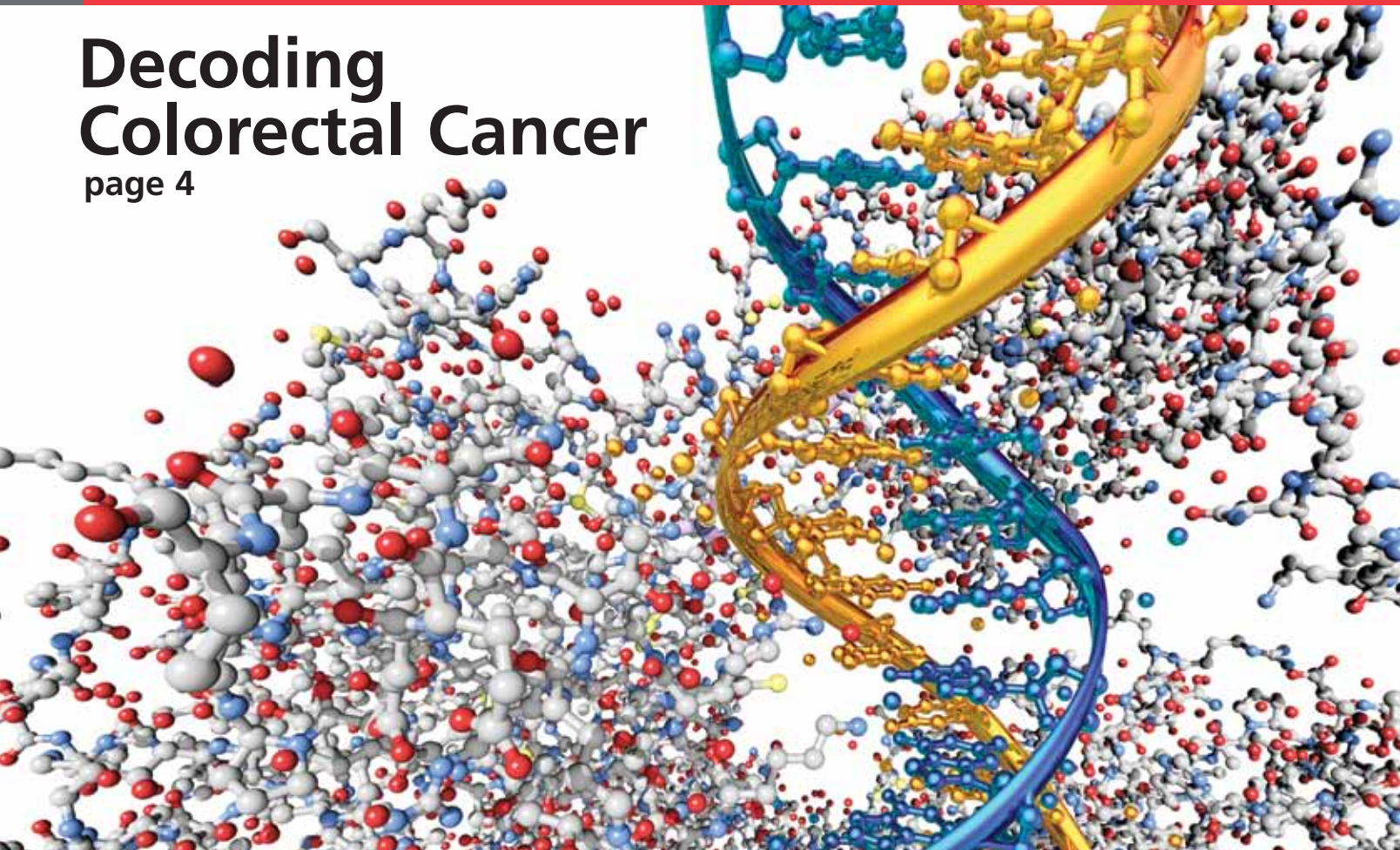


UH Innovations In Cancer

University Hospitals Seidman Cancer Center

Decoding Colorectal Cancer

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7 The benefits of a multidisciplinary, multimodality approach

Highlights and Happenings



Welcome to the Winter 2011 issue of *UH Innovations in Cancer*, presenting some of the latest developments at the Seidman Cancer Center, University Hospitals Case Medical Center. This issue features examples of our nationally recognized work in the field of colorectal cancer, from etiology, risk factors and the genetics of the disease through diagnosis, treatment and hope for long-term survivorship.

Highlights include an article about pioneering work in the field of colon cancer genetics by **Sanford Markowitz, MD, PhD**, Howard Hughes investigator and the Francis Wragg Ingalls Professor of Cancer Genetics. **Neal J. Meropol, MD**, the Lester E. Coleman, Jr. Professor of Cancer Research and Therapeutics, discusses the growing awareness that colon cancers represent a heterogeneous group of diseases with molecular differences that carry important information for the selection of optimal therapy. Just one example of our research in this area is a pilot study headed by **Smitha Krishnamurthi, MD**, of a nontoxic agent, vitamin D, in patients with colon cancer to determine whether it can increase expression of a key gene, first linked to colon cancer by Dr. Markowitz, with tumor suppressor activity.

Amitabh Chak, MD, Gastroenterologist, UH Case Medical Center, and Professor, Case Western Reserve University School of Medicine, reports on his extraordinary research into the genetics and treatment of familial Barrett's esophagus. **Conor P. Delaney, MD, PhD**, Jeffrey L. Ponsky Professor of Surgical Education, and Chief, Division of Colorectal Surgery, explains how the UH Seidman Cancer Center uses a multimodality approach employing interdisciplinary teams to provide optimal, highly coordinated, individualized therapy for patients with colorectal cancers.

This is a special time for us, as we highlight recent news that UH Ireland Cancer Center is now UH Seidman Cancer Center in recognition of an extraordinary gift from Jane and Lee Seidman.

I hope you enjoy this issue of *UH Innovations in Cancer*.

Warm regards,

Stanton L. Gerson, MD
Director, University Hospitals Seidman Cancer Center and
Case Comprehensive Cancer Center

UH Innovations in Cancer Winter 2011, Volume 2, Issue 1

Contributors: Stanton L. Gerson, MD; Nathan Levitan, MD; Amitabh Chak, MD; Conor P. Delaney, MD, PhD; Sanford Markowitz, MD, PhD; Neal J. Meropol, MD

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Among the nation's leading academic medical centers, University Hospitals Case Medical Center is the primary affiliate of Case Western Reserve University School of Medicine, a nationally recognized leader in medical research and education.

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SCHOOL OF MEDICINE
CASE WESTERN RESERVE
UNIVERSITY

The commitment to exceptional patient care begins with revolutionary discovery. Faculty at the Case Western Reserve University School of Medicine, who also are physicians at UH Case Medical Center, are at the forefront of medical research and innovation. The School of Medicine is the largest medical research institution in Ohio and among the nation's top medical schools for research funding from the National Institutes of Health.

Progress in Understanding Barrett's Esophagus

Basic research and clinical expertise combine to provide new insights

■ BY AMITABH CHAK, MD



Amitabh Chak, MD,
Gastroenterologist,
University Hospitals
Case Medical Center

Clinicians and scientists at Seidman Cancer Center, University Hospitals Case Medical Center are investigating the genetics, surveillance and treatment of Barrett's esophagus, in which abnormal columnar epithelium containing goblet cells replace the normal squamous epithelium of the distal esophagus. Barrett's esophagus is found in up to 10 percent of patients with GERD and is linked to increased risk of developing esophageal adenocarcinoma and gastroesophageal junctional adenocarcinoma. While most patients with Barrett's esophagus do not progress to adenocarcinoma, those who develop high-grade dysplasia may have a risk of 10 percent or more per patient-year of developing esophageal cancer.

A FAMILIAL ETIOLOGY

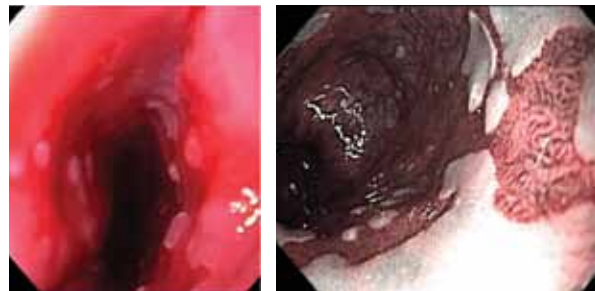
Our research team at UH Seidman Cancer Center, working in conjunction with the Familial Barrett's Esophagus (FBE) Consortium (see sidebar), identified the syndrome of FBE, which includes Barrett's esophagus and its associated cancers. We discovered that Barrett's esophagus, esophageal adenocarcinoma and gastroesophageal junctional adenocarcinomas aggregate in certain families; FBE is a complex genetic disease caused by a combination of underlying genetic and environmental factors.

The genetics of FBE are not yet clearly understood, but it is not simply a genetic susceptibility to GERD or obesity that in turn increases the risk of developing Barrett's esophagus. Segregation analysis of 881 pedigrees collected through the FBE Consortium recently provided extensive epidemiological evidence that supports the probability that FBE involves one or more rare, autosomally inherited dominant susceptibility alleles.

NONFAMILIAL BARRETT'S ESOPHAGUS

Nonfamilial forms of Barrett's esophagus and the associated cancers also occur. Obesity, Caucasian race, male gender, smoking and alcohol consumption are known risk factors for both the nonfamilial and familial forms, in addition to a history of GERD.

Several pathways have been implicated in Barrett's esophagus and esophageal adenocarcinoma. For example, one recent study from our laboratory points to a possible role for the insulin-like growth factor pathway. Other pathways that have been linked to Barrett's esophagus and esophageal cancer include E-cadherin silencing, the glutathione S-transferase family and epidermal growth factor receptor (EGFR) family signaling.



Left, Barrett's esophagus with squamous islands seen with white light endoscopy; right, same mucosa seen with narrow band imaging; characteristic capillaries are discernible as dark whorls.

MORE EFFECTIVE SCREENING

One of the reasons esophageal adenocarcinoma has such a poor five-year survival rate – less than 15 percent – is that it is often not detected until late stages, which currently lack highly effective treatments. At UH Seidman Cancer Center, we are trying to develop low-cost methods to screen for Barrett's esophagus using unsedated transnasal endoscopy.

Our center has also participated in a multicenter, sham-controlled study of interventional radiofrequency ablation of dysplastic Barrett's esophagus to see whether it would decrease the rate of neoplastic progression. Results were published in *The New England Journal of Medicine* in 2009. The patients who received radiofrequency ablation had a high rate of complete eradication of dysplasia (90.5 percent of those with low-grade dysplasia versus 22.7 percent in control group, $P < 0.001$; 81.0 percent of those with high-grade dysplasia versus 19.0 percent of control group, $P < 0.001$). They also had a reduction in disease progression (3.6 percent versus 16.3 percent, $P = 0.03$) and fewer cancers (1.2 percent versus 9.3 percent, $P = 0.045$).

Consortium Connection

The Familial Barrett's Esophagus Consortium, initiated in 1998, is a multicenter study to explore the epidemiology and genetics of Barrett's esophagus and adenocarcinoma. Patients with Barrett's esophagus or esophageal cancer are asked to complete a questionnaire detailing symptoms and family history and to provide a blood sample to be sent to a central NIH-approved repository for further genetic studies. For more information, please contact Wendy Brock, RN, at **216-844-3853** or **Wendy.Brock@UHhospitals.org**.

Unraveling the Secrets of Colorectal Cancer Genes

Recent research investigates their importance in diagnosis and treatment

■ BY SANFORD MARKOWITZ, MD, PHD



Sanford Markowitz, MD, PhD, Francis Wragg Ingalls Professor of Cancer Genetics, Department of Medicine (Hematology and Oncology), Seidman Cancer Center, University Hospitals Case Medical Center, and Case Western Reserve University School of Medicine

According to the most recent data from the American Cancer Society (ACS), colorectal cancer is the second leading cause of cancer-related deaths in the United States for men and women combined. The ACS estimates that more than 140,000 cases of colorectal cancer will occur in the United States in 2010 and lead to more than 51,000 deaths.

The Markowitz laboratory at the Seidman Cancer Center, University Hospitals Case Medical Center, and the Department of Medicine, Case Western Reserve University School of Medicine has focused on explicating genetic events that determine why certain individuals develop colon cancer, elucidating pathways that are normally involved in preventing cancer development, and developing tests for early detection of colon cancers and precancers at early stages when the disease is highly curable.

INHERITED DEFECTS

Together with other UH Seidman Cancer Center faculty, (**Georgia Wiesner, MD, Joseph Willis, MD, and Robert Elston, PhD**), we have led a study of families in which two or more siblings have developed colon cancer (or large precancerous polyps of at least 1 cm in size) by age 65 or younger. With the help of *CBS Evening News* anchor Katie Couric, this “colon cancer sibling study” has recruited colon cancer families throughout the country.

In 2003 our team discovered that many of the individuals who developed colon tumors had inherited the same abnormal region on chromosome 9. In 2009, further studies by our team (including new faculty members **Kishore Guda, DVM, PhD, and Thomas Gerken, PhD**) enabled us to identify a gene in this region, the GALNT12 gene, in which nonfunctional gene variants were inherited in certain individuals who developed colon cancers. GALNT12 is one of the genes required for the biochemical synthesis of intestinal mucins, which create the mucous barrier of the colon. Our hypothesis is that the defects in these cancer-associated GALNT12 variants create defective colonic mucins that promote gut inflammation and tumor development. As many additional gene encoded enzymes are required for the proper synthesis of intestinal mucins, we also expect this discovery is the tip of the iceberg.

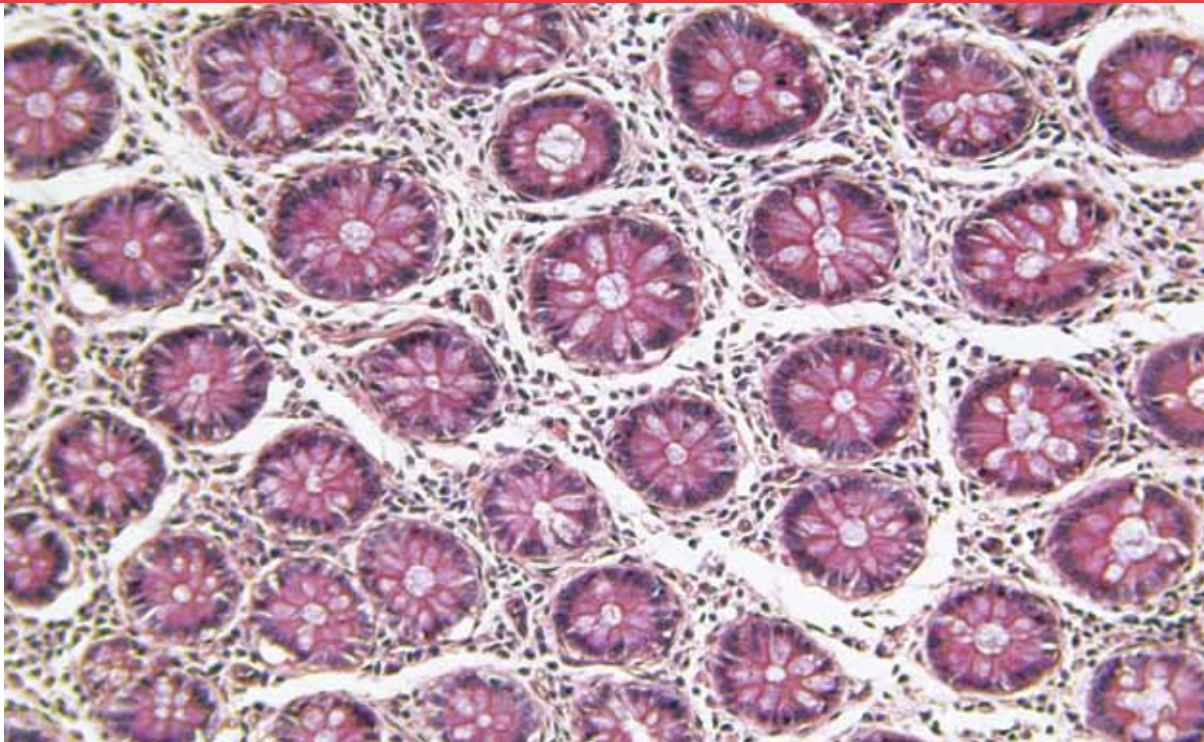
Further studies of the relationship of this gene family and inherited risk for colon tumor development are under way.

ROLE OF 15-PGDH

In 1995, my laboratory discovered that mutations that inactivate the transforming growth factor beta 1 (TGF-beta) pathway play an important role in the development of many colorectal cancers. TGF-beta receptors are colorectal cancer suppressor genes; our laboratory has shown that more than one-third of human colorectal cancers demonstrate mutations in these particular genes.

We recently discovered that a key effector of TGF-beta tumor suppression is 15-PGDH, a gene whose expression is directly controlled and activated by TGF-beta. Without an intact TGF-beta signaling pathway, 15-PGDH activity disappears. The 15-PGDH gene encodes the 15-hydroxyprostaglandin dehydrogenase (15-PGDH) enzyme, which provides the rate-limiting step in prostaglandin catabolism. By degrading prostaglandins, 15-PGDH opposes the activity of cyclooxygenase-2 (COX-2), an enzyme that is critical to the synthesis of prostaglandins, and whose increase is a critical early event in the development of colon tumors. COX-2 inhibitors (NSAIDs) can shrink colon polyps in human patients. 15-PGDH is able to exert a potent tumor suppressor activity by directly opposing the COX-2 colorectal cancer oncogene. When colon cancers lose the TGF-beta signaling pathway, the resulting loss of 15-PGDH activity leaves COX-2 activity unopposed and sets in motion the cellular changes leading to the development of colorectal cancer. In cell culture models, restoring wild-type 15-PGDH expression strongly suppresses the tumorigenic capacity of colorectal cancer cell lines. Conversely, in mice, knockout of 15-PGDH alleles confers marked susceptibility to colon tumor development.

Most recently, we discovered that certain individuals have very low levels of 15-PGDH in their colons. We studied a group of these individuals who had previously had premalignant colon adenomatous polyps, and were being treated with the drug celecoxib (an NSAID COX-2 inhibitor) to prevent new colon polyps from forming. Surprisingly, whereas celecoxib was highly



Bowel adenocarcinoma mucin secreting cancer cells

effective in preventing new colon tumors in individuals with high levels of colonic 15-PGDH, celecoxib was ineffective in individuals with low colonic 15-PGDH. We also showed the same result in mice that were genetically engineered to have low 15-PGDH levels. Key investigators in these studies included UH Seidman Cancer Center investigators **Joseph Willis, MD, Dawn Dawson, MD, Min Yan, PhD, Seung-Jae Myung, MD, Stephen Fink, PhD, Jill Barnholtz-Sloan, PhD,** and **Ronald Rerko, MS,** and Harvard Medical School investigator Monica Bertagnoli, MD.

These discoveries emphasize the importance of the TGF-beta and the 15-PGDH system in suppressing colorectal cancer. These genes may provide new targets for drug development or gene therapy approaches to the treatment or prevention of colorectal cancer. Measuring the amount of 15-PGDH present in colon cells may help us to identify individuals who might benefit from COX-2 inhibitors to prevent colorectal cancer formation. We are now attempting to develop drugs to increase or to reactivate 15-PGDH expression to prevent colorectal cancer in individuals at risk for the disease and to treat colon cancers.

EARLY DETECTION

Deaths from colon cancers could be prevented by screening more individuals. We have developed a new noninvasive test for early detection of colon cancers and colon adenomatous polyps. We first examined the human colon cancer genome to identify a marker DNA sequence that would be highly reflective of aberrant DNA methylation that is absent in the normal colon tissues.

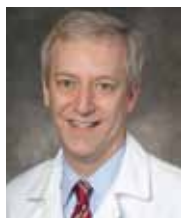
Our laboratory found such a sequence in the exon-1 of the vimentin gene. By using a sensitive methylation-specific polymerase chain reaction (PCR) assay, we were able to detect this aberrant methylation in DNA extracted from the stool of colorectal cancer patients. This test was able to detect 77 percent of early stage I and stage II colon cancers. The results were consistent regardless of the location of the colorectal cancer. A version of this test is now commercially available for use by patients and their physicians, and the American Cancer Society has added stool DNA testing to its approved lists of tests for screening for colon cancer. Our laboratory is now working to identify additional markers to further increase the sensitivity and specificity of stool DNA testing as a screening test for colorectal cancers.

Additionally, in 2009, in collaboration with investigators at Johns Hopkins University, we reported results of a colon cancer blood test based on detecting the methylated vimentin DNA in the blood of colon cancer patients. Nearly half of early stage colon cancer cases were detected by this approach. Studies are now planned to improve the sensitivity of this blood-based test, and to determine if it can provide a way to detect early recurrences of colon cancer following initial surgery. Key UH Seidman Cancer Center investigators in this work include **Joseph Willis, MD, Helen Moinova, PhD, Wei-Dong Chen, MD, Li Li, MD, PhD, Gregory Cooper, MD,** and **Amitabh Chak, MD,** along with Bert Vogelstein, MD, at the Sidney Kimmel Comprehensive Cancer Center at Johns Hopkins. To discuss colorectal research, call Sanford Markowitz, MD, PhD, at **216-368-1976**.

A Personalized Strategy

Advances made in selecting therapies and designing clinical trials for colon cancer

■ BY NEAL J. MEROPOL, MD



Neal J. Meropol, MD, Lester E. Coleman, Jr. Professor of Cancer Research and Therapeutics; Chief, Division of Hematology and Oncology, Associate Director for Clinical Programs, Seidman Cancer Center, University Hospitals Case Medical Center and Case Western Reserve University School of Medicine, Associate Director for Clinical Research, Case Comprehensive Cancer Center

Colon cancer is rapidly becoming a model for the personalized approach to cancer therapy. A “one size fits all” strategy to treatment selection is no longer appropriate. This new direction initially began with the expansion of our treatment armamentarium and the recognition that patient preference was an important determinant of treatment choice. For example, the side effect profiles of oxaliplatin and irinotecan differ markedly, whereas their efficacy in patients with advanced disease is similar. These differences in side effects (e.g., neuropathy vs. alopecia) led to personalized treatment decisions. More recently, tumor molecular characteristics have begun to drive the selection of colon cancer therapy.

The next step in developing a personalized treatment approach is to grow awareness that colon cancers represent a heterogeneous group of diseases that can be molecularly characterized; these differences are now known to be important considerations in the selection of optimal therapy for a given patient.

Our own research and that of our colleagues has taught us that RAS gene mutations confer resistance to epidermal growth factor receptor (EGFR) inhibitors. This knowledge led to clinical recommendations that patients with these mutations not be offered treatment with this class of drugs. Recent data have suggested that alterations in BRAF, a signaling protein downstream from RAS, may have similar effects, although this is still a controversial issue.

Another distinct group of colon cancers is characterized by microsatellite instability, a hallmark of deficient DNA mismatch repair. While it has been long known that defects in DNA mismatch repair characterize hereditary nonpolyposis colorectal cancer (HNPCC; Lynch syndrome), 15 percent of sporadic colon cancers also have this phenotype. These tumors may have different behavior as well as different responses to treatment. Tumors with defective mismatch repair tend to be more indolent, and there is evidence that they are resistant to treatment with 5-fluorouracil. As a result, there is general consensus that patients with dMMR stage II colon cancers should not receive adjuvant therapy with 5-FU. It is still an open question whether patients with this subset of colon cancers might benefit from different drugs in either the adjuvant or metastatic disease settings.

Our colleague, **Sanford Markowitz, MD, PhD**, recently discovered a new tumor suppressor gene, 15-hydroxyprostaglandin dehydrogenase (15-PGDH). This gene may impact the behavior and treatment response of colon cancers. **Smitha Krishnamurthi, MD**, in collaboration with Dr. Markowitz, is initiating a pilot study of vitamin D in patients with resectable colorectal cancer to see whether it can increase expression of the 15-PGDH gene. We hope this clinical trial will lead to a novel nontoxic approach to colon cancer treatment and prevention.

We are also exploring the use of circulating tumor cells as an aid in determining colon cancer treatment effectiveness and disease prognosis. In collaboration with the National Surgical Adjuvant Breast and Bowel Project (NSABP), and my colleague Steven J. Cohen, MD, of the Fox Chase Cancer Center, we are studying whether the presence of circulating tumor cells can be used in selecting treatment for patients with potentially resectable hepatic colorectal metastases (NCI trial NSABP-C-11). In addition, I am chairing a study through the Eastern Cooperative Oncology group that seeks to determine whether expression of thymidylate synthase can be used to select initial treatment for metastatic disease (ECOG 4203).

Another exciting area is the use of molecular profiling to stratify prognosis in patients with early stage colon cancer. Recent reports regarding gene expression profiling for patients with stage II colon cancer suggest the possibility that we will be soon integrating molecular features beyond mismatch repair in our decisions regarding adjuvant therapy.

The next generation of clinical trials in colon cancer must integrate the latest molecular insights in order to accelerate our progress against this disease. In recognition of the biologic complexities that bear on the design of these studies, the NCI is sponsoring a forum on this topic in early 2011. I have the honor to be one of the chairs of this meeting and we are excited about the opportunity to bring together the world's experts in a collaborative effort to bring the latest discoveries to patients. To discuss the future of colorectal treatments, contact Neal J. Meropol, MD, at **216-983-4752**.

Interdisciplinary, Multimodality Cancer Treatment

Patients are benefiting from the coordinated care of a team of experts

■ BY CONOR P. DELANEY, MD, PHD

At Seidman Cancer Center, University Hospitals Case Medical Center, patients with colorectal cancer are treated by multidisciplinary teams that offer individualized, multimodality approaches to cancer treatment.

Our surgeons coordinate the care of our patients with the network of experts in oncology available through UH Seidman Cancer Center. The team for a patient with colorectal cancer may include a surgical oncologist, gastrointestinal radiation specialist and medical oncologist, in addition to nursing and other health care support staff. These specialists are led by **Neal J. Meropol, MD, Smitha Krishnamurthi, MD,** and Chair of Radiation Therapy **Mitchell Machtay, MD.** These teams routinely review each patient's preoperative evaluations to select an initial course of treatment. As our knowledge of tumor biology continues to grow, we have more and more treatment options to consider, and this multidisciplinary approach allows us to weigh these choices and recommend the optimal strategy at any stage of colorectal cancer. This individualized approach may include any of a wide variety of therapeutic options, including surgical resection, intraoperative radiotherapy (IORT), external beam radiotherapy (EBRT), chemotherapy and molecular targeted drug therapy.

SURGICAL RESECTION

Whenever possible, we offer minimally invasive colon cancer surgery, which has been shown to safely and cost effectively accelerate postoperative recovery and reduce postoperative complications, without affecting tumor recurrence rate or overall cancer survival rate. Hospital stay is reduced by an average of two days, and time to full recovery by about 50 percent. Our patients recover from surgery with minimal pain and scarring and can more quickly regain a normal quality of life. Individuals should be evaluated by a surgeon experienced in minimally invasive surgery to be considered for a laparoscopic approach.

ADJUVANT CARE

Additional adjuvant care is considered in relation to the TMN stage and resection margins reported by the

pathologist. Patients with high-risk stage II cancers (such as those with T4 lesions or poorly differentiated histology) and those with stage III colorectal cancer are typically offered adjuvant therapies to reduce the risk of disease recurrence. Adjuvant chemotherapy is associated with an approximately 30 percent reduction in the risk of disease recurrence and a 20 percent to 30 percent decrease in mortality in patients with stage III cancer.

Advanced metastatic colorectal cancer has been associated with a poor prognosis, but recent advances in chemotherapy treatment options have led to improvements in two-year survival rates. There are many new cytotoxic chemotherapy and targeted agents available for treating metastatic colon cancer, such as 5-FU, oxaliplatin, capecitabine, irinotecan, cetuximab and bevacizumab; these may afford some patients with locally advanced and recurrent colon cancer a chance for long-term survival.

DETERMINING GENE STATUS

Teaming with experts in the diagnosis of gene status allows us to determine which patients are likely to benefit from certain new treatments. National Comprehensive Cancer Network Guidelines now recommend that KRAS gene status of either the primary tumor or a site of metastasis should be determined as of the pre-treatment work-up for all patients diagnosed with metastatic colorectal cancer. These guidelines recommend that only patients with tumors that are characterized by the wild-type KRAS gene receive epidermal growth factor receptor (EGFR) inhibitors such as cetuximab and panitumumab. A number of recent studies have demonstrated that the tumor KRAS gene status is highly predictive of outcome with anti-EGFR therapies. As we learn more about the genetics of colorectal cancer, we can anticipate additional routine analyses of tumor gene status to guide the selection of therapy.

This cooperative approach to multimodality therapy means that our patients experience a much more comprehensive, optimal approach to colorectal cancer treatment. To consult on a patient with colorectal cancer, contact Conor Delaney, MD, PhD, FRCSI, FACS, FASCRS, at **216-844-8087**.



Conor P. Delaney, MD, PhD, FRCSI, FACS, FASCRS (Hon), Jeffrey L. Ponsky Professor of Surgical Education; Chief, Division of Colorectal Surgery; Vice-Chair, Department of Surgery; Director, Institute for Surgery and Innovation; University Hospitals Case Medical Center and Case Western Reserve University School of Medicine



NEWS UPDATE

An Inside Look at UH Seidman Cancer Center

Stanton Gerson, MD, Director of the new University Hospitals Jane and Lee Seidman Cancer Center, discusses the \$260 million, 375,000-square-foot center, set to open in spring 2011 at the UH Case Medical Center campus.

Innovations in Cancer: What led to the name for the new center?

Stanton Gerson, MD: In recognition of the extraordinary generosity of Jane and Lee Seidman, who donated \$42 million to support this new cancer hospital, UH is naming the Jane and Lee Seidman Cancer Center in their honor. We are also calling our integrated network of nine outpatient cancer programs located throughout the region the University Hospitals Seidman Cancer Center.

In the last year I had the opportunity to sit down with the Seidman family and share our vision. I saw firsthand their excitement with the uniqueness of the new cancer hospital and the impact it will have and how much pride they had in placing their name on it.

UH Seidman Cancer Center builds on the legacy established by the late R. Livingston Ireland Jr., a UH Case Medical Center board member who lobbied vigorously for significant state funding to further UH's cancer program in the early 1980s. In recognition of Mr. Ireland's successful efforts the cancer center was named in his honor. A lasting tribute will be displayed prominently in the UH Seidman Cancer Center.

Innovations: Tell us more about the design of the hospital.

Dr. Gerson: The UH Seidman Cancer Center is a unique facility that will allow us to offer the highest quality family- and patient-centered care. We began by ensuring ease of access and a very efficient use of space so that our patients will be able to move easily between appointments. Our multidisciplinary physician team approach to care will make use of specially designed, disease-specific clinical pods. The overall environment is also important to the patient experience, and the building incorporates a mix of smaller, private spaces and larger, more open areas, as well as a west-facing glass wall that brings in ambient light.

Innovations: How will the new UH Seidman Cancer Center facilitate patient access to clinical trials?

Dr. Gerson: We feel strongly that access to clinical trials is an important aspect of providing exceptional care, and our patients



currently have access to more than 300 trials. The new, integrated spaces will make it easier for our clinical investigators to interface with patients. The layout also provides places for our research nurses in the exam and treatment areas.

Innovations: What types of advanced technology will be in the new facility?

Dr. Gerson: The UH Seidman Cancer Center has been designated an "Imaging Center of Excellence," by the equipment manufacturer Philips Inc., with more than \$30 million in state-of-the-art equipment to be used exclusively for cancer patients. We will have one of only four PET-MR imaging machines in the world and the only one at a cancer center. The facility also enables us to offer intraoperative MRI, and it will also house our current image-guided radiation therapy (CyberKnife®) and our da Vinci Robotic Surgery System.

Your Feedback Is Important

As a medical professional, your input is invaluable in helping us shape future issues of *UH Innovations in Cancer*. We want to know what's important to you. Do you want to read about cutting-edge research, learn about the latest technology, or hear firsthand case studies of how others in your specialty are improving and saving lives? Tell us what you want to read about and your name will be entered to **win one of two Apple iPads!** Simply visit UHhospitals.org/innovations.



Image courtesy of Apple