We need to create a new balance of more industry-sponsored trials relative to investigator-initiated studies to ensure that we have sufficient funding to support our research enterprise. Toward that goal, we are actively seeking sponsored research projects. We need to do more face time with pharmaceutical representatives at the AACR, ASCO, and other major oncology meetings. I am encouraging any senior faculty member who has an established relationship with a sponsor to take on a junior faculty member in ICT as a co-investigator so that he or she can also establish a relationship with that company.

Internally, we already have strong relationships with departments such as Breast Medical Oncology, Gastrointestinal Medical Oncology, Sarcoma, and Thoracic/Head and Neck Medical Oncology, alliances we need to strengthen and extend to other departments throughout MD Anderson. Faculty in such internal departments refer about 80 percent of the patients with advanced cancer that we assess for an appropriate clinical trial. We are also making ties with other stakeholders in the institution. We have a place in the Moon Shot Programs helping to build early phase clinical trials—whether cytotoxic, molecularly targeted, or immunologic. We already have a well-established relationship with the IPCT. They offer a growing molecular profiling platform, and we provide tumor biopsy material to them for additional assessment from patients who show a dramatic response to a Phase I clinical trial as part of IPCT’s Unusual Responder project. An example is a patient referred to us with advanced metastatic colorectal cancer who had a BRAF mutation and had a complete response to a protocol combining a BRAF inhibitor and a MEK inhibitor. That patient’s story is featured on pg. 2 of this newsletter.

An increasing amount of sponsored research is coming from pharmaceutical companies for clinical trials targeted to a specific mutation. Adding molecular screening increases costs and lengthens the time before trial enrollment. When patients show up in the clinic without a profile already done, six to eight weeks to get the profile done is added to the time frame. Time is already in short supply for these patients with advanced cancer who have already failed all conventional therapy. At that point, we have to find an interim treatment to stabilize them while their molecular profiling is underway. A modest amount of molecular screening is now being done in the IPCT; our lung cancer and gastrointestinal cancer patients already come into the clinic with an IPCT-conducted profile in hand—a trend we expect to become increasingly common. We are anticipating a future when all incoming patients arrive with their “mutational smorgasbord” data in hand, ready to enroll in a clinical trial matching their profile. Right now, it’s all in transition.

We are planning to increase the number of expansion cohorts added to promising Phase I clinical trials, which will then bridge quickly to Phase II trials conducted in our disease-centered departments.

We are also seeking ways to run the clinics more efficiently by improving technology. For example, we are equipping clinical staff with electronic tablets to record patient data instantly.

Our true strength lies in our energetic, talented, and ambitious faculty and staff, who are committed to our mission and have so many great ideas. I strongly believe that ICT will play a central role in the future of our institution.

We, the faculty and staff of the Department of Investigational Cancer Therapeutics, wish to express our heartfelt gratitude to Dr. Wolff for taking on the responsibility of leading our clinical, research, and educational activities, in addition to his other responsibilities as deputy division head for clinical and educational affairs and program director of the Division of Cancer Medicine’s Hematology/Oncology Fellowship Program, all while continuing to take care of his patients as a professor in the Department of Gastrointestinal Medical Oncology.

Effective June 1, 2013, Funda Meric-Bernstam, MD, medical director of the Khalifa Institute for Personalized Cancer Therapy and professor in Surgical Oncology, serves as the new chair of Investigational Cancer Therapeutics. She will present her ambitious plans for the department in the next issue of this newsletter.
Mr. Gloria Humble was not looking for a cure when she was diagnosed with stage 4 colorectal cancer in Lafayette, Louisiana, but a cure found her when she came to MD Anderson’s Clinical Center for Targeted Therapy (CCTT) for a second opinion, on the advice of her physician son. When Mrs. Humble saw a tumor growing out of the drain placed after surgical treatment of the malignancy, she was prepared to join her husband, who had died recently, and wasn’t planning to seek further treatment. “I didn’t believe in chemotherapy, and I didn’t want radiation either,” she said. “But my son said I had to do something about it, and suggested we seek a second opinion at MD Anderson.”

Mrs. Humble was referred to Dr. David Fogelman, assistant professor in the Department of Gastrointestinal Medical Oncology, who immediately called Dr. Gerald Falchook, above, right, assistant professor in the Department of Investigational Cancer Therapeutics. Mrs. Humble had received no systemic anti-cancer therapies before referral for a Phase I investigational trial.

When Mrs. Humble arrived for treatment in the CCTT, she had widespread metastases to her abdominal wall and abdominal lymph nodes, liver, inguinal lymph nodes, retroperitoneal and pelvic lymph nodes, and perirenal nodules, said Dr. Falchook. Mrs. Humble’s molecular profile revealed that she had a BRAF mutation, and she was subsequently enrolled in the Phase I clinical trial of BRAF inhibitor dabrafenib combined with MEK inhibitor trametinib on December 22, 2011—a day she calls her “miracle day.”

Dr. Falchook decided on this combination trial for Mrs. Humble because, although only modest antitumor activity had been seen using BRAF inhibiting monotherapy in patients with BRAF-mutant colorectal cancer, improved efficacy was seen when combining dabrafenib and trametinib, a selective MAPK kinase inhibitor, in a study of BRAF-mutant melanoma [N Engl J Med 2012;367:1694-703]. “We hoped to see improved efficacy with this combination in BRAF-mutant colorectal cancer as well,” Dr. Falchook noted.

“There was one slot left on that clinical trial, and I got it,” she said. Within only four days, Mrs. Humble could no longer see tumors protruding from her abdominal drain. “My tumors began shrinking right away,” she said. “And I never felt bad on this drug. I felt fine.” The only adverse effect Mrs. Humble experienced was pyrexia (fever) after about three months on the trial, which occurs in about half of patients on this regimen. She was hospitalized when questioning revealed that she was not oriented to time and place secondary to the high fever. Once her dosage had been reduced, pyrexia and related symptoms all resolved.

According to Rosa Mostorino, BS, CCRP, clinical studies coordinator for this trial, about halfway into her second cycle of treatment on the regimen, Mrs. Humble already had a partial response of 45 percent tumor reduction measured by RECIST, and 75 percent decrease by Cycle 5, nearly five months after starting treatment. Mrs. Humble was in complete remission by the first day of Cycle 9, which was 8.4 months after starting treatment, and continues on treatment a year and a half past her treatment start date. She and her daughter drive to MD Anderson monthly for follow-up visits with Dr. Falchook and advanced practice nurse Lindsay Gaido, MSN, below, right.

Mrs. Humble was impressed with the helpfulness of MD Anderson staff from the time she first arrived at MD Anderson with her daughter-in-law Spring, when they were having trouble finding their way to the clinic. They were approached by a passing employee, who said, “You look like you’re lost” and led them all the way to the check-in desk. “She was late for work taking us where we needed to go,” Mrs. Humble marveled. About her nurses and doctors, Mrs. Humble said, “You know they care about you personally. They are there for the people. A nurse told me, “This is where the miracles happen.” Mrs. Humble had her “miracle day,” thanks to the personal care by her clinical team, along with a personalized clinical trial matched to her molecular profile. Now that she is cancer free, she is also free to celebrate many more years enjoying her multi-generational family that now includes five great-grandchildren. “We are celebrating every day,” added Spring.
ICT Inpatient Service Expands to Deliver Extraordinary Care to Sickest Patients with Advanced Cancer

By Carol Howland

The Department of Investigational Cancer Therapeutics’ inpatient team provides care to our sickest patients, says Gerald Falchook, MD, medical director of the unit. About 70 percent of patients are admitted because of signs and symptoms associated with the growth of their tumors, such as severe dyspnea, pain from progressive tumor growth and ascites, or bowel obstruction, he noted. Less than five percent of this group are admitted for drug-related side effects. About 30 percent of patients are admitted every three to four weeks to receive hepatic arterial infusion chemotherapy for liver-predominant tumors. This regional mode of therapy can generate robust responses even in patients who had developed resistance to the same drugs previously given systemically, because administering chemotherapy directly into the artery that supplies the liver enables higher concentrations of the drugs to be given to the liver tumors without increasing toxicity. “We have observed patients who receive treatment this way respond for as long as five years,” notes Dr. Falchook.

The department now has the largest Phase 1 Program in the world, and as the outpatient volume has increased, the number of patients admitted to the inpatient service has increased as well. The ICT inpatient service now is the largest among the solid-tumor services in the Division of Cancer Medicine, with an average of 16-18 patients on any given day, ranging from approximately 12 patients on Mondays to 24 or more patients on Fridays. According to Nicole Vaughan-Adams, MSN, OCN, associate director, Clinical Nursing, the unit moved from P12 to G19 in October 2012 to accommodate 16 more beds, and will add another dozen beds for a total of 48 available beds by September 2013. “The move makes it easier for our inpatient team to deliver care to our patients,” says Dr. Falchook. “The G19 unit gives us more rooms and bigger rooms so that we are able to keep all our patients on one floor instead of scattered over several floors.” The average length of stay is about four days, comparable to that of other solid tumor services.

In addition to the nursing staff on G19 who provide 24-hour care, the patients are cared for seven days a week by four ICT inpatient physician assistants and advanced practice nurses. ICT clinical staff work closely with our Supportive Care Center staff to take care of patients with the most distressing symptoms. Also assigned to the inpatient unit to help support patient needs is one PharmD-level clinical pharmacist, a social worker, and a case worker. “Our pharmacist Amy Pai is an incredible source of knowledge about medications. Her participation on our inpatient service ensures better care of our patients,” says Dr. Falchook. “She is especially helpful when medications are changed, and she educates patients before they leave the hospital to make sure they take the correct medications when they go home.”

Clinical Center for Targeted Therapy Poised to See More Patients via Largest Phase I Program

By Parvathy Hariharan

David Hong, MD, medical director of the Clinical Center for Targeted Therapy (CCTT)—the Department of Investigational Cancer Therapeutics’ outpatient clinic—is excited to talk about plans for the center. “We have now become, within five years, the largest Phase I Program in the country,” he states.

With 127 clinical studies on the department’s priority list, CCTT is indeed a thriving hub of activity. Eleven oncologists work two full clinic days each week to see 50-100 patients each day. One mid-level provider works with each physician. Phase 1 fellows also rotate within the clinic. In a given week, two fast-track clinics operate for three days, and one fast-track clinic operates for two days. The support staff include five nurses, five patient service coordinators, two certified nurse assistants, three pharmacists, and one receptionist. The clinic is also in the process of hiring more clinical staff, says Terri Alexander, MSN, OCN nurse manager for the CCTT. “Our goal is to provide excellent service even on our busiest days,” she states.

While the majority of patients to date have been referred by MD Anderson oncologists, the CCTT especially welcomes referrals of patients from outside of MD Anderson. External referrals currently account for about 15 percent of CCTT patients. Physicians who wish to refer patients to the CCTT should contact the Patient Access Center at 713-792-1160. “The patient access specialist begins gathering the information we need to start working on finding treatment for the patient on day one,” Alexander says. “It is always important that we receive clinical information regarding the patient as well as pathology slides for verification and potential molecular profiling.”

The patient access specialists also obtain insurance information and other payment method information from the patients to secure their financial clearance to begin treatment in the CCTT.

Molecular profiling has been conducted for most CCTT patients since late 2009. Usually taking six to eight weeks from the time of tumor biopsy, this analysis is performed by the Institute for Personalized Cancer Therapy, which currently has assays for 46 genes from the patient’s existing paraffin-preserved biopsy tissue. The most common genes assayed include PI3K, EGFR, KRAS, BRAF, p53, and c-Met. In the meantime, the clinic enrolls the patients on a suitable clinical trial in the hope of halting disease progression until genetic profiling suggests they may have a better response to a different trial. Dr. Hong says that internally referred breast, colorectal, and melanoma patients routinely come with a molecular profile in hand, but increasing numbers of patients are arriving at the CCTT with a molecular profile done by a private organization such as Foundation Medicine.

A number of new protocols that will provide more clinical trial options for patients in the CCTT are expected to be introduced in the coming year. These include a pan BRAF inhibitor, a second-generation MDM2 inhibitor, a CHK1 inhibitor, a TGF-β inhibitor which is the first of its kind, an aurora kinase inhibitor targeting CDKN2A, and PDL1 and PDL1 immune-modulating agents. “A lot of new protocols looking for specific molecular abnormalities and genetic signatures are emerging now,” Dr. Hong says.
ICT Inpatient Service continued from page 3

One attending faculty member rotates on the service each week to oversee patient care, along with an ICT clinical fellow. This is a great learning experience for the fellows, notes Dr. Falchook. “The fellows have the opportunity to treat many end-stage complications of cancer,” he says. “They become skilled in discussing end-of-life issues, including hospice and do-not-resuscitate orders.”

Dr. Falchook is especially grateful for the midlevel providers (physician assistants and advanced practice nurses) and other clinical staff dedicated to delivering the very best, compassionate care to patients on the inpatient unit, commending them for their critical thinking skills, and above all, because they truly care about their patients. The midlevel providers assigned to the ICT inpatient unit are Sarah Baldwin, ANP, MSN, Laura Beatty, PA-C, MPAS (left), Jennifer Dempsey, MPAS (right), and Holly Kinahan, ANP, MSN, OCN.

“They are our trusted partners in caring for patients with very complicated problems,” he states. “Patients go out of their way to tell me how lucky they are to have them working with us. They are superstars, passionate about the work they do.”

Patients and their family members and friends, in turn, have sent numerous letters expressing their gratitude to Dr. Falchook and his staff. “Dr. Falchook, we both appreciated your answering questions and dealing with our concerns with such kindness and patience,” is just one of many examples, as is this note: “Thank you for working so hard to help [patient]. He and I appreciated all that you did for us. He was a fighter, and you encouraged him. I know how much he valued your efforts. His goal was to find a way to help the next person with this awful disease. Maybe he did.”

CCTT continued from page 3

While Dr. Hong says that he cannot predict the impact of the Affordable Care Act on the future of their studies, he anticipates that under the new law, all insurance companies will have to pay for the standard of care while a patient is getting treatment on a clinical trial. “Currently, it is a hodgepodge, a patchwork of financial clearance throughout different states,” Dr. Hong says.

To gain more space to evaluate and treat patients, the CCTT is moving to the eleventh floor of the Main Building. A working committee is pooling input from the staff to coordinate the move, which is expected to happen sometime in the next year and a half. “The time that we spend with our patients is longer than with typical cancer patients, and the space will be crafted around the process, with more clinics and consultation rooms,” Dr. Hong says. “It will be nicer for our patients and also our research coordinators and nurses, who are the backbone of our clinical research operations.”
# Active Phase I Program Protocols

## June 2013

### Protocol | Drug Mechanisms | Principal Investigator
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EMD 1214063 | cMET inhibitor | Gerald Falchook, MD
VGX-100 or VGX-100 + bevacizumab | VEGF monoclonal antibodies | Gerald Falchook, MD
MED00639 | Monoclonal antibody as notch inhibitor | Gerald Falchook, MD
Vemurafenib + carboplatin + paclitaxel | BRAF inhibitor with alkylating agent and antimitotic agent | Gerald Falchook, MD
MEK11437E: rollover study to continue GSK1120212 | MEK inhibitor | Gerald Falchook, MD
Pazopanib + sorafenib | Angiogenesis inhibitor with HDAC inhibitor | Siquing Fu, MD, PhD
VT2725 | Wilms’ tumor gene product 1 (WT1) antigen peptide | Siquing Fu, MD, PhD
Adi-PEG 20 + cisplatin | Pegylated arginine deiminase (alkylating agent) + chemotherapy for metastatic melanoma or other argininosuccinate synthetase (ASS) deficient advanced solid malignancies | Siquing Fu, MD, PhD
SOR-C13 | 13-mer synthetic peptide (for advanced solid tumors that express TRPV6 ion channel) | Siquing Fu, MD, PhD
Oral BYL719 (+/- fulvestrant) | PI3K inhibitor (for tumors with mutant PIK3CA gene) | Stacy Moulder, MD
Dasatinib + crizotinib | BCR-ABL, c-KIT, EPHA2 and PDGFR(β) inhibitor with ALK, c-MET, and ROS1 receptor tyrosine kinase inhibitor | David Hong, MD
MK-8242 | HDAC2 inhibitor | David Hong, MD
Nab-paclitaxel, gemcitabine, + bevacizumab | Recombinant monoclonal antibody, nanoparticle albumin-bound paclitaxel, chemotherapy agent | David Hong, MD
ABT-348 alone, ABT-348 + carboplatin, or ABT-348 + docetaxel | Aurora kinase inhibitor and VEGF inhibitor with alkylating agent/chemotherapy or antimitotic agent | David Hong, MD
ISIS 481464 | Antisense oligonucleotide STAT3 inhibitor | David Hong, MD
AMG 337 | c-Met inhibitor (for C-Met-dependent tumors) | David Hong, MD
LY2606383 | CHK1 inhibitor | David Hong, MD
INC280 | c-Met inhibitor | David Hong, MD
Ipilimumab + imatinib mesylate | Anti-CTLA-4 antibody combined with c-Kit inhibitor | David Hong, MD
Vemurafenib + cetuximab + irinotecan | BRAF inhibitor + EGFR inhibitor and DNA topoisomerase I inhibitor (for BRAF V600E mutant advanced solid malignancies) | David Hong, MD
IMC-TRI (LY3022859) | TGFβI monoclonal antibody | David Hong, MD
DS-3032h | Oral MDM2 inhibitor | David Hong, MD
Sirolimus + cetuximab | mTOR inhibitor, anti-EGFR monoclonal antibody | Filip Janku, MD, PhD
Sirolimus or vorinostat with hydroxychloroquine | mTOR, HDAC inhibitors combined with autophagy inhibitor | Filip Janku, MD, PhD
Lapatinib with 1) sirolimus or 2) metformin | Tyrosine kinase inhibitor with mTOR inhibitor or antihyperglycemic agent | Filip Janku, MD, PhD
Sorafenib or crizotinib with vemurafenib | BRAF inhibitor combined with CRAF, BRAF, KIT, RET, VEGFR, PDGFR inhibitor | Filip Janku, MD, PhD
Anakinra denosumab or crizotinib + everolimus | IL-1R antagonist, anti-RANKL monoclonal antibody, or MET/ALK inhibitor combined with mTOR inhibitor | Filip Janku, MD, PhD
Ipilimumab + lenalidomide | Anti-CTLA-4 antibody combined with antiangiogenic agent | Filip Janku, MD, PhD
Dasatinib, bevacizumab, + metronomic paclitaxel | Src inhibitor + anti-VEGF monoclonal antibody + microtubule inhibitor | Filip Janku, MD, PhD
GSK2118436 + pazopanib | BRAF inhibitor + anti-angiogenic agent (BRAF mutation required) | Filip Janku, MD, PhD
Oral LSK974 | Wnt pathway inhibitor (for Wnt ligand-dependent malignancies) | Filip Janku, MD, PhD
BKM120 + MEK162 | PI3K inhibitor + MEK inhibitor | Filip Janku, MD, PhD
Doxil, bevacizumab + temsirolimus | Anthracycline antibiotic, monoclonal antibody, and mTOR inhibitor | Daniel Karp, MD
Tensiomycin pharmacodynamics | mTOR inhibitor | Daniel Karp, MD
Tensiomycin + metformin | mTOR inhibitors | Aung Naing, MD
Aerosol Interleukin-2 | IL-2 inhibitor (for pulmonary metastases) | Aung Naing, MD

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**TREATMENT PLANNING CONFERENCE**

Referring physicians and nurses who want to present patients for possible Phase I clinical trial inclusion are invited to attend the weekly treatment planning conference held every Wednesday from 8:00 a.m. to 9:00 a.m. in the Rotary House, first floor conference rooms A/B/C.

Emailing the patient’s name and record number to Kristie Lawhorn, RN, research nurse supervisor, by noon Tuesday is recommended, but not mandatory, to add a case to the meeting agenda.
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<th>Drug Mechanisms</th>
<th>Principal Investigator</th>
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<td>ATR-101</td>
<td>ATR-101 Achiral, lipophilic Acyl-CoA: cholesterol acyltransferase (ACAT) inhibitor (for adrenocortical carcinoma)</td>
<td>Aung Naing, MD</td>
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<td>Glutamine</td>
<td>Glutamine Amino acid, proinflammatory cytokine inhibitor (for patients with oral mucositis on mTOR inhibitor-based regimen for radiation-related esophagitis)</td>
<td>Aung Naing, MD</td>
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<td>Bevacizumab, temsirolimus alone and in combination with valproic acid or cetuximab</td>
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<td>Sarina Piha-Paul, MD</td>
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<td>MK-8669 (ridaroflorolimus) + MK-2206 and MK-8669 + MK-0752 doublets (MK-MK)</td>
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<td>Sarina Piha-Paul, MD</td>
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<td>Vemurafenib + everolimus</td>
<td>Vemurafenib + everolimus BRAF inhibitor combined with mTOR inhibitor</td>
<td>Vivek Subbiah, MD</td>
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<td>Vandetanib + everolimus</td>
<td>Vandetanib + everolimus EGFR/VEGFR/RET inhibitor and mTOR inhibitor</td>
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<td>Vemurafenib</td>
<td>Vemurafenib BRAF inhibitor (for patients with BRAF V600 mutation)</td>
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<td>DCVax-direct, autologous activated dendritic cells</td>
<td>Vivek Subbiah, MD</td>
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<td>Hepatic arterial infusion of oxaliplatin + systemic fluorouracil, leucovorin and bevacizumab with or without cetuximab</td>
<td>Apostolia Tsimberidou, MD, PhD</td>
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<td>Hepatic arterial infusion of irinotecan with: 1) systemic bevacizumab + cetuximab, 2) systemic bevacizumab + oxaliplatin, 3) systemic bevacizumab</td>
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<td>DCVax-direct, autologous activated dendritic cells</td>
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<td>BAX69</td>
<td>BAX69 Anti-macrophage migration inhibitory factor antibody</td>
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<td>Bevacizumab and temsirolimus with 1) carboplatin, 2) paclitaxel, 3) sorafenib</td>
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<td>Erlotinib + pralatrexate</td>
<td>Erlotinib + pralatrexate EGFR inhibitor combined with dihydrofolate reductase (DHFR) inhibitor</td>
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<td>Erlotinib + dasatinib</td>
<td>Erlotinib + dasatinib EGFR inhibitor and anti-metabolite</td>
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<td>Single-agent hormone blockade and combination with targeted agents</td>
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<td>LOR-253 HCI</td>
<td>LOR-253 HCI KLF-4 stimulator and angiogenesis inhibitor</td>
<td>Jennifer Wheler, MD</td>
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<td>OBI-139</td>
<td>OBI-139 Ribonuclease protein antagonist</td>
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<td>Brentuximab vedotin</td>
<td>Brentuximab vedotin Antimicrotubule CD30 antibody with protease-cleavable linker (for CD30-positive nonlymphomatous malignancies)</td>
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<td>Pazopanib + everolimus</td>
<td>Pazopanib + everolimus Angiogenesis inhibitor combined with PI3K inhibitor (PI3KCA mutation positive/PTEN loss)</td>
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<td>BAY 80-8246 + pacitaxel</td>
<td>BAY 80-8246 + pacitaxel PI3K inhibitor combined with microtubule inhibitor</td>
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<td>MGCD265 + erlotinib or docetaxel</td>
<td>MGCD265 + erlotinib or docetaxel c-MET, VEGFR-1,-2,-3, and tyrosine kinase inhibitor with EGFR inhibitor or antimetastatic agent (for patients with advanced NSCLC or other advanced cancers)</td>
<td>Jennifer Wheler, MD</td>
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<td>Pazopanib or pemetrexed with crizotinib</td>
<td>Pazopanib or pemetrexed with crizotinib Angiogenesis inhibitor or chemotherapy combined with ALK inhibitor</td>
<td>Ralph Zinner, MD</td>
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<td>Pazopanib + GS1120212</td>
<td>Pazopanib + GS1120212 VEGFR/PDGFR/RAF inhibitor combined with MEX inhibitor (enriched with patients having advanced differentiated thyroid cancer)</td>
<td>Ralph Zinner, MD</td>
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