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Working Together to Conquer Breast Cancer:
A Report to the Lynn Sage Cancer Research Foundation

Progress reports from investigators whose research benefited from funding from the Lynn Sage Cancer Research Foundation in fiscal year 2013

February 2014

Philanthropy That Inspires

The Lynn Sage Cancer Research Foundation (LSCRF) is a pivotal partner of Northwestern Memorial Hospital and the Robert H. Lurie Comprehensive Cancer Center of Northwestern University. Every accomplishment generated in the laboratory, in a classroom, at a bedside stems from our deep commitment to understand and eradicate breast cancer.

We are pleased to mark another year of initiatives made possible by your spirited dedication to the memory of Lynn Sage and to all who struggle to overcome a diagnosis of breast cancer.

The following report provides updates on the research efforts supported by your philanthropy in fiscal year 2013. Our continued critical inquiries and investigations of a wide range of breast cancer-related questions bring us ever closer to a future filled with stronger promise for patients fighting the disease. With your inspirational partnership, we move closer to the day when understanding is absolute, practice is exact, and struggles to combat and survive breast cancer are stories of an era past.

Project #1: *Effects of Weight Loss on Breast Density Using Both Digital Mammography and Breast MRI* (Grant awarded in fiscal year 2010)

Principal Investigator
Dr. Nora Hansen, MD, director of the Lynn Sage Comprehensive Breast Center, Northwestern Memorial Hospital; associate professor of Surgery, Surgical Oncology Division, Northwestern University Feinberg School of Medicine

Background
High breast density is one of the strongest known risk factors for breast cancer. In fact, the results of a meta-analysis of eight cohort studies demonstrated a five-fold greater risk of breast cancer for women with the highest breast density compared to those women with the lowest breast density. Curiously, obese patients have a higher rate of breast cancer even though mammography results indicate that they have lower breast density. This suggests that mammographic imaging may not be as accurate in obese patients. Breast MRI has been shown to be helpful in identifying abnormalities in patients with dense breasts and may be a more accurate way to measure breast density since it measures by volume rather than two-dimensionally.

Northwestern Memorial Hospital and the Robert H. Lurie Comprehensive Cancer Center of Northwestern University proudly share an extraordinary partnership with The Lynn Sage Cancer Research Foundation (LSCRF), soon to mark a third decade of inspiring evolution. The role LSCRF plays in bringing novel research initiatives to fruition is pivotal in generating a future of hope in the spirit of Lynn Sage.

We are pleased to share in this report the progress made possible in fiscal year 2013 by your ongoing philanthropy. The work of these talented investigators carries a promise of powerful possibilities in preventing and fighting breast cancer. Their findings – supported by your dedication – move us ever closer to a future in which understanding is exact, treatments and therapies are safe and effective, and stories of survival dominate.

Project #1: ***Investigating the role of a novel THAP11-HCF1 Complex in Human Breast Cancer*** (Grant awarded in fiscal year 2013).

Principal Investigator
Debabrata Chakravarti, PhD, Professor, Department of Obstetrics and Gynecology, Division of Reproductive Biology, Northwestern University Feinberg School of Medicine

Background
Breast cancer is a complex disease that involves transcriptional regulation. Dr. Chakravarti's team has discovered a novel transcriptional regulator termed THAP11, which is expressed in breast cancer cells. They hypothesized that THAP11 may play a role in breast cancer cell proliferation.

Summary of Progress to Date
The team started by analyzing expression of THAP11 and its partner protein ZNF143 in non-tumorigenic, tumorigenic and metastatic breast cancer cell lines to determine whether there is any difference in THAP11 and ZNF 143 protein expression. THAP11 is expressed at a high level in the metastatic breast cancer cell line MDA MB231. Subsequently, they developed shRNA construct to knock down THAP11 and ZNF143 expression and analyzed their expression. Under such knockdown condition, THAP11and ZNF143 regulates proliferation of metastatic breast cancer cell growth, suggesting that these two proteins may have roles in breast cancer that are yet to be discovered.

Project #2: ***A Novel Near-IR Flourescent Porpyrazine to Improve Breast Cancer Surgery Outcomes through Intraoperative Real Time Assessment of Breast Tumor Margins*** (Grant awarded in fiscal year 2013).

Principal Investigator
Brian Hoffman, Professor, Department of Chemistry, Northwestern University, Weinberg College of Arts and Sciences

Background
The success of breast cancer surgery is directly related to the ability to remove tumors completely. There is a tremendous need for new methods of "seeing" tumor deposits during surgery that are invisible to the naked eye. Techniques that make the tumor cells fluorescent (in situ fluorescence imaging) represent an important emerging cancer detection method. We have identified a novel lead porphyrazine macrocycle (pz 247) as an ideal candidate for real-time imaging of tumor margins during breast cancer surgery. Injection of pz 247 into the vein 24-48 hours before surgery results in the preferential accumulation of the drug in tumors, which then emit harmless near-infrared (NIR) light that can be detected during surgery. Our preliminary data confirms that this system detects tumors at the margins of breast tumors. Our aims: 1) to optimize the synthesis and formulation of pz 247 for toxicology and for infusion into humans to support an FDA application; 2) to establish the minimal effective dose and evaluate the sensitivity of detection of pz 247; and 3) to evaluate the safety and tolerability of pz 247.

Summary of Progress to Date
The replacement of X-ray mammography with MRI for breast cancer screening is highly

desirable, but the absence of a tumor-specific molecular contrast agent that would highlight tumor tissue regardless of its apparent similarity to surrounding healthy tissue, limits MRI efficacy. We earlier initiated the development of conjugates as potential MRI contrast agents. One of these conjugates was taken up by tumor cells *in vitro,* but poor synthetic yields precluded further development and testing *in vivo.*

The team has now achieved the design, synthesis, and characterization of novel second generation conjugates. We find that these compounds indeed exhibit excellent cellular uptake and MR contrast enhancement both *in vitro* and *in vivo.* These findings serve as the basis for going forward under Lynn Sage support with extensive *in vivo* MRI studies in athymic nude mouse tumor models.

Project #3: *A Phase II Trial of Cabergoline in the Treatment of Metastatic Breast Cancer* (Grant awarded in fiscal year 2013).

Principal Investigator
Virginia Kaklamani, MD, Associate Professor, Department of Medicine, Division of Hematology-Oncology, Northwestern University Feinberg School of Medicine

Background
The purpose of this Phase II trial is to determine the efficacy and safety of cabergoline in women with metastatic breast cancer, whose cancer expresses the prolactin receptor. Based on the results of the proposed clinical trial, Dr. Kaklamani will be able to offer effective targeted therapy to women with metastatic breast cancer that expresses the prolactin receptor in the form of a well-tolerated, inexpensive, medication called cabergoline.

Summary of Progress to Date
Five patients have been enrolled to date. Toxicity has been minimal, and prolactin levels have decreased significantly. Efficacy has not been reported to date.

Project #4: *Validate a Lynn Sage Familial Breast Cancer Risk Screening Tool and Protocol* (Grant awarded in fiscal year 2013).

Principal Investigators
Julian C. Schink, MD, The John and Ruth Brewer Professor of Gynecology and Cancer Research, Director of Gynecologic Oncology,Associate Director for Clinical Affairs,Robert H Lurie Comprehensive Cancer Center,Northwestern University Feinberg School of Medicine; (*Note: Dr. Schink is now Chief of Obstetrics, Gynecology and Women's Health, Spectrum Healthcare.)Melissa Simon, MD,MPH, Associate Professor, Departments of Obstetrics and Gynecology, General/Preventive Medcine and Medical Social Sciences, Northwestern University Feinberg School of Medicine has assumed the role of Principal Investigator*

Background
We have developed the "Lynn Sage Familial Breast Cancer Risk Screening Tool and Protocol" and made adjustments to the tool and protocol based on input from leaders and staff from the Lynn Sage Breast Center, the Cancer Genetics program, the Northwestern Ovarian Cancer Early

Detection Program and the Robert H. Lurie Comprehensive Cancer Center. Our hypothesis is that use of a risk screening tool will increase the timely identification and referral of the majority of patients with inherited risk who would benefit from genetic and high risk counseling. In this intervention, breast imaging patients who consent to participate will be given the "Lynn Sage Familial Breast Cancer Risk Screening Tool". We developed the tool based on key components of the NCCN Guidelines for Genetic/Familial High Risk Breast and Ovarian Cancer and Breast Cancer Risk Reduction. Patients will be provided an information sheet on Hereditary Breast and Ovarian Cancer and contact information for genetic counseling. The coordinator will review the screening forms with 4 or more "Yes" answers, contact patients for additional information and schedule genetic consults as indicated.

Summary of Progress to Date
We have completed the first half of the project through:

Prepared intervention to facilitate genetic/familial high risk:

- Reviewed Intervention and "Lynn Sage Familial Breast Cancer Risk Screening Tool" and protocol withmultiple project collaborators
- Adjusted intervention (screening tool and protocol) based on collaborator input 3 times due to changes in collaborators' approach
- Developed supporting processes for intervention and training materials for staff
- Developed patient communication materials regarding genetic counseling and BRCA testing to be used in intervention
- Amended the research protocol for intervention and submitted to the IRB on 3 separate occasions; changes were a result of collaborator adjustments due to other research efforts and to incorporating the perspectives and agreement of additional collaborators.
- Addressed IRB questions and supported amended protocol through IRB process. The protocol is: STU00041300 4R for Guideline Indicated BRCA Genetic Assessment of Breast Center Patients

Developed baseline data:

- Collected patient cohort 1 retrospective data of BRCA test timing for breast cancer patients as compared to surgery, from 1/1/2010 to 6/30/2010, prior to the genetic counseling barriers study (83 patients)
- Collected patient cohort 2 retrospective data, from 9/1/2011 to 3/31/2011, after initiation of the barriers study (83 patients) of BRCA test timing for breast cancer patients as compared to surgery.

We are ready to conduct the intervention and have the agreement from all collaborators and IRB approval to complete the following:

- Test intervention with Lynn Sage Comprehensive Breast Imaging Center patients
- Measure intervention impact
- Share findings, develop reports and prepare manuscript for abstract submission and potential publication

Project #5: ***Pinpointing the Neuropathology of Cognitive Impairment in Breast Cancer Survivors.*** (Grant awarded in fiscal year 2013).

Principal Investigator
Joel L. Voss, PhD, Assistant Professor, Department of Medical Social Sciences, Northwestern University Feinberg School of Medicine

Background
The goal of this research is to better understand the neural mechanisms for cancer- and treatment-related cognitive impairments among breast cancer survivors. This information is crucial for validating this condition and for developing effective treatments based on knowledge of the neurotoxic effects. These objectives have significant potential for improving quality of life in breast cancer survivors suffering from cognitive impairments.

Summary of Progress to Date
Dr. Voss and his team met their recruitment goal of 16 individuals with a recent history of adjuvant chemotherapy for breast cancer, as well as 16 matched comparison subjects without a history of breast cancer or chemotherapy. Cognitive testing and MRI was successful and provides compelling new evidence for the specificity and severity of cognitive problems in breast cancer survivors.

Dr. Voss's team used specialized testing that allows us to separate specific disruptions. Specificity is important because honing in on a particular brain region, such as the hippocampus, is critical for developing targeted therapies to improve cognition. Their extensive testing indicated that survivors had complaints of cognitive problems, yet tested within the normal range on most standardized (pre-existing) tests of cognitive function. The team's specialized testing revealed drastic impairments in hippocampal-dependent function. They found that healthy controls demonstrated robust behavioral indicators of hippocampal function in specialized tests and robust activation of the hippocampus when performing these tests, but these effects were virtually absent in chemotherapy survivors. This drastic impairment in hippocampal function in survivors is in contrast to their relatively normal standardized test performance, which we take to indicate that survivors recruit other parts of their brains to overcome their hippocampal deficits, leading to relatively normal performance but high levels of perceived effort and difficulty.

Additional analyses of the MRI data are ongoing, as more detailed and time-consuming efforts are required to understand how normal hippocampal interactions with the rest of the brain are disrupted in survivors. These additional analyses could support the team's interpretation that self-reported cognitive impairments are due to compensatory recruitment of additional processing resources by survivors to overcome their hippocampal problems. In addition, they are collecting blood samples from our participants in order to determine the relationship between markers of inflammation and impaired hippocampal function. If chemotherapy-related inflammation is related to hippocampal damage, then anti-inflammatory drugs could be used to reduce the harmful effects of treatment on cognition without disrupting the adjuvant properties of chemotherapy for cancer. Future efforts will include analysis of MRI data with respect to inflammation.

Project #6: *Novel Role of PDEF in Breast Cancer Progression* (Grant awarded in fiscal year 2013).

Principal Investigator
Ming Zhang, MD, Associate Professor,Department of Molecular Pharmacology and Biological Chemistry,Robert H. Lurie Comprehensive Cancer Center, and Center for Genetic Medicine, Northwestern University Feinberg School of Medicine

Background
Breast cancer is considered a genetic disease. Its initiation and progression often result from genetic mutations or epigenetic modification of certain master regulators. Recent studies of this laboratory have demonstrated that PDEF is a master regulator that controls breast cancer progression. Clinically, low expression of PDEF was significantly associated with basal-like breast cancer, and with the low survival rate of cancer patients. Dr. Zhang's hypothesis is that PDEF may block tumorigenesis by inhibiting cancer stem cell properties and enhancing mammary cell differentiation. Our data showed that PDEF inhibits breast cancer stem cell property and cell differentiation, and is involved in DNA damage response. This study will lead to therapeutic intervention against highly invasive and metastatic breast cancers.

Summary of Progress to Date
The study identified a novel role of PDEF in regulating breast cancer stem cells. It showed that loss of PDEF expression in human breast cancers is linked to cancer progression and metastasis. Dr. Zhang found that low expression of PDEF was significantly associated with basal-like and claudin subtype of breast cancer. Further, he demonstrated that PDEF is capable of transactivating the tumor suppressor gene, maspin, which inhibits breast cancer stem cell property.

Project #7: Aromatase Link Between Obesity and Breast Cancer Risk (Grants awarded in fiscal year 2012 and fiscal year 2013).

Principal Investigator
Hong Zhao, PhD, Research Associate Professor, Department of Obstetrics and Gynecology, Division of Reproductive Biology, Northwestern University Feinberg School of Medicine

Background
That obesity is a risk factor for breast cancer has been recognized for decades, yet the mechanisms that link obesity to breast cancer are unclear. Providing a plausible mechanism that identifies obesity as a causal factor in breast cancer that is supported by convincing data will be a key motivational factor for behavior change to reduce breast cancer. A common risk factor for developing breast cancer in women is cumulative and excessive exposure to estrogen. This study seeks to characterize the role of aromatase and local tissue estrogen in expediting early tumor development and the related mechanisms, and may serve as the basis for using aromatase inhibitors in the prevention of breast cancer development in patients with genetically-driven breast cancer. The overall hypothesis is that high-fat diet induced obesity increases breast aromatase expression, local estrogen production and breast cancer risk. Aromatase inhibitors can counteract the obesity-related effects in breast tissue of postmenopausal women with high breast cancer risk.

Summary of Progress to Date
The experiments outlined in the proposal have been performed. The aromatase transgenic mouse has been successfully crossed with the breast cancer mouse model to generate the double transgenic mouse, and it has been determined that local estrogen excess increased tumor incidence, tumor onset, tumor growth rated, and tumor histological type in genetically modified ERBB2-driven mammary tumorgenesis (AIM 1). Recruitment of large cohorts has begun. These cohorts will start treatment of breast cancer using aromatase inhibitor (AIM 2).

Project #8: *huATN-658, A Humanized Urokinase Plasminogen Activator Receptor Targeting Monoclonal Antibody for the Treatment of Estrogen and Receptor Negative Breast Cancer* (Grant awarded in fiscal year 2012).

Principal Investigator
Andrew Mazar, PhD, Director, Center for Developmental Therapeutics; Entrepreneur-in-Residence, Chemistry of Life Processes Institute; Professor, Department of Molecular Biosciences, Northwestern University

Background
This research project focuses on understanding the mechanism of action and building the rationale for translating a novel monoclonal antibody (ATN-658), into the clinic for the treatment of metastatic breast cancer (MBC). ATN-658 targets the urokinase plasminogen activator receptor (uPAR). ATN-658 has been humanized and Dr. Mazar's team has been developing this antibody in collaboration with the NCI (National Cancer Institute). uPAR has been shown to be important to breast cancer progression, and ATN-658 is a first-in-class therapeutic targeting this receptor.

Summary of Progress to Date
Dr. Mazar's team has continued to evaluate ATN-658 using a novel model of breast cancer metastasis. They first evaluated the expression of HER2, and found HER2 expression had no effect on the anti-tumor activity of ATN-658. Now, the team is evaluating the activity of ATN-658 alone and in combination with chemotherapy to see whether these treatments affect the growth of these tumors. If ATN-658 does affect the growth of tumors, they will carry out an extensive analysis to determine which signaling pathways are being affected. This work will complete the full evaluation of ATN-658 in metastatic breast cancer models.

Project #9: *Progesterone Antagonists for the Prevention of Breast Cancer* (Grant awarded in fiscal year 2012).

Principal Investigator
Seema Khan, MD, PhD, Professor, Department of Molecular Pharmacology and Biological Chemistry, and Professor, Department of Pathology, Northwestern University Feinberg School of Medicine

Background
Progesterone is increasingly recognized as an important component of the hormonal causes of breast cancer. Blockade of the effects of progesterone on the breast may be a powerful tool for breast cancer prevention. The development of several effective anti-progesterone agents presents

an opportunity for the development of novel strategies for breast cancer prevention, but the toxicities of these agents have been a barrier to their use in healthy women. Of the existing anti-progesterone agents, three are candidates for testing against breast cancer cells; these may retard growth of breast cancer cells and cause breast cancer cell death. These drugs also have chemical structures suitable for delivery through the skin, potentially avoiding toxicity in humans.

New anti-progesterone drugs (CDB4124 and its derivatives) offer an important, but so far unutilized, opportunity for breast cancer prevention. Future plans include the testing of the most suitable anti-progesterone agent in models that include spontaneous and progesterone-pathway-specific tumor models, and then in early-phase clinical trials.

Summary of Progress to Date
Progress has been made in identifying a progesterone proliferation signature in breast cancer cells, as well as benign mammary tissues that are antagonized by the anti-progestrone drugs. Pathway analysis of the genes revealed that cell cycle was highly represented by these genes. Therefore, one physiological process that promotes a tumor-permissive environment in the breast is proliferation. To further enrich and customize the proliferation data set to apply to benign/neoplastic tissues, microarray data was cross-referenced with those of another study investigating benign mammary tissues donated from premenopausal women volunteers.

The hypothesis is that 16 genes are upregulated by progesterone/progestins in both breast cancer and benign mammary cells, which are involved in promoting proliferation and which are also effectively antagonized by the antiprogestins.

Dr. Khan's team has tested whether CDB4124 can preventbreast cancer induction by a progestin. They monitored mammary tumor formations, but induced mammarycarcinogenesis did not happen. The mouse model was switched to a rat model for thesecond trial. The team has tested the efficacy of the new anti-progesterone drugto retard tumor growth. Tumor incidence, latency, multiplicity, and burden were recorded weekly.

Tumor latency was increased with CDB4124 treatment, whereas tumor incidence and burden was decreased, compared to other treated groups. Results indicated that natural progesterone promotes certain tumor formation, suggesting good potential as a breast cancer prevention agent.In the upcoming year, the team's goal is to characterize the 16 signature genes and potentially use them as markers of antiprogestin response and/or decreasing tumor promoting potential.

Project #10: *Cyclophin A as a Target in Breast Cancer* (Grant awarded in fiscal year 2012).

Principal Investigator
Charles V. Clevenger, MD, PhD, Professor, Department of Pathology, Northwestern University Feinbery School of Medicine (*Note: Dr. Clevenger is now Chair*, *Department of Pathology, Carolyn Wingate Hyde Professor in Cancer Biology, Virginia Commonwealth University).*

Background
Cyclophilins are enzymes that regulate protein structure and function. In this role, they can serve as signaling switches and regulate the activity of cell surface receptors, kinases, and transcription factors that contribute to the growth and spread of breast cancer. Recently, Dr. Clevenger's lab has

documented a critical role in breast cancer cells for cyclophilin A (CypA) in the activation of a signaling protein implicated in the biology of several cancers. CypA is a pharmacologic target for cyclosporine A (CsA), and its non-immunosuppressive analog, NIM811. Dr. Clevenger and his team initially hypothesized and demonstrated that CsA could inhibit estrogen receptor positive and negative breast cancer signaling, growth, and progression. Subsequent studies with NIM811 in cells has revealed therapeutic potential equal to or exceeding CsA. The goals of this proposal are: 1) to map the biology of CypA in breast cancer, 2) to characterize the spectrum of NIM811 actions against breast cancer, and 3) to translate these findings into pre-clinical and phase I trials. These studies could open the use of NIM811 as a novel drug with minimal toxicity, for both therapeutic and chemopreventive purposes.

Summary of Progress to Date
Project research has focused on examining the effects of a cyclosporine, an immunosuppressive agent and its non-immunosuppressive analogs NIM811 and SCY618801 against breast cancer cells. This research made excellent progress in the last year demonstrating that: 1) NIM811 and SCY618801 are effective against breast cancer in the lab, 2) these agents synergize with other recognized breast cancer agents such as doxorubicin and dasatinib, 3) NIM811 is effective against both primary breast tumors and their metastasis. These are significant findings, and given the minimal toxicity of both NIM811 and SCY618801, the next step will be to consider the design of a clinical trial in breast cancer patients with these agents.

Project #11: ***Effect of Percutaneous 4-OHT on Mammographic Breast Density and MRI Enhancement in Patients with Newly Diagnosed DCIS*** (Grant awarded in fiscal year 2011).

Principal Investigator
Lilian Wang, MD, Assistant Professor of Radiology, Breast Imaging Section, Northwestern University Feinberg School of Medicine

Background
As discussed in the previous study, mammographic breast density has been correlated with breast cancer risk. Studies have demonstrated the risk of breast cancer for women with increased breast density is four to six times that for women with less breast tissue. Unlike most other breast cancer risk factors, breast density is a modifiable factor, which may be influenced by hormonal regulation. Hormone replacement therapy in postmenopausal women has resulted in increased breast density, while therapy with tamoxifen has been shown to decrease breast density.

This study investigates the effect of oral tamoxifen on background enhancement on MRI and breast density on mammography. Both increased background enhancement and breast density have been correlated with increased cancer risk.

Summary of Progress to Date
At this time, eight patients have undergone MRI evaluation. An additional patient has been recruited. Recruitment is slow due to lack of a study coordinator, limited number of eligible patients, and patient hesitation to undergo MRI evaluation. There have been no changes in the study protocol or methods during last year. Due to the small number of participants, no conclusions can be made at this time.

Project #12: ***Effect of Weight Loss on Breast Density Using Both Digital Mammography and Breast MRI*** (Grant awarded in fiscal year 2010).

Principal Investigator
Nora Hansen, MD, Associate Professor, Department of Surgery, Chief, Division of Breast Surgery, Northwestern University Feinberg School of Medicine

Background
High breast density is one of the strongest known risk factors for breast cancer. In fact, studies demonstrate a five-fold greater risk of breast cancer for women with the highest breast density compared to those women with the lowest breast density. Obese patients have a higher rate of breast cancer even though mammography results indicate that they have lower breast density. This suggests that mammographic imaging may not be as accurate in obese patients. Breast MRI has been shown to be helpful in identifying abnormalities in patients with dense breasts and may be a more accurate way to measure breast density since it measures by volume rather than two-dimensionally.

To test this hypothesis, Dr. Hansen and her team are evaluating breast density using mammograms and the more sophisticated breast MRI on a group of obese patients undergoing gastric bypass surgery and a control group of obese women not undergoing the surgery. Taking measurements prior to and one year after the surgery, the work aims to determine if significant weight loss will have an impact on breast density. The expected outcome is that significant weight loss will reduce breast density in the patient population who underwent gastric bypass surgery, thereby reducing their risk for breast cancer.

Summary of Progress to Date
The study coordinator attended 15 of the educational classes in the Northwestern Memorial Faculty Foundation Gastrointestinal clinic (NMFF GI clinic), but was not able to recruit any patients from the classes. The team was contacted by 12 potential control subjects via the clinicaltrials.gov and cancertrials.gov websites on which the study is listed without any electing to participate. One of the deterrents for the study is the magnetic resonance scan of the breast using the contrast agent; potential subjects are not comfortable having the dye injection.

Recruitment has been primarily from the nutrition educational classes at the NMFF GI clinic. Given some difficulty with recruiting patients to this study, entry criteria have been expanded. Initial recruitment was to consider patients undergoing gastric bypass surgery, because this resulted in the fastest weight loss. The study now includes patients undergoing the lap band procedure, as well as the gastric sleeve procedure. Patients on hormone replacement are not being considered, as long as they remain on the same hormonal regimen throughout the study period. With this expanded entry criteria, the team hopes to be able to successfully recruit patients.

Thank You

Our progress is a significant step towards the day when women and families battling breast cancer can find peace in victory and comfort in the knowledge that others may not have to endure the same struggles.

On behalf of Northwestern Memorial Hospital, the Robert H. Lurie Comprehensive Cancer Center of Northwestern University, the many researchers whose work is made more meaningful, and the families whose lives are made better by your thoughtful generosity, we thank you.

If you would like additional information about any of the initiatives discussed in this report, or if we may be of assistance in any way, please do not hesitate to contact:

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