

SPOTLIGHT ON TRIALS

Two Alliance Studies, One Drug: A Closer Look at Cabozantinib

In January 2011, the U.S. Food and Drug Administration (FDA) granted cabozantinib orphan-drug status for the treatment of certain types of thyroid cancer – a status granted to treatments for diseases that affect fewer than 200,000 people in the United States. In November 2012, the FDA approved cabozantinib (Cometriq™) for the treatment of medullary thyroid cancer, one of the rarer types of thyroid cancer. Cabozantinib is currently undergoing clinical trials for multiple oncology indications. In November 2011 Exelixis, the developer of the drug, entered into a Cooperative Research and Development Agreement (CRADA) with the National Cancer Institute's Cancer Therapy Evaluation Program (NCI-CTEP) to develop clinical trials with cabozantinib. The agreement was expanded in May 2012. Alliance researchers will evaluate the efficacy of cabozantinib in kidney cancer and ocular melanoma under this program.

Why Cabozantinib?

Cabozantinib, also known as XL184, is a small molecule inhibitor of the tyrosine kinases, primarily c-Met (MET) and vascular endothelial growth factor receptor type 2 (VEGFR-2). These two key kinases are involved in the development and progression of many cancers. Other additional kinase targets include RET, AXL, KIT, and TIE-2. Pre-clinical studies demonstrated that XL-184 potently inhibited multiple receptor tyrosine kinases in various cancer cell lines and animal xenograft models and

caused reductions in tumor growth, metastasis and angiogenesis.

Recently, preclinical models have shown that treatment with selective inhibitors of VEGF signaling can result in tumors that are more invasive and aggressive compared to control treatment. In preclinical studies, upregulation of MET has been shown to occur in concert with development of tumor invasiveness after selective anti-VEGF therapy, and may constitute a mechanism of acquired or evasive resistance to agents that target VEGF signaling without inhibiting MET. Treatment with cabozantinib in similar preclinical studies resulted in tumors that were less invasive and aggressive compared to control or selective anti-VEGF treatment. As a result, cabozantinib has the potential for improving outcomes in a range of indications, including those where selective anti-VEGF therapy has shown minimal or no activity.

Promising results have been reported from early phase clinical trials of castration-resistant prostate cancer, non-small cell lung cancer, hepatocellular cancer and other solid tumors. The safety profile reported is comparable to that of other VEGFR TKIs and includes diarrhea, stomatitis, palmar-plantar erythrodysesthesia syndrome, decreased weight, decreased appetite, nausea, fatigue, oral pain, hair color changes, dysgeusia, hypertension, abdominal pain, and constipation.

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ALLIANCE A031203 Randomized Phase II Study Comparing Cabozantinib with Commercially Supplied Sunitinib in Patients with Previously Untreated Metastatic Renal Cell Carcinoma

More than 60,000 people will develop renal cell carcinoma (RCC) in the United States this year, approximately 20 to 30 percent will present with metastatic disease and a significant number of patients with localized disease (20 to 40 percent) will experience systemic recurrence.¹

Treatment options have improved in recent years, with the availability of targeted therapies. Currently, there are several options for first-line therapy with the vast majority of patients receiving treatment with a VEGF-targeted tyrosine kinase inhibitor (TKI) such as sunitinib. VEGF-targeted therapy has proven to be a successful strategy in RCC since most patients with clear cell RCC carry inactivating mutations in the von Hippel-Lindau (VHL) tumor suppressor gene.² Loss of VHL function leads to increases in hypoxia-inducible factor (HIF) levels, which in turn lead to increases in HIF-regulated genes, including VEGF. In large randomized clinical trials, the use of VEGF-targeted therapies including sunitinib, sorafenib, bevacizumab, and pazopanib has resulted in marked gains in progression-free survival (PFS), and trends towards improvements in overall survival (OS).³ However, patients treated with these agents generally have disease progression within six to 11 months and more potent VEGF inhibitors are needed.⁴

Cabozantinib is a potent inhibitor of VEGFR-2 that may offer advantages over the two FDA-approved front-line VEGF TKIs: sunitinib and pazopanib since it is also a potent inhibitor of MET.⁵ VEGF and MET play an important role in RCC through multiple mechanisms:

1. MET and VEGF receptors cooperate to promote tumor survival through angiogenesis, invasion, motility, proliferation and survival.⁶
2. Emerging data indicate that MET could be important in resistance to VEGFR inhibitors. In fact, inhibition of MET overexpression prevents hypoxia-induced invasiveness.⁷
3. Sporadic clear cell RCC has dysregulation in the MET pathway.⁸
4. VHL loss results in increased Hepatocyte Growth Factor-driven invasiveness. HGF is the ligand for MET.⁹
5. Multiple shRNAs against MET (among 88 kinases) preferentially inhibited the viability of RCC VHL cells, suggesting that MET in itself is an attractive target for RCC.¹⁰

Bone metastases are commonly seen in RCC encompassing 30 percent of metastatic cases.¹¹ Patients with bone metastases seem to have less benefit from VEGF TKIs as shown in a recent large French study, even when adjusted for known prognostic factors in advanced RCC.¹² Cabozantinib has been reported to induce bone scan responses, durable pain relief, and reductions in bone turnover markers in patients with metastatic castration-resistant prostate cancer; therefore, its use in other solid tumors with bone metastases such as RCC, where current standard VEGF TKI treatments seem suboptimal, is attractive.¹³

Overall, cabozantinib may present three major advantages over available and FDA-approved VEGF TKIs: 1) more potent VEGFR-2 inhibition; 2) MET inhibition as a mechanism to counteract subsequent VEGFR inhibitor resistance; and 3) favorable effect on bone metastases.

To date, one Phase 1b trial (XL184-008) included patients with metastatic refractory RCC. The study

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included 25 patients with RCC who failed a median of two prior lines of systemic therapy (mostly VEGF and mTOR inhibitors) and were mostly intermediate or poor-risk. Preliminary results showed a best ORR of 28 percent by RECIST criteria and a Kaplan Meier estimate of median PFS of 14.7 months.¹⁴ Two of the seven patients who had a response had been treated with more than four prior agents. In addition, bone pain and pain response were decreased with cabozantinib in a small number of patients who had symptomatic bone metastases.

These results in a heavily pretreated population are similar to the activity of FDA-approved VEGF-targeting agents when used in previously untreated patients (e.g., sunitinib had an ORR of 31 percent and a PFS of 11 months in a predominantly favorable/intermediate risk groups.) FDA-approved agents in the second line setting, such as everolimus and axitinib, have been reported to have shorter PFS in the 4 to 5 month range after progression on first-line sunitinib.¹⁵⁻¹⁶

ALLIANCE A031203 is a randomized phase II trial of cabozantinib versus sunitinib in patients with previously untreated metastatic renal cell cancer with intermediate/poor risk. These patients represent 80 percent of all patients and those who will likely need systemic therapy. The primary objective of this study is to compare PFS in patients treated with cabozantinib versus patients treated with sunitinib. At the time of progression, patients' treatment will be unblinded and those who had been receiving sunitinib will be permitted to crossover to cabozantinib.

Secondary objectives are two-fold: 1) to compare the ORR of patients treated with cabozantinib versus patients treated with sunitinib, and 2) to evaluate whether patients treated with cabozantinib have improved OS when compared with patients treated with sunitinib.

Eligible patients must have histologically confirmed RCC with clear cell components. Patients must meet intermediate/poor risk criteria, have measurable disease as indicated by RECIST criteria and should be untreated with systemic agents. Adequate bone marrow, cardiac, renal, and hepatic function will be required, and patients with active malignancies other than renal cell carcinoma will be ineligible.

About 140 people will take part in this study, which includes one correlative study, Alliance A031203-ST1. The correlative science study objectives are to determine whether patients with tumors having high MET expression by immunohistochemistry (IHC) have an improvement in PFS compared to patients with tumors exhibiting low MET expression on both arms of this study. There currently are no prognostic biomarkers for treatment with MET inhibitors.

The study protocol for ALLIANCE A031203 is currently in development and it is projected to be activated in April 2013.

The Study Chair is Toni K. Choueiri, MD, of the Dana-Farber Cancer Institute, e-mail: toni_choueiri@dfci.harvard.edu.

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ALLIANCE A091201 Randomized Phase II Study Comparing the MET Inhibitor Cabozantinib to Temozolomide/Dacarbazine in Ocular Melanoma

Ocular melanoma (OM) is the most common primary intraocular malignancy in adults.¹ It is an aggressive form of cancer that can involve any of three areas of the eye: the iris (the pigmented area surrounding the pupil), the ciliary body (a thin tissue layer in responsible for aqueous humor production), and/or the choroid or posterior uvea (the vascular layer between the retina and the sclera that nourishes the retina). Approximately 85

percent of ocular melanomas are uveal in origin, with primary conjunctival and orbital melanomas being less common.²⁻³ Uveal melanoma has an incidence of five cases per million people per year in the U.S. and represents about five percent of all melanomas. Although OM and cutaneous melanoma arise from the same cell type, the molecular pathobiology is very different.³⁻⁴ The incidence of uveal melanoma varies with skin pigmentation and ethnicity. It may be more common in people who have atypical mole syndrome, (dysplastic naevus syndrome). Approximately an eight-fold higher incidence exists in fair-skinned Caucasians whereas Asian and dark-skinned populations exhibit a lower incidence.⁵ Unlike cutaneous melanoma, exposure to ultraviolet light has an unclear role in the development of this disease.

The development of metastasis in uveal melanoma is common and occurs in approximately 50 percent of patients with posterior uveal melanoma within 15 years after the initial diagnosis and treatment.⁶ In the Collaborative Ocular Melanoma Study Group study, 1,003 patients with non-metastatic uveal melanoma were followed for at least five years after diagnosis.⁷ Approximately half of patients developed metastatic disease, typically to the liver and often to lung and bone, and the incidence of new metastases continued to increase with time.

The outcome for patients with metastatic uveal melanoma is notably dismal. Uveal melanoma is thought to be particularly resistant to systemic treatment, and no systemic therapy has been demonstrated to improve survival.⁸ Drugs commonly used to treat advanced cutaneous melanoma rarely achieve durable responses in patients with uveal melanoma. Nathan et al compared the outcome between 139 patients with non-uveal melanoma and 16 patients with uveal melanoma who were treated with dacarbazine (DTIC), BCNU, cisplatin,

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and tamoxifen (Dartmouth regimen).⁹ The response rates were 33 percent and 6 percent respectively.

In a review of the MD Anderson Cancer Center experience of 143 treated patients with ocular melanoma, there was only a single objective response observed.¹⁰ Retrospective reviews of the Eastern Cooperative Oncology Group (ECOG) and Southwest Oncology Group (SWOG) experiences revealed similar findings.¹¹ More recently, the anti-CTLA4 monoclonal antibody ipilimumab has become a standard treatment for uveal melanoma; however, it is not clear that the drug has significant efficacy in this sub-histology of all melanomas. In a retrospective analysis of uveal melanoma patients treated with ipilimumab, no responses by RECIST criteria were noted and three patients eventually had stable disease as best response.¹²⁻¹³ Given the lack of effective systemic treatment options for these patients, outcomes are poor once metastatic disease occurs, and the median survival from the time of the development of distant metastatic disease is six to 12 months.^{12,14-15} It is clear that novel strategies and more effective therapies are desperately needed for this disease.

Uveal melanoma is well characterized to harbor activated MET and data from cell lines suggest that inhibition of MET by small molecule RTK inhibitors may block proliferation and migration of cancer cells in this disease. As described above, cabozantinib inhibits multiple receptor tyrosine kinases (RTKs) implicated in tumor growth, metastasis and angiogenesis, including c-Met.

Dacarbazine (DTIC) is an imidazole dimethyltriazene prodrug that has been approved for use in the treatment of metastatic malignant melanoma and Hodgkin's disease since the 1970s. DTIC is currently the only widely registered chemotherapy drug for metastatic stage IV melanoma. DTIC is a non-classical alkylating agent that causes DNA mis-pairing and strand breakage, leading to

cell death (necrosis). Its exact mechanism is not completely understood. It is a cell cycle nonspecific drug, meaning that it causes cell damage and death throughout the life cycle of a cell, and not at any one particular time. When a patient is treated with DTIC, 50 percent of the drug is metabolized by the liver and 50 percent excreted in the urine. DTIC is considered a standard treatment for metastatic melanoma with reported response rates from five to 15 percent.

Temozolomide (Temozar) is an oral imidazotetrazinone pro-drug that converts under physiological conditions to the same active alkylating agent as DTIC. In a large randomized phase III study comparing oral temozolomide versus intravenous DTIC in patients with advanced melanoma, median survival time was 7.7 months for the temozolomide-treated patients and 6.4 months for the DTIC-treated patients.¹⁶ There were no major differences identified in drug safety, but more importantly there were no significant differences identified in clinical response rates either. Complete or partial responses were seen in 13.5 percent and 12.1 percent of patients treated with temozolomide and DTIC, respectively. Similarly, stable disease was reported in 17.9 percent and 15.8 percent of patients treated with temozolomide and DTIC, respectively. Although temozolomide has not been licensed for use in malignant melanoma, it is still used extensively in therapeutic trials and clinical practice.

ALLIANCE A091201, a one-stage phase II trial, will assess the anti-tumor efficacy of cabozantinib in uveal melanoma. Specifically, this study will assess whether cabozantinib can improve the four-month progression-free survival (PFS) rate in patients with ocular melanoma from 15 percent, as reported with temozolomide and dacarbazine, to 40 percent with cabozantinib. The molecular impact of cabozantinib on metastatic uveal melanoma lesions in liver and bone will be evaluated using

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FDG-PET/CT imaging. Secondary objectives of this study are to estimate the distribution of progression-free survival times and overall survival times using the method of Kaplan Meier, estimate the confirmed response rate as determined by RECIST criteria, assess the toxicity profiles, and correlate the response with MET molecular status.

Patient eligibility requirements will include histologically confirmed uveal melanoma that is metastatic or unresectable. Patients who received prior therapies are eligible, except those who have had treatments aimed at or against c-Met or VEGF/R, and the chemotherapy agents temozolomide and dacarbazine. Patients who have had cytotoxic chemotherapy or prior radiation therapy within specific timeframes are ineligible.

About 66 people will take part in this study, which includes one correlative science substudy, Alliance A091202-ST1. Preliminary data suggest that activation of the oncogene c-Met can be observed in approximately 60 to 80 percent of both uveal melanoma cell lines and primary tissue specimens. Several groups have characterized the *in vitro* activity of c-Met inhibitors in blocking proliferation and reducing the metastatic phenotype in this disease. Among other objectives, this substudy will describe the relationship between pre-treatment MET expression or GNAQ/GNA11 mutation and clinical benefit.

The study protocol for ALLIANCE A091201 is currently in development and it is projected to be activated in April 2013.

The Study Chair is Jason J. Luke, MD, of the Dana-Farber Cancer Institute, e-mail: jason_luke@dfci.harvard.edu.

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Congratulations!

Alliance Grant Preparation Teams



I'm pleased to announce that a number of important funding proposals have been submitted to the NCI on behalf of the Alliance. These include applications for funding as an NCTN Network Operations Group and Network Statistics and Data Center, as well as Alliance-sponsored applications for two Integrated Translational Science Centers.

A great many people contributed countless hours to these efforts. **Gini Fleming, Edith Perez, Dan Sargent, Phil Febbo, and Guido Marcucci** provided exceptional scientific leadership for the four separate proposals. Special thanks to **Trini Ajazi, Denise Collins-Brennan, Kathy Mrozek, Denise Marsano, Sherry Breaux, Katherine Faherty, Karen Chuang** and **Mary Cate Zipprich** for getting the Network Operations U10 out the door in fine shape. The Alliance Reporting Workgroup, led by **Stacey Guy**, developed and generated reports over several months.

These applications represent many months of hard work by Alliance leaders and staff, beginning two and a half years ago with the re-organization of the Statistics and Data Center. Most important, these grants demonstrate capabilities that can only come through dealing with the difficult challenges of a merger, creating a new group that is truly greater than the sum of its parts. The Alliance Program leaders: **Heidi Nelson, Jan Buckner, Gini Fleming, Dan Sargent, and Phil Febbo**, deserve the credit for crafting the scientific vision, and the many Alliance committee leaders and members deserve our thanks for making this vision a reality. Finally, particular thanks to Edith Perez, who was charged with the considerable task of coordinating all the outstanding publications of our group.

You should all be very proud of what has been accomplished and I am confident that we will make a very strong impression on the review committee.

Here's hoping 2013 is a great year for all!

Monica Bertagnoli
Chair, Alliance for Clinical Trials in Oncology



Individualized Breast, Gynecologic, Colon Cancer Management: New Data, Updates Relevant to Patient Care

Sponsored by the Mayo School of Continuous Professional Development in conjunction with the 26.2 with Donna - The National Marathon to Finish Breast Cancer / February 15-16, 2013 / Prime F. Osborn III Convention Center / Jacksonville, Florida

Course Co-Directors: Michele Y. Halyard, MD, Carolyn Landolfo, MD and Edith A. Perez, MD.

This two-day course is designed for medical oncologists, radiation oncologists, surgeons, advanced registered nurse practitioners (ARNP), physician assistants (PA), oncology nurses, registered pharmacists, pharmacy technicians, and general practitioners (internal medicine and family medicine). Residents in training and fellows from these specialties are encouraged to attend.

Topics in breast cancer, gynecologic malignancies and colon cancer will include screening, diagnosis, and state of the art treatment. Emphasis will be given to management guidelines, molecular diagnosis and individualization of cancer therapy based on molecular profiles. There is a special session devoted to the area of supportive care in oncology, with an emphasis on optimal surveillance after treatment for each of the selected malignancies. **To register:** Visit <http://www.mayo.edu/cme/hematology-and-oncology-2013j145>

Society for Clinical Trials 34th Annual Meeting



Sheraton Boston Hotel / May 19-22, 2013 / Boston, Massachusetts

***Ideal learning opportunity** for leading edge trialists, policy experts, biostatisticians, ethicists, epidemiologists, regulators, and students! The Society for Clinical Trials, created in 1978, is an international professional organization dedicated to the development and dissemination of knowledge about the design, conduct and analysis of government and industry-sponsored clinical trials and related health care research methodologies.*

Keynote Speakers: “Transforming 300 Billion Points of Data into Diagnostics, Therapeutics, and New Insights into Disease” by Atul Butte, MD, PhD, Curtis Meinert Lecture and “Challenges for Health Behavior Trials from Design to Practice: The Example of Unhealthy Alcohol Use” by Richard Saitz, MD, MPH, FACP, FASAM, Founders Lecture

May 19: Full-day and half-day workshops

May 20-22: Engaging invited sessions; distinctive contributed papers and posters; and networking opportunities with others in the clinical trials community

To register: Visit www.sctweb.org. The SCT meeting is open to members and non-members. Non-members who register receive a one-year membership and a subscription to the SCT journal, *Clinical Trials: Journal of the Society for Clinical Trials*.

Positions Open on Alliance Oncology Nursing Committee

The Alliance Oncology Nursing Committee (A-ONC), chaired by Lisa A. Kottschade RN, MSN, CNP, announces the availability of two positions on the A-ONC. The two positions are Vice Chair and GI Committee liaison. Job descriptions for both positions as well as instructions on how to apply are listed below.

A-ONC Vice Chair Job Description

Responsibilities of the Vice Chair include, but are not limited to:

1. Mandatory attendance at all Alliance group and committee meetings. Collaboration with the A-ONC Chair to develop agendas for meetings/teleconferences.
2. Attendance at Alliance administrative meetings when the Chair is unable to attend.
3. Participation in regular conference calls to discuss A-ONC business and future initiatives.
4. Leadership of A-ONC initiatives
5. Communication and collaboration with the broader Alliance membership regarding administrative issues and projects of mutual interest

Minimum requirements for position include:

1. Registered Nurse with Bachelor's degree required; with Masters or doctorate degree preferred.
2. Employment at an Alliance institution
3. Demonstrated commitment to the Alliance and its mission
4. Excellent writing and organizational skills
5. Minimum of two years experience working with research protocols within the cooperative group setting
6. Main employer will allow time to fulfill position responsibilities (documented via letter or email). The anticipated time commitment - 8 hours/month and two or three days traveling twice a year for Alliance meetings

A-ONC GI Liaison Job Description

Responsibilities of the A-ONC GI Liaison include but are not limited to:

1. Mandatory attendance at all group and committee meetings.

2. Review of all new protocols/forms/amendments for the GI malignancy-related protocols and act as a resource for other Alliance nurses regarding protocol execution (available to response via phone and/or e-mail).
3. Collaboration across committees on projects, publications, educational initiatives, etc.;
4. Provision of nursing perspective and expertise regarding study design and methods;
5. Serving as a principal and/or co-investigator on Alliance trials;
6. Publication and dissemination of Alliance-related research findings.

Minimum requirements for position include:

1. Registered Nurse
2. Employment at an Alliance institution
3. Demonstrated commitment to the Alliance and its mission
4. Minimum of one year experience working with research protocols within the cooperative group setting and experience in GI malignancies
5. Main employer will allow time to fulfill position requirements (documented via letter or e-mail). Anticipated time commitment 2-4 hours/month and two or three days traveling to Alliance meetings twice a year

How to Apply: Those interested in A-ONC membership (for either position) should submit a CV/resume and a one-page letter of interest to Lisa Kottschade, RN, CNP (Kottschade.Lisa@mayo.edu) by February 8, 2013. Specifically, your letter should address the following points:

1. Please provide a letter of financial support from your PI or supervisor, or indicate other sources.
2. Your area of expertise (clinical, education, administrative, research). Please be specific. For example, if you possess clinical expertise, describe your specific disease or modality-focus (breast cancer, prevention, symptom control, etc.).
3. Explain what contributions you will make to the committee.
4. Provide evidence that your current supervisor will support your participation. (An e-mail communication from your supervisor, addressed to Lisa Kottschade, is satisfactory.)

Alliance Members on the Move



Daniel J. Sargent

Daniel J. Sargent, PhD, Ralph and Beverly Caulkins Professor of Cancer Research at the Mayo Clinic, will begin a one-year term as President of the Society for Clinical Trials (SCT) in May 2013. In this role, Dr. Sargent will oversee all activities of the SCT, working with the Society's board of directors. The SCT is an educational, charitable and scientific organization dedicated to working internationally to advance human health through advocating the use of clinical trials, leading the development and dissemination of optimal methods and practices in clinical trials, and educating and developing all clinical trial professionals. It includes representatives from government, academia, industry, for-profit and non-profit sectors. The SCT differs from groups specializing in one discipline, disease, or therapeutic area because it recognizes the need for understanding and communications at all levels. It has an active partnership with the American Society of Clinical Oncology (ASCO), where the SCT and ASCO have co-sponsored a clinical trials methodology workshop at ASCO the last two years, coordinated by Susan Halabi, PhD, Professor of Biostatistics and Bioinformatics at Duke University and Faculty Statistician for the Alliance.

Dr. Sargent is also Adjunct Professor of Biostatistics at Duke University, University of Iowa and University of Minnesota, and Group Statistician for the Alliance.



Richard L. Schilsky

Richard L. Schilsky, MD, Chief of Hematology/Oncology in the Department of Medicine and Deputy Director of the University of Chicago Comprehensive Cancer Center, has been named to the newly created position of Chief Medical Officer (CMO) of the American Society of Clinical Oncology (ASCO). The CMO position was created by the ASCO Board of Directors to provide additional senior leadership and support to ASCO's fast-growing quality programs, public policy and communications efforts, as well as fundraising for ASCO's affiliated Conquer Cancer Foundation. Dr. Schilsky, former Group Chair of the Cancer and Leukemia Group B and Alliance member, will begin his new position in February 2013.

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Call for Alliance Members on the Move

Are you an Alliance member on the move or do you know someone who is? If so, we want to know. Please send names of Alliance members on the move, along with a brief description about their recent achievements, to *Alliance News* at jowens@uchicago.edu.

Alliance BioSpecimen Management System Launch Delayed

The new Alliance BioSpecimen Management System (BioMS) has been delayed due to additional system testing. The web-based tool, which must be used at enrolling sites to log, ship and track biospecimens collected from Alliance clinical trial participants, is now scheduled to launch in mid- to late February 2013.

All Alliance staff and members should continue the current procedures for logging and tracking biospecimens, including the continued use of the CALGB STS system, if applicable. Everyone who will eventually utilize the BioMS system for Alliance biospecimen tracking should plan to participate in ongoing training sessions. A calendar of training sessions, along with a library of training videos and other BioMS-related documentation, is available at the BioMS URL: <https://cbmiapps.wustl.edu/confluence/display/BP/BioSpecimen+Management+System+-+BioMS;jsessionid=9CD5C21C6BF7BD0DD276B50F166A6774>.

Questions: To get more information about the new biospecimen management system, contact the BioMS Help Desk by e-mail at BioMSHelp@bmi.wustl.edu or by phone at 1-855-552-4667.

Call for Photos / New Alliance Website

Want to see your institution featured prominently on the new Alliance website? If so, send us your photos. We welcome photos of all Alliance members and institutions. Just send them to us with a confirmation that all individuals pictured have given their consent for web posting to *Alliance News* at jowens@uchicago.edu. Also, make sure to include a caption with the date, location, and names of individuals in the photos.

Alliance Committee Meetings March 14-17, 2013 InterContinental Chicago O'Hare

Open to Alliance committee members only

Invitations for the March Committee Meetings

were sent in early January. If you are a committee member and have not received an invitation, please contact Katherine Faherty at 617-525-3022 or kefaherty@partners.org. Also note that the Breast Committee will meet on Sunday, March 17. For the draft schedule, visit the Alliance website.

Future Meeting Dates

2013 Group Meeting

November 7-9, 2013

Open to Alliance members

2014 Committee Meetings

May 8-10, 2014*

Open to Alliance committee members only

*date changed from March 27-29, 2014

Group Meeting

November 6-8, 2014

Open to Alliance members

All 2013-14 meetings will be held at the **InterContinental Chicago O'Hare**
5300 N. River Road, Rosemont, IL

For meeting and travel inquiries, contact Katherine Faherty
e-mail: kefaherty@partners.org
phone: 617-525-3022

For more information on the Alliance and updates about meetings, visit AllianceforClinicalTrialsinOncology.org