC.

Diagnostic Radiology **Wei Yang, MBBS, FRCP**

Current Work:

1. Multimodality Imaging Staging Work-up for all newly diagnosed IBC patients to include same day mammography, US with protocol biopsy (as necessary), and MRI
2. Collection of tumor samples for tumor bank and related optical imaging protocols (Collaboration with DiHua Yu, MD, PhD and Rice Bioengineering on Komen Promise Grant)
3. Response monitoring with MRI and PET-CT
    a Assessment of Residual Disease (Breast)
    b Assessment of Residual Disease (Axilla)
    c Imaging Predictors (Survival)

Opportunities:

1. We should work with Pathology (Savitri Krishnamurthy) and new technology companies to investigate the feasibility of assessment core samples for tumor cellularity (virgin tumor).
2. Explore opportunities to assess functional imaging parameters with genomic information on collected tumor samples.

Epidemiology **Randa A. El-Zein, MD, PhD**

The goal of this core is to provide the epidemiological support for the Morgan Welch Inflammatory Breast Cancer Research Program. The epidemiological component includes two important areas, namely: i) Traditional Epidemiology and ii) Molecular Epidemiology. During the past year and through support from the Morgan Welch Inflammatory Breast Cancer Research Program, we were able to achieve the following:

Epidemiological Achievements

To date, a total number of 235 patients have been enrolled into the IBC Registry.

The risk factor questionnaire that includes socio-demographic information, reproductive, medical, family history, breast health history as well as detailed information on tobacco, alcohol use, occupational and environmental exposures, was completed on 100 patients [some of whom were enrolled into the study the previous year but did not complete the risk factor questionnaire]. Figure 1 summarizes selected demographic characteristics of the study population and Table 1 represents the selected risk factors associated with the disease.

Approximately 37 new patients were enrolled. A new database was successfully developed. The database can be linked to the clinical and tissue specimens to facilitate collaborations among the program investigators. In addition, the risk factor questionnaire and database have been adapted for use by the other partner sites.

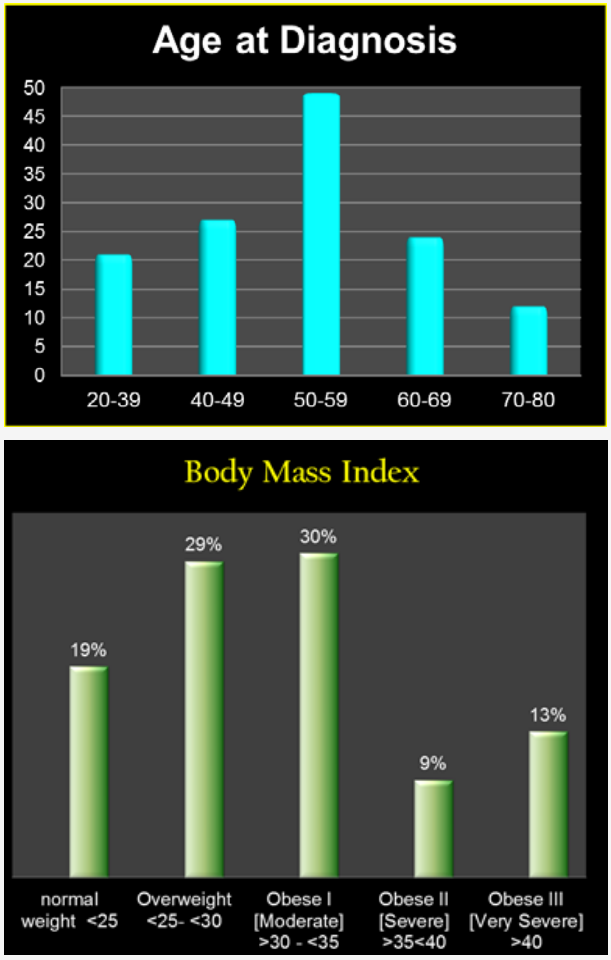

Figure 1. Selected characteristics of the Inflammatory Breast Cancer patients seen at MD Anderson.

Molecular Epidemiological Achievements

On the molecular level, little is known about the underlying molecular alterations involved in the pathogenesis of IBC. It has been suggested that the mechanisms for the induction of chromosomal damage are similar in different tissues, and therefore the extent of chromosomal damage evaluated in lymphocytes reflects damage in cancer-prone tissues. To date, we have approximately 160 patients with completed clinical and risk factor questionnaire on who blood was obtained for the molecular epidemiological studies.

We conducted a case-control study on IBC patients and age and ethnicity matched controls to assess the level of overall genetic instability in blood lymphocytes using the multi-endpoint cytokinesis-block micronucleus assay, an established biomarker for DNA damage and genomic instability. All chromosome damage endpoints were significantly higher among the IBC cases as compared to controls with a mean + SE = 5.4+ 0.15 vs.1.7+ 0.12; 8.2+ 0.14 vs. 1.1+ 0.12; 1.3+ 0.1 vs. 0.4+ 0.1; $p<0.0001$ for micronuclei, nuclear bridges and nuclear buds endpoints respectively, Figure 2.

Table 1: Selected Studied Risk Factors

Probable risk factors	Association *
Younger age at diagnosis	+++
Younger age at menarche	+
Younger age at live first birth	+
High BMI (≥30)	+++
Oral contraceptive use	+
Ever pregnant	+
Longer duration of breast feeding	+
White vs. African American ethnicity	+++
Hormone receptor status (negative)	+
Residence in Northern African countries	+

*+++Indicates relative risk >3; + relative risk >1 and <3

We furthered our investigation to compare the level of genetic instability in IBC vs. non IBC patients and to identify non-random events associated with the disease. We used G-Banding to map the specific alterations to known sites in the genome. Our data indicate that cells from IBC patients have higher levels of genetic

alterations/cell. In addition, the frequency of specific chromosome losses involving chromosome 1,3, 8 and 17 was significantly higher in IBC patients as compared to non-IBC patients, suggesting that chromosome damage in IBC is non -random event involving specific chromosomes that contain fragile sites and genes crucial to carcinogenesis.

In addition, among the triple negative IBC patients, the accumulated genetic alterations/cell were found to be significantly higher than in non-triple negative patients.

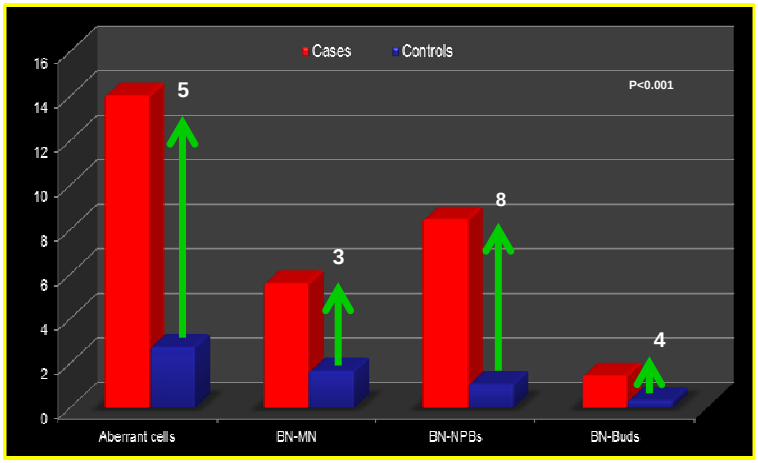

Figure 2: Distribution of genetic damage endpoints among IBC cases and controls

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BREAST MEDICAL ONCOLOGY **NAOTO T. UENO, MD, PHD**

IBC Database

The goal of the IBC clinical database is to develop an understanding of the natural history and the biology of inflammatory breast cancer and to determine the potential role of Statin.

Since last October we have generated an IBC clinical database for all IBC patients and we also conducted a retrospective chart review of patients diagnosed with inflammatory breast cancer from 1970 to 2011 and those that received treatment at MD Anderson Cancer Center.

Objectives:

- Determine the IBC long term natural history
- Defining the molecular subtypes in IBC
- Determine whether multidisciplinary treatments are effective in IBC
- Determine whether statin reduces the recurrence of primary IBC

Total number of IBC patients identified: 1214 (including 240 stage IV patients)

Preliminary results indicate that IBC shows poorer prognosis. IBC stage III patients have 5-year overall survival rates of less than 50%, and 10-year overall survival rates of less than 40%. In stage IV patients, the 5-year overall survival rate is less than 30%.

For this review, we defined multidisciplinary treatment as performing all of the following treatments—neoadjuvant therapy, surgery and radiation therapy—and in this specific order. We hypothesized that multidisciplinary treatment improves clinical outcome of IBC patients. The result showed that multidisciplinary treatment is more effective when to compared to other treatment models such as surgery first or radiation therapy prior to surgery. Further, patients who never underwent surgery showed the poorest outcome. In the Stage IV population, undergoing surgery was associated with a good prognosis. TNBC subtype bore the strongest benefit from multidisciplinary treatment.

Further, we hypothesized that statin reduces inflammatory reactions in the body and possibly reduces recurrence risk in IBC. There is some evidence that circulating inflammatory markers such as acute phase proteins and IL-6 increase risk of recurrence and mortality. There are 993 primary IBC patients in the database and of those, 109 patients were

using statin. We defined statin users as patients that have been prescribed statin at least once. The primary endpoint for our review was disease free survival (DFS) and the secondary endpoint was overall survival (OS). The median follow up time was 39.2 months for statin users and 31.4 months for non-statin users. Both showed that statin use was associated with improved DFS and OS.

From the result of our retrospective study into the role of statin in IBC, we plan to conduct a prospective randomized clinical study (See Figure 3).

Identifying IBC targets (PA11-1129: Identify the targets)

This project has multiple objectives as we seek to identify unique IBC-targets that we enable us to better address the disease. There are among 50 candidate genes.

Objectives:
To determine the clinical characteristics in patients with IBC

To determine the correlation among biomarkers including ER, PR, HER-2/neu as well as other candidate markers such as Ki67,p53,E-cadherin,EGFR, CK5/6, IGF-1/ PDGF, RhoC GTPase, WISP3,CXCR4 and VEGFR1,2,3 and so on.

To determine the correlation between clinical parameters and clinical outcomes such as local recurrence rates, distant metastasis free survival (DMFS), and overall survival (OS), and also whether the candidate makers have any independent prognosis under breast cancer subtype in IBC.

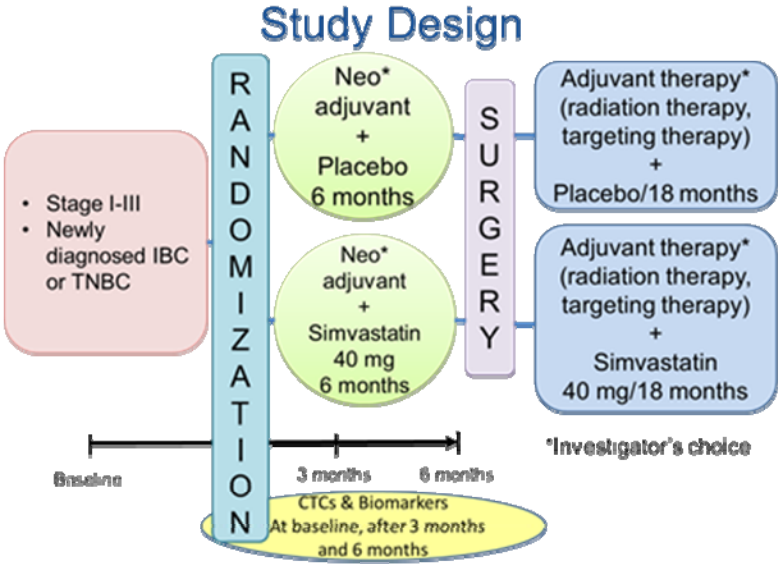


Figure 3. Clinical trial design for study of role of statin in IBC

We brushed up the IBC clinical database at MD Anderson and collaborated with Dr. Savitri Krishnamurthy to collect patient tissue samples. Using these samples, we are planning to examine the candidate genes that have potential as prognostic markers and treatment targets for IBC using IHC testing, RPPA, and mRNA arrays.

IBC mRNA project

Using the world consortium IBC mRNA data, we tried to identify the triple-negative breast cancer (TNBC) subgroups (Table 2). This project was presented at ASCO and the manuscript is being submitted to peer-reviewed journal.

Results: We found 7 subtypes for both Triple negative (TN)-IBC and TN-non-IBC. While the correspondence between our findings and those of Lehmann et al. was not perfect, there was a very significant correlation ($P<2.2x10-16$). We found no association between TNBC subtype and IBC status (P=.5023). As expected, we found that patients with IBC had significantly worse recurrence-free survival (RFS) than a comparison cohort of patients with advanced non-IBC that included not only patients with TNBC but also patients with ER-positive and HER2-amplified tumors (P=.0054). However, TNBC subtype did not predict RFS. IBC status was not a significant predictor of RFS or overall survival in the TNBC cohort. **Conclusions:** Both TN-IBC and TN-non-IBC are heterogeneous. TNBC subtypes are unrelated to IBC status. TN-IBC and TN-non-IBC have the same subtypes and clinical outcome.

ER (+) IBC project

Using the world consortium IBC mRNA data, we detected 97 different genes between IBC ER (+) and non-IBC state matched ER (+) subgroup. Based on this information, we then hypothesized that hormonal IBC-specific gene signatures can predict the poor prognosis of non-IBC ER (+) breast cancer.

	TN-IBC(%)		TN-non-IBC(%)	
Subtype 1 (M)	4	(9.7)	7	(13.2)
Subtype 2 (IM)	3	(7.3)	12	(22.6)
Subtype 3 (BL1)	9	(21.9)	8	(15.0)
Subtype 4 (BL2)	3	(7.3)	2	(3.7)
Subtype 5 (LAR)	6	(14.6)	6	(11.3)
Subtype 6 (US)	8	(19.5)	10	(18.8)
Subtype 7 (MSL)	8	(19.5)	8	(15)

Table 2. Subtypes of triple negative IBC and non-IBC breast cancers.

There are 97 different genes between IBC ER (+) and non-IBC state matched ER (+) subgroup. Using these gene signatures, we performed the cross validation of whether we could predict IBC status. Result showed that we could identify the IBC status using these gene signatures (all 4 models could identify the IBC status). However, we do not know the exact biological meaning of these 97 different genes or whether IBC ER (+) group has specific biology in comparison to non-IBC ER (+) group.

Clinically, IBC ER (+) group showed poorer prognosis than non-IBC ER (+) group. Thus, there is a possibility that this gene signature may reflect the aggressive nature of any breast cancer. Therefore, we hypothesize that the gene signature may predict the poor prognosis group in non-IBC ER (+) population.

Resource used: There are 3 public breast cancer mRNA databases.

Transbig (n=198. ER (+) n=124) Marinz (n=200. ER (+) n=155) Wang (n=286. ER (+) n=178)

These populations did not receive any systemic cancer treatments except surgery. Therefore, we can examine the natural history of ER (+) BC population.

TIG1 Project Report

Inflammatory breast cancer (IBC) is the most lethal and aggressive form of breast cancer. The molecular mechanisms for the tumorigenic and metastatic properties of IBC are largely unknown. In this study, we show that high expression of *tazarotene-induced gene 1 (TIG1)* significantly correlated with shorter survival of patients with IBC and demonstrate that TIG1 mediated cell proliferation, migration, and invasion of IBC cells *in vitro* and tumor growth of IBC cells in a xenograft animal model. We also identified receptor tyrosine kinase Axl as a functional partner of TIG1. Our data

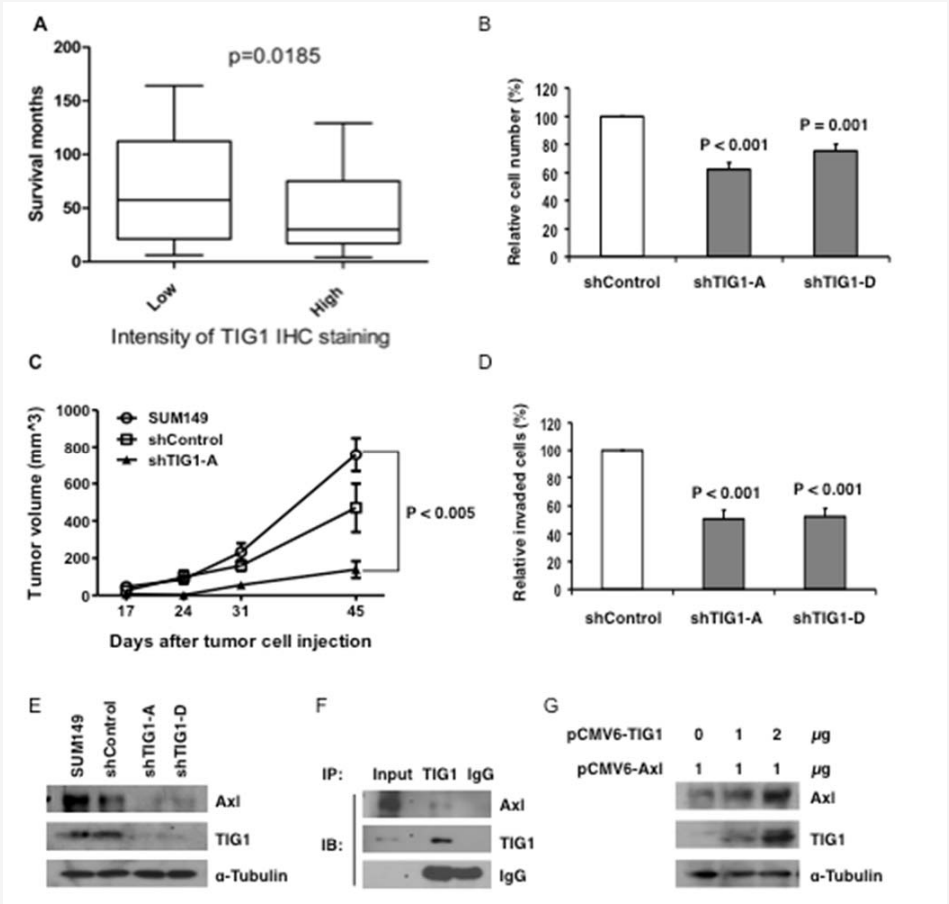


Figure 4. (A) High TIG1 expression was significantly associated with reduced patient survival duration. (B) Silencing TIG1 reduced proliferation of IBC cells. © Silencing TIG1 inhibited tumor growth in a xenograft model. (D) Silencing TIG1 reduced invasion of IBC cells. (E) TIG1 knockdown decreased the expression of Axl in IBC cells. (F) TIG1 interacted with Axl in IBC cells. (G) Ectopic expression of TIG1 increased the protein level of Axl.

showed that TIG1 interacted with and stabilized Axl by inhibiting its proteasome-dependent degradation. Indeed, Axl expression positively correlated with TIG1 expression in IBC human tissues. Last, we show that TIG1 depletion in IBC cells decreased Axl expression and inactivated NF-κB, which led to downregulation of MMP-9, indicating that TIG1 regulates invasion of IBC cells through mediation of the Axl signaling pathway. Taken together, our results link a novel tumorigenic gene, *TIG1*, to the key tumorigenic gene *Axl* in IBC. Further studies are needed to confirm that TIG1 is a promising IBC-specific therapeutic target in the treatment of patients with IBC.

SURGICAL ONCOLOGY **ANTHONY LUCCI, MD**

Our group is focused on improving outcomes for IBC patients through:

Outstanding surgical care of the IBC patient

We have shown that our local-regional control rates for IBC here at MDA are at 90%, which is a significant improvement over previously published reports from other institutions. *(see J Clin Oncol* 30, 2012 (suppl; abstr 1102)

Development of novel surgical protocols to minimize morbidity for IBC patients

We have launched a protocol to assess PET and US imaging of the axilla before and after neoadjuvant chemotherapy to determine which IBC patients can safely forego axillary lymph node removal. The data from this study should significantly improve lymphedema rates in IBC patients.

Study of disseminated (bone marrow) and circulating (blood) tumor cells in IBC patients

We published a report in the *Lancet Oncolo*gy in 2012 (Jul;13(7):688-95) demonstrating that presence of circulating tumor cells predicted decreased relapse-free and overall survival in breast cancer patients. This model is concurrently being tested in IBC patients, and the preliminary data is highly significant (See Figure 5). We plan to use this information to assess response to novel therapies for IBC in the near future.

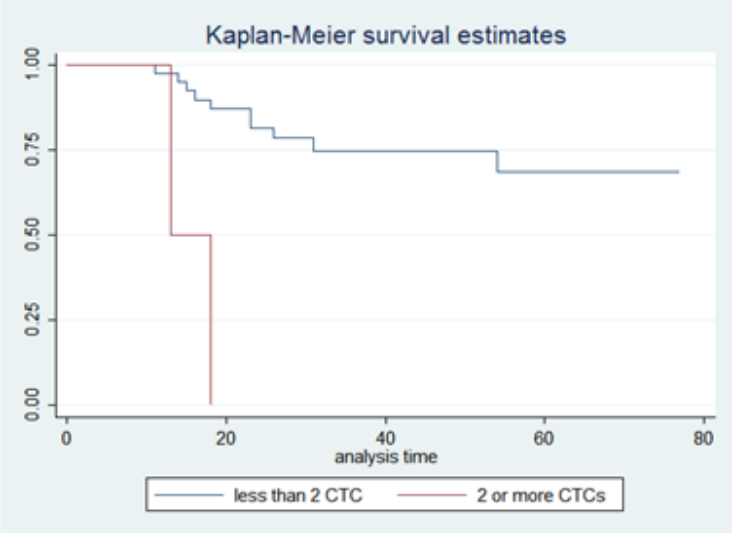


Figure 5. Circulating tumor cells and RFS in IBC patients
CTC = Number of Circulating Tumor Cells, OS = Overall Survival
P = 0.0022

Identification of novel targets on disseminated and circulating tumor cells in IBC

We have developed a microfluidic chip system to assess CTCs in IBC patients. This system allows us to perform FISH analysis on captured CTCs, and then document and morphologically evaluate the CTCs that have amplification of HER2 (see Figure 6a & b). Interestingly, we found that a significant number of patients with HER2 negative primary tumors had HER2-amplified CTCs. We hope to use this information for novel treatments of HER2-negative patients with IBC with anti-HER2 agents. This data was presented

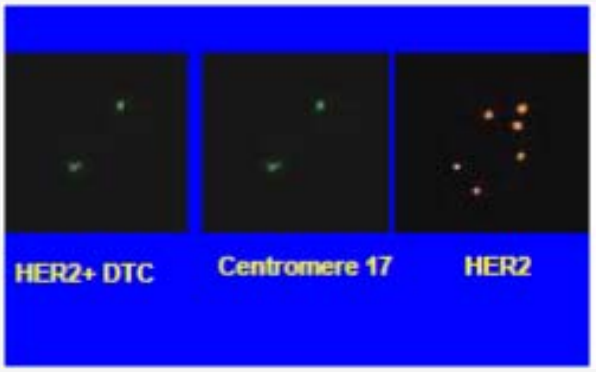


Figure 6a: Illustration of a CK + and CD45- CTC. Note the Numerous orange signals indicating increased copy numbers of HER2. The primary tumor was negative for HER2 gene amplification.

at ASCO 2012 (*J Clin Oncol* 30, 2012 (suppl; abstr TPS10631), and is now submitted for publication.

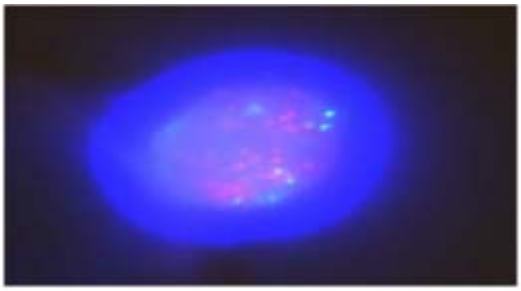


Figure 6b: Illustration of a disseminated tumor cell (DTC) in bone marrow showing increased copy numbers of HER2 as evidenced by increased orange signals. The primary tumor was **negative** for HER2 gene amplification.

Assessment of the role of the CXCR4/SDF-1 axis in IBC and development of novel targets for metastasis prevention

We published two reports evaluating a novel CXCR4 inhibitor, including one in a xenograft mouse model of IBC (*Clin Exp Metastasis*. 2010 Apr;27(4):233-40). We are now embarking on a collaborative project with Dr. Steven Millward to determine if tyrosine sulfation of CXCR4 (which significantly enhances enhanced affinity for its ligand SDF-1) results in enhanced growth, migratory signaling, and increased metastatic potential of IBC cells.

Development of a novel predictive *in vitro* assay for anti-cancer drug selection

We selected a rare subpopulation (0.01%) of SUM149 cells (triple-negative IBC subtype) which is capable of surviving body-like metabolic challenges (lack of Gln, Glc). These cells are highly adaptable, anchorage-independent, resistant to doxorubicin, paclitaxel, celecoxib, etc., and metastatic in nude mice. This work was published in 2012 in *PLoS One* (7 (5):e36510. Epub 2012 May 3). We plan to use this model to develop cancer subtype-specific in vitro models for evaluating new therapies against IBC. Optimally, we will utilize FDA-approved drugs to speed implementation into the clinic, and align these *in vitro* models with clinical IBC studies.

RADIATION ONCOLOGY AND STEM CELL BIOLOGY **WENDY WOODWARD, MD, PHD**

Many lines of evidence implicated IBC as a disease of stem cells. It is enriched for triple negative breast cancers which are less differentiated cancers. It is associated with high rates of metastasis and recurrence believed to be mediated by cancer stem cells. Stem cell markers such as ALDH1 and EZH2 are prognostic in IBC patient outcomes. All gene expression signatures developed from mammary gland stem cells are expressed in IBC patient samples. For this reason understanding and targeting stem cell biology is a critical core in the IBC research program.

Our core has several major projects dedicated to changing outcomes in IBC through understanding stem cell biology. First, IBC is characterized by radiation resistance with high rates of local failure after radiation therapy. While aggressive radiation treatment regimens have been successful, a radiosensitizing agent to promote better outcomes with less toxicity is highly desirable. Our screening program has developed a stem cell specific radiation resistance screen through which we have a top promising radiosensitizer to take forward in clinical trials. Specifically, laboratory and clinical data suggests statin use radiosensitizes IBC stem cells. We are actively pursuing additional studies to elucidate the mechanism of this effect. In addition, we have identified several dietary flavonoids that target IBC stem cells though inhibition of b-catenin in breast cancer stem cells. Second, while stem cell targeting agents are needed, it is critical we understand the plasticity of breast cancer stem cells to design successful clinical trials. We have demonstrated that commonly used medications that inhibit histone deacetylase and appear to target cancer cells can promote stem cell properties from single non-stem like cells. These data highlight the critical need to assess therapies for their impact on cancer cell subpopulations and demonstrate the need for better assays of response in micrometastatic disease. Finally, without clear evidence for IBC specific biology in numerous studies of the tumor cells from IBC patients, we are examining differences in the breast tissues of patients that develop IBC and demonstrating the effects of normal cells on IBC tumor progression. Specifically, we have demonstrated for the first time that IBC skin

invasion is promoted by normal cells from the bone marrow, mesenchymal stem cells through an EGFR targeted mechanism. Further, the normal breast in IBC patients is distinct from non-IBC patients with spatially distinct stem cells throughout the normal tissues, and infiltrating immune cells that can been picked up in biopsies even prior to the diagnosis of IBC. We are working to demonstrate the relationship between stem cells, mesenchymal stem cells and immune cells in preparing the breast to mediate the IBC phenotype and propose novel treatment of the normal tissue may lead to better prevention and treatment.

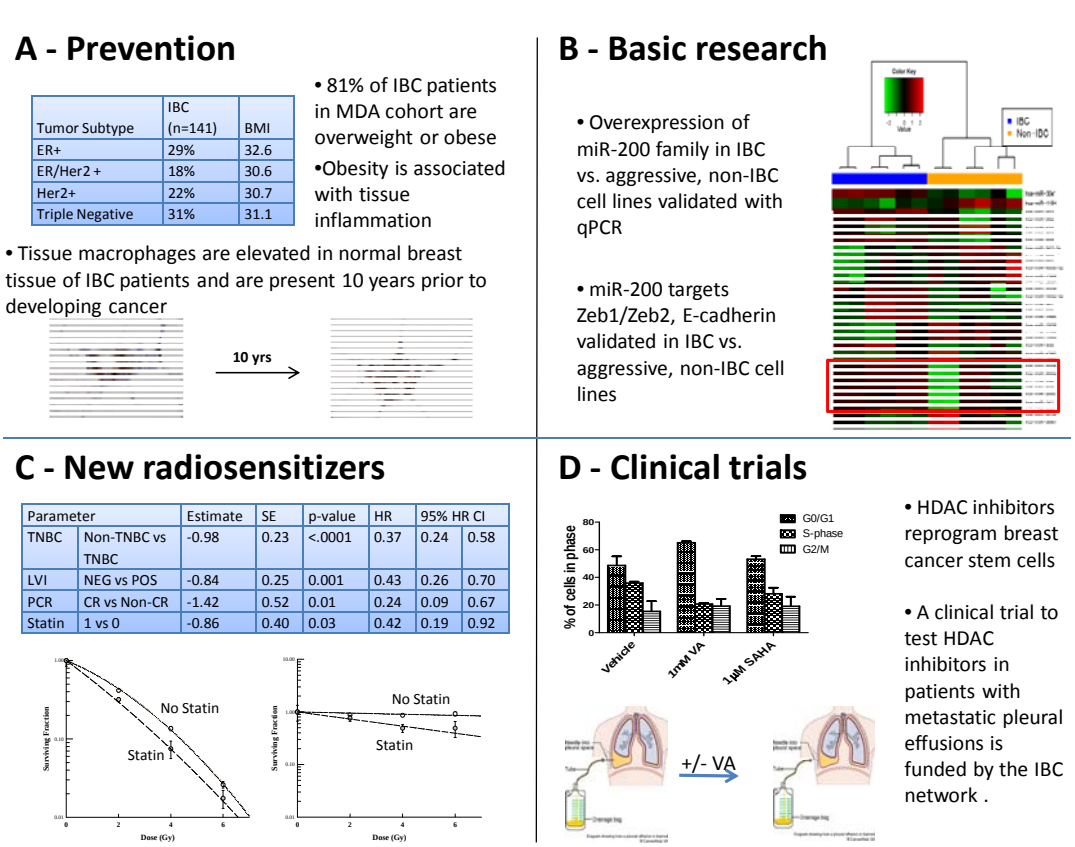


Figure 7. A- Prevention of IBC: Studying the normal tissue adjacent to cancers is revealing clues as to what changes occur prior to the cancer that may contribute to the IBC phenotype. Significant differences are appreciated in the spatial distribution of stem cells and in the infiltrate of immune cells such as macrophages. Left image is Bx 10 years prior to Dx BMI 23.5 and Right image is Bx 10 years prior to Dx BMI 35.5. B- Basic Research in IBC: comparing IBC and non-IBC cells lines cultures in stem cell promoting culture vs. standard culture revealed miRNA that are differentially expressed in IBC. These targets are being studied to develop therapeutic strategies and to understand the unique biology of IBC. C – New Therapies: SUM149 treated in 2D cultures (left) and stem cell promoting 3D cultures (right) reveal a dramatic sensitization of stem cells with the use of statins. In IBC patients on statins the risk of local failure after radiation was reduced more than half. D. IBC is a disease of stem cells and our work clearly demonstrates that the stem cell state is in equilibrium with differentiated cells. Harnessing this equilibrium we are using HDAC inhibition to halt destructive differentiated cells in metastases and promote quiescent stem cells to improve outcome.

In conclusion, great progress has been made in prevention, basic research, radiosensitizers and stem cell plasticity leading to two upcoming trials for stage III and stage IV IBC patients. Our work has demonstrated the importance of the microenvironment in mediating the IBC phenotype and opened the door to greatly expanding targets for treatment and prevention.

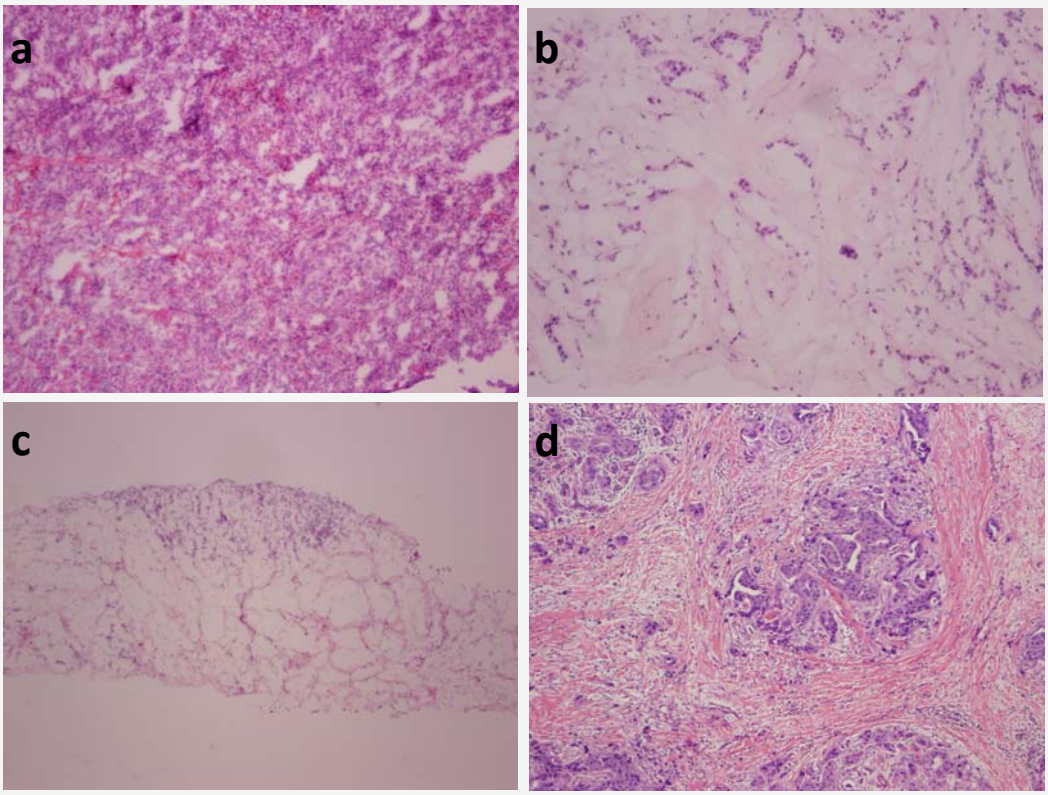
PATHOLOGY **SAVITRI KRISHNAMURTHY, MD**

Our group is focused on addressing all pathology related issues for the Morgan Welch Inflammatory Breast Cancer Research Program and Clinic.

Clinical Care:

We have provided highest quality of patient care related to pathology work up of patients seen in the IBC clinic. This includes providing the basic tissue diagnosis and expediting the work up of the prognostic and predictive markers of

any type of specimens obtained from IBC patients such as core biopsy, skin punch biopsy and mastectomy.



8a, b, c. Examples of frozen sections of core biopsy of inflammatory breast cancer.
8d. Example of invasive tumor with patchy distribution, demonstrating challenge of bio-banking inflammatory beast cancer.

Tissue Registry:

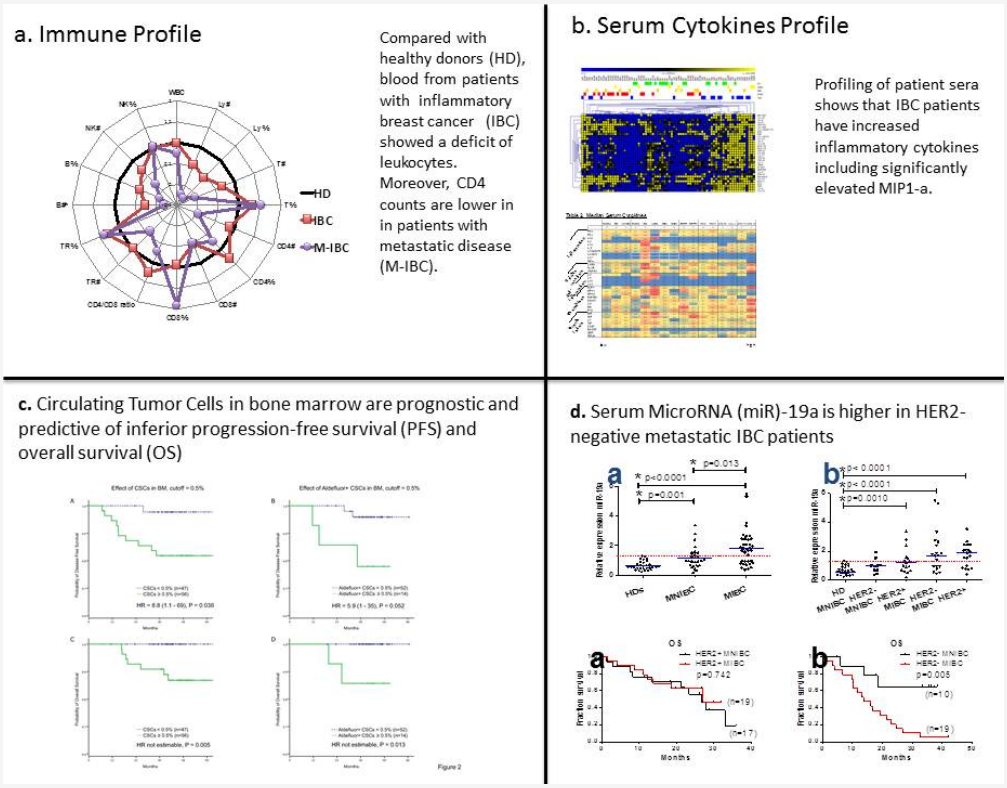
We have played a key role in the maintenance and operation of the IBC tissue registry which forms a key resource for all current and future research related to IBC. We established the standard of operations for tissue collection and storage of tissues in the IBC tissue registry including collection of frozen tissue (snap frozen), optimal cutting medium (OCT) frozen , RNALater and formalin fixed and paraffin embedded tissue of breast core biopsy, skin punch biopsy and mastectomy specimens of patients with IBC. In addition to creating the tissue resources, we are also involved with quality assurance and disbursal of high quality tissue for research of IBC. The quality assurance of the collected tissues forms a key component to establishing the tumor cellularity of the collected tissue and ensuring the presence of invasive tumor as collected IBC tissue may have only intraductal carcinoma or lymphovascular tumor emboli. Moreover, we have developed an additional resource of collecting paraffin blocks in patients who underwent surgery outside of MD Anderson as cohort II of the registry protocol.

We have created tissue microarrays of pre-chemotherapy and post-chemotherapy tissues which forms an important resource for protein expression and biomarker research of IBC.

Ongoing Work:

- We are actively studying to understand all the issues related to bio-banking of IBC pre-chemotherapy tissues. This would add immense value to selecting optimal quality assured tissue for genomics and proteomics projects from the available tissue in the registry for obtaining key answers for the distinction of IBC from non-IBC.
- We are investigating potential modalities such as optical imaging for immediate assessment of the core biopsies to improve our ability to collect high quality tissue with optimal cellularity.
- We are working to understand all pathology related issues of IBC, in particular, methods to ascertain residual burden of disease in a more accurate and reproducible fashion.
- We are actively creating resources to test and validate potential biomarkers of interest identified by genomics and proteomics projects on frozen and fixed tissues to identify markers that can be useful for diagnosis, treatment and response to therapy of IBC.

IBC is a rare and aggressive breast cancer whose etiology is unknown and genetic profiling has not identified differences between IBC and non-IBC. Earlier work by others has suggested that viruses in particular, the Mouse Mammary Tumor Virus MMTV), may play a role in the etilogy of IBC. As viruses are known to induce an immune response by the host, Dr. Hui Gao (Research Scientist) studied the immunology of IBC and non-IBC patients. Her initial studies have identified that IBC patients have lower total leukocytes (WBC) and lower CD4+ T-helper lymphocytes (Fig 9a).



In addition to cellular immunophenotypes and function, Evan N. Cohen (Pre-doctoral Candidate) assessed the presence of inflammatory proteins in the serum of IBC and non-IBC patients. He showed that IBC patients have increased levels of inflammatory cytokines (IL-1, IL-6, and TNF-a) and chemokines such as MIP-1a and IL-8, compared with levels in sera of non-IBC patients (Fig 9b). In the next year, we propose to identify a cytokine/ chemokine profile that is unique to IBC patients.

Circulating Tumor Cells (CTC) is a major area of interest in my laboratory. Antonio Giordano, MD, PhD (Visiting Scientist from the University of Naples "Federico II", Naples, Italy) and Dr. Hui Gao have been studying the predictive and prognostic values of CTC in breast cancer. Dr. Giordano has shown that although CTCs with an epithelial phenotype are not very prognostic in patients with HER2 over amplification, CTC undergoing EMT (EMT-CTC) are predictive of clinical outcome (Fig 9c). Dr. Giordano received the ASCO 2012 Young Investigator Award from Conquer Cancer Foundation and the 2012 AACR-Bristol-Myers Squibb Oncology Fellowship in Clinical Cancer Research awards to expand his initial work on CTCs.

Finally, because the limited availability of tissue from IBC patients, we are working on serum biomarkers that will allow repeated sampling for diagnosis as well as for the monitoring of patients on therapy. Simone Anfossi (Pre-doctoral Candidate) has been working on the isolation and profiling of microRNA (miRNA) in sera of breast cancer patients (Fig 9d). He has identified one miRNA, miR-19a, that differentiates metastatic patients from those with locally advanced disease and is able to differentiate between HER2+ and Her2- metastatic IBC patients. Current work is underway to establish a miRNA profile for IBC and non-IBC patients.

These studies would not have been possible without the support and dedication of our patients, support laboratory personnel (Sanda Tin, Qiong Wu and Raul Josh Garza), clinical personnel (physicians, nurses and research), and funding from the Morgan Welch IBC Research Program and Clinic through the Texas Rare Disease Grant.

Patients with TNBC are negative for estrogen receptor, progesterone receptor, and low for HER2 expression, and have poor prognoses because tumors often metastasize, leading to death. Preventing metastasis as well as inhibiting the tumor growth, is crucial to improving the prognosis of TNBC. We previously showed that patients with ERK2-overexpressing TNBC were at higher risk of death than those with low-ERK2-expressing tumors. The MEK-ERK pathway has not been explored as a potential therapeutic target of TNBC metastasis. Interestingly, when we treated the TNBC cells lines with the MEK1/2 ATP uncompetitive kinase inhibitor (selumetinib), it did not reduce cell viability in a two-dimensional (2D) cell culture. However, in a three-dimensional (3D) cell culture model, selumetinib changed the mesenchymal phenotype of TNBC to an epithelial phenotype. Cells undergoing epithelial-mesenchymal transition are well known to potentially contribute to the metastatic process and have increased ability to form mammospheres. Based on these observations, we tested the hypothesis that targeted inhibition of the MEK-ERK pathway by selumetinib prevents lung metastasis in TNBC.

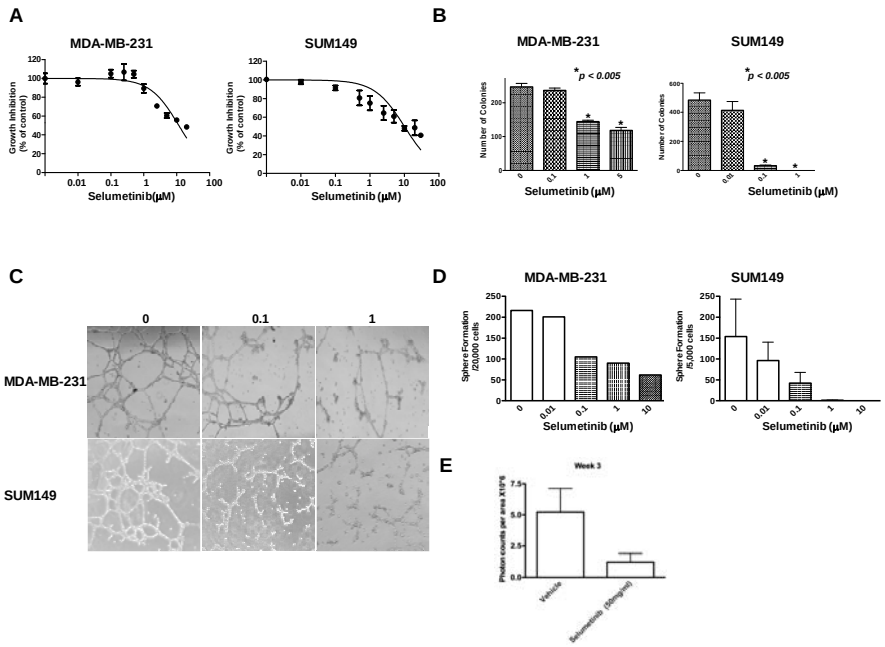


Figure 10. **The MEK inhibitor selumetinib (AZD6244 – ARRY-142886) prevents lung metastasis in a triple-negative breast cancer (TNBC) xenograft model.** ***A,*** Effect of selumetinib on cell proliferation was assessed by WST-1 assay and both TNBC (MDA-MB-231 and SUM-149) cells were resistant to selumetinib. ***B,*** soft agar colony formation of TNBC cells treated with selumetinib exhibited significant inhibition of anchorage-independent growth, an indicator of *in vivo* tumorigenicity ***C,*** TNBC cells grown in a 3D-cell culture treated with selumetinib changed from a mesenchymal phenotype to an epithelial phenotype. ***D,*** mammosphere formation presented as the average number of spheres per 20,000 and 5,000 cells plated was reduced after treatment with selumetinib ***E,*** TNBC xenograft model established with MDA-MB-231–LM2-4175 was treated with selumetinib showed significantly fewer lung metastases than did mice injected with a vehicle.

Next, TNBC cell lines treated with selumetinib, showed inhibition of anchorage-independent growth, an indicator of in vivo tumorigenicity ($P<0.005$), decreased CD44+CD24-/low (breast cancer stem cells) expression, aldefluor activity and mammosphere forming efficiency. Most importantly, mice treated with selumetinib formed significantly fewer lung metastases than did control mice injected with a vehicle ($P<0.05$, 2-sided t test.) Our data shows that the MEK inhibitor can inhibit breast cancer stem cells suggesting that our findings have clinical implications for the use of MEK inhibitors in the prevention of metastasis.

SURGICAL ONCOLOGY—RESEARCH **BALRAJ SINGH, PHD**

Tackling Tumor Heterogeneity to Develop Combination Therapies for IBC

An aggressive disease like IBC is characterized by a tremendous cellular heterogeneity, which poses serious difficulties in therapy development. How do we choose therapies out of hundreds of available drugs that have been developed against a variety of "therapeutic targets", which will be effective against the heterogeneous disease? To address this problem, we have decided to focus on the most adaptable subpopulation of cancer cells. We have evidence that in

vitro selection for metabolic adaptability selects rare (<0.01% in population) cancer cells present in SUM149 cell line (triple-negative IBC) that are highly tumorigenic and metastatic in nude mice. We observed metastases to lungs, liver, brain, and skin (Singh et al., PLoS ONE, 2012). Our study revealed a linkage between metabolic adaptability and structural adaptability in metastatic cancer cells, which can be exploited for tackling tumor heterogeneity in IBC.

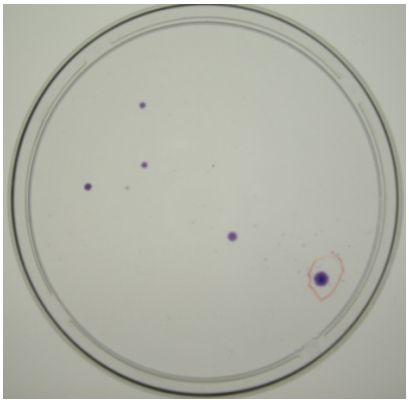


Figure 11. Selection of metabolically adaptable subpopulation of IBC cells. We plated 0.5 million SUM149 cells in a culture medium lacking glutamine. The rare cells that survived and proliferated to yield colonies in 5 weeks were stained with crystal violet dye.

Inherent receptor/ligand interactions that can occur on the surface of tumor cells can act as a dynamic molecular address that can enable targeted delivery of drugs and imaging agents to tumors (Arap et al., 2004). We hypothesize that such molecular addresses exist within inflammatory breast cancer (IBC), and can be exploited for ligand-based imaging and early detection. To this end, we have devised two research arms that will concurrently characterize the receptor/ligand interactions within IBC via peptide-based phage display. The first research arm will utilize a combinatorial peptide library (consisting of more than 1x1010 unique 7-mer or 8-mer peptides) to epitope-map the circulating pool of human antibodies elicited against tumors in IBC patients. Using 454-based sequencing, we will be able to rapidly identify thousands of peptide motifs from multiple IBC patient samples, in an attempt to identify those peptides that reflect tumor-associated antigens within this disease. Our second approach characterizes a known receptor target (glucose regulating protein-78, GRP78) expressed on breast tumor cells, including IBC (Figure 12). To date, we have characterized a peptide (WIFPWIQL, amino acid sequence) that specifically binds GRP78 protein and are currently testing the efficacy of this peptide to target GRP78 expressing IBC tumors in vitro and in vivo (see Figure 13).

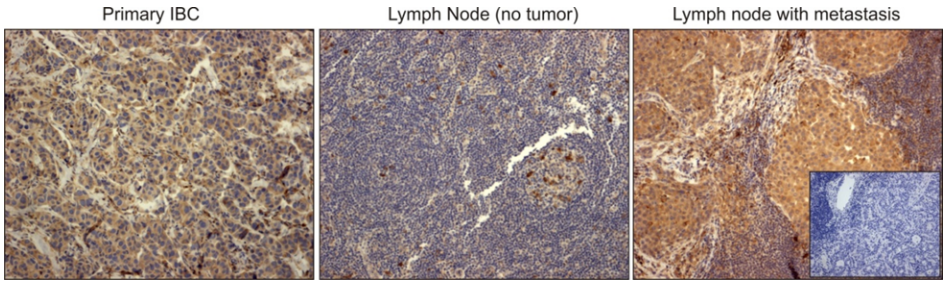


Figure 12. Expression of GRP78 is elevated during progression of human IBC. GRP78 expression was detected by immunohistochemistry in primary (left panel) and secondary (right panel) IBC tumors. Tumor-free lymph nodes (middle panel) displayed minor expression of GRP78 in the germinal center. *Right panel inset*, metastasis-positive lymph node stained with an isotype antibody control.

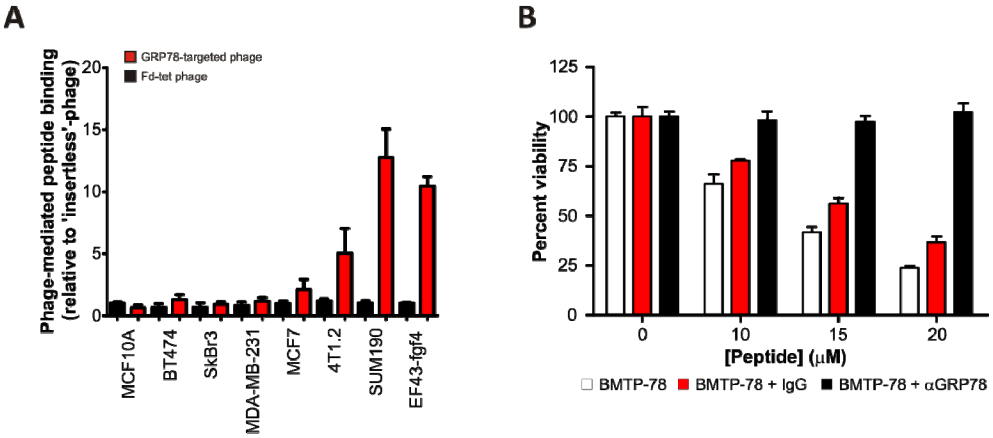


Figure 13. The GRP78 homing peptide, WIFPWIQL, mediates cell binding, internalization and homing towards IBC cells. A) Phage displaying the WIFPWIQL peptide (compared to no peptide control, Fd-tet) bound to IBC cells and metastatic breast cancer cells with high affinity compared to non-IBC cells. **B)** Cell viability was assessed using a WST-1 assay on 4T1.2 cancer cells following 16 hours treatment with BMTP78 (a WIFPWIQL-peptide drug conjugate). BMTP78 killed aggressive breast cancer cells in a dose defendant manner, which could be abrogated in the presence of a GRP78 neutralizing antibody.

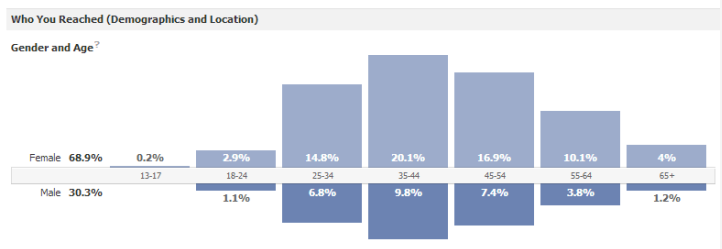
Program Outreach

The Morgan Welch IBC Research Program and Clinic is conscious of the importance of spreading awareness and understanding of inflammatory breast cancer and our unique role to lead the dialogue with patients, healthcare professionals and the general public. It is through outreach and social media that we are able to advocate for learning of disease symptoms and presentation and the appropriate multi-modality treatment approach for patients. Moreover, it is an opportunity for us to disseminate new knowledge from research so that patients, family and healthcare providers are apprised of the latest findings. Ultimately, outreach grants us the occasion to be in touch with the humanity of the disease, to be with and work with the patients and the people we are fighting for everyday.

Facebook

facebook.com/inflammatorybreastcancer

In September of 2011, we launched the Morgan Welch IBC Program Facebook page to engage patients and the community in a dialogue of all things inflammatory breast cancer. Through our page we have shared our position regarding the value of a multi-disciplinary team approach to IBC care and the latest breaking research news in breast cancer. Our Facebook wall has been a place for patients to ask questions and get answers from the experts. Moderated by Dr. Naoto Ueno, Dr. Wendy Woodward, Danielle Walsh (Program Manager) and Summer Jackson (Clinical Studies Coordinator), the page has grown to more than 600 likes. In FY12 alone, our content was seen by more than 28,000 people through individuals visiting our page or seeing our posts through sharing. Our total daily impressions for any content associated with our page was 551,137 impressions. The demographics for our followers include both men and women ages 18 to >65 years, with a majority being women between the ages of 35-54. Moreover, our reach is international, with followers from 39 different countries. Facebook has provided a dynamic forum for us to exchange ideas and best practices, acknowledge our Program's and others' achievements , and to engage in conversation with our patients, advocates and others in the health care community. It will continue to be a valuable resource for the program.



Twitter

twitter.com/inflammatorybca

Prior to our Facebook page launch, the Morgan Welch IBC Program joined Twitter, a real-time information network known for short, 140-character tweets of information. This unique social media tool fosters dynamic exchanges of information that have the potential to spread exponentially across the Twitter community. We use Twitter in multiple ways—as a direct outlet for Facebook updates, to share commentary or the latest news as it breaks, to promote open clinical trials, and to engage patients, advocates and other health care professionals in dialogue. To this end, we established and registered #ThinkIBC as an official healthcare hashtag to facilitate conversations and information about inflammatory breast cancer. Twitter has been a powerful resource for reinforcing the MD Anderson brand and for marketing our unique program. We share tweets from other MD Anderson programs including Physician Relations, Professional Oncology Education, and Cancer Frontline and from individual faculty experts, fostering the spirit of community and collaboration. With each tweet, retweet and reply, the Morgan Welch IBC Program is building and strengthening relationships with those we serve.

5th Anniversary Celebration

October 2011 marked the fifth anniversary of the opening of the Morgan Welch IBC Clinic at MD Anderson, the first multi-disciplinary clinic in the world dedicated to this rare disease. To honor this occasion, we shared a series called “5 for 5” via Facebook and email to highlight program accomplishments over the past five years. Weekly in October, members of the program cited what they believed to be our top five accomplishments. Jie Willey, Administrative Director of Protocol Research, shared these top five:

1. Leading center for International Tissue and Serum Registry for IBC.
2. Great communication and team effort towards translational and clinical research in IBC.
3. Large clinical trial portfolio focused on IBC.
4. Willingness of our graceful patients to volunteer to participate in IBC clinical trials.
5. Dedicated research staff and investigators conducting IBC clinical trials.

In appreciation for all who founded our program and helped us to build our program over the past five years, we hosted a celebration of thanks at Treebeards in downtown Houston. The event was attended by Program faculty, staff and our dearest friends and advocates from all over the country. Executive Director Naoto Ueno gave remarks and Danielle Walsh read from a letter sent by Massimo Cristofanilli, both applauding our efforts and motivating all to continue our work on behalf of our patients.

We also hosted a more intimate IBC Ambassador Luncheon as an expression of thanks to the individuals who contribute time and effort to promoting the Morgan Welch Program and our research in the community. A slide presentation reflected the five years of Program history and the memories that have been made thus far. Dr. Naoto T. Ueno, Executive Director was the speaker and we were treated to guest appearances by Dr. Ronald DePinho, MD Anderson President, Dr. Tom Buccholz, Division Chair-Radiology and Special Advisor to the IBC Program and Dr. Gabriel Hortobagyi, Department Chair - Breast Medical Oncology. Asfaneh Keyhani, IBC Lab Manager, took interested guests on a tour of our multi-disciplinary laboratory.

Pink Ribbon Parade

The Morgan Welch IBC Program sponsored a pink ribbon in the Breast Health Collaborative of Texas' Pink Ribbon Parade, a public event meant to raise awareness of breast cancer. The design of our one-of-a-kind ribbon honoring patients with IBC was commissioned to local Houston artist Liz Conces Spencer. Ribbons made their debut at a kick-off party in front of City Hall in Houston with Mayor Anise Parker and other Parade sponsors including Pink Ribbons Project, Baylor College of Medicine, and The Rose. The ribbon sculptures then paraded around Houston-area October Breast Cancer Awareness events and were seen at Tour de Pink and the Susan G. Komen Race for the Cure.

October Inflammatory Breast Cancer Awareness Month

Earlier in the year, the State of Texas declared October 2011 as Inflammatory Breast Cancer Awareness Month and as such, the Morgan Welch IBC Program sought to actively bring awareness to our cause. We hosted IBC information booths at the Breast Health Summit in Houston and at Alvin Octoberfest at Alvin Lutheran Church. Program members and advocates represented our group in teams at Susan G. Komen –Houston Race for the Cure, the 3rd Annual TeamKaren 5K and Fun Run, and IBC Network Foundation's Hunt for Hope. In conjunction with the 94.5 The Buzz's Rod Ryan Show's Boobs Rock events benefitting MD Anderson, we handed out IBC literature and free gifts to more than 300 people and Danielle Walsh was invited to represent Dr. Anthony Lucci in on-air interview with the popular morning radio show. Finally, we were honored to attend the annual Zeta Tau Alpha Think Pink! Luncheon supporting our Zeta Tau Alpha Houston Alumnae Association Fellowship Award in IBC.

The Morgan Welch IBC Program steadfastly seeks opportunities to educate and empower patients, healthcare providers and the general public with the knowledge and understanding of IBC. By partnering with the community, we can enhance the future for all IBC patients.


THE
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INFLAMMATORY BREAST CANCER
RESEARCH PROGRAM & CLINIC

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