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For Immediate Release

**'PERSONALIZED MEDICINE' ADVANCES COLON CANCER TREATMENT**  
***Testing for K-RAS gene mutation helps pinpoint individual therapies***

**Plainview, NY, June 2009** – Colon cancer is the second leading cause of cancer deaths in the United States today. If you're one of the 150,000 cases of colon cancer diagnosed each year, a new test for K-RAS gene mutation will help your oncologist identify the medicines that will help you most. That's the recent conclusion of both the American Society for Clinical Oncology and the National Comprehensive Cancer Network.

Olga Falkowski, MD, a board-certified pathologist and associate medical director of Acupath Laboratories, Inc., which conducts leading-edge molecular and immunohistochemical testing, agrees. "Until recently, treatment for colon cancer patients was determined by results of controlled clinical trials. Now, advances in the identification and understanding of molecular and biochemical events leading to the development of cancer and tumor growth, mean cancer treatments can be targeted to an individual's own biology."

**What are K-RAS genes?**

K-RAS is a protein encoded by the K-RAS gene. The protein product of the unmutated K-RAS gene performs an essential function in normal tissue signaling. Mutated K-RAS genes, however, are potent "oncogenes" (genes that speed up cell division) that play a role in many cancers. A single amino acid substitution is responsible for activating a mutation. The transforming protein that results is implicated in various malignancies, including colorectal cancer.

**Why is gene mutation testing important?**

Dr. Falkowski explains, "Gene mutation testing is currently the most reliable way to predict whether a colon cancer patient will respond to one of the EGFR inhibiting drugs – such as cetuximab (Erbix) and panitumumab (Vectibix) – most often used in colon cancer treatment." (EGFR stands for epidermal growth factor receptor.)

"K-RAS gene mutation testing is a significant step on the path to offering cost-effective, individualized treatments for those with colon cancer," she concludes.

**What does this mean for colon cancer treatment?**

A study recently published in the *New England Journal of Medicine* reports that patients without the gene mutation usually respond favorably to treatment by the EGFR drugs, while patients with the mutated gene do not. The study also revealed that between 20 percent and 50 percent of colon tumors test positive for the mutated K-RAS gene.

Patients with advanced, metastatic colon cancer who had failed standard chemotherapy for their disease and for whom no further treatments were available were randomly assigned to receive either cetuximab or no further treatment (except best supportive care) for their disease.

Patients treated with cetuximab continued the medication until the cancer progressed or they could tolerate the therapy no longer. Then researchers examined samples of the patients' tumors for the K-RAS gene mutation, finding four-out-of-ten patients in both groups had it.

More importantly, they discovered:

- Patients with the K-RAS mutation survived an average of about 4.5 months regardless of whether or not they received the cetuximab.
- Patients without the K-RAS mutation survived an average of about 9.6 months if they were treated with cetuximab and only 4.8 months if they were provided only supportive care.

Patients without the mutated K-RAS gene doubled their survival time by receiving cetuximab. These patients also did better than the others on quality of life scores.

### **What does this mean to me?**

"Now there's an identified genetic marker that predicts – although it doesn't assure – a specific treatment that will prolong life and its quality for the majority of advanced colorectal cancer patients without the K-RAS gene mutation," says Dr. Falkowski. "Physicians are also exploring cetuximab as a 'first line' treatment for colon cancer, along with chemotherapy, and not simply for use when no further treatment options are available. Knowing this, colon cancer patients can work with their doctors to receive timely testing and the best possible treatment for their disease."

### **What causes colorectal cancer?**

Although the exact causes of colorectal cancer are not known, researchers are beginning to understand how certain changes in DNA can cause normal cells to become cancerous. DNA is the chemical in each of our cells that makes up our genes – the instructions for how our cells function. Some genes contain instructions for controlling when our cells grow, divide and die. Genes that speed up cell division are called "oncogenes." Others that slow down cell division or cause cells to die are called "tumor suppressor genes." Cancers can be caused by DNA mutations that "turn on" oncogenes or "turn off" tumor suppressor genes.

In most cases of colorectal cancer, the DNA mutations that lead to cancer are acquired during a person's life rather than having been inherited. And while there doesn't seem to be a single pathway to colon cancer, in many cases the first mutation occurs in the APC gene. This leads to an increased growth of colorectal cells because of the loss of the "brake" on small cell growth. Further mutations may then occur in genes such as K-RAS, leading cells to grow and spread uncontrollably.

### **Bio: Olga Falkowski, MD.**

Dr. Falkowski is board-certified in anatomic and clinical pathology by the American Board of Pathology. She is the Unit Chief of Breast Pathology and the Associate Medical Director of Acupath Laboratories in Plainview, NY. Before joining Acupath in 2005, Dr. Falkowski served as an attending pathologist at the Long Island Jewish Medical Center. Prior to that, she served as an attending pathologist at New York University School of Medicine where she also fulfilled a surgical fellowship. Dr. Falkowski received her medical degree from and completed her residency in general pathology at the First Moscow Medical School in Russia. Subsequently, she fulfilled her anatomic and clinical pathology residency at St. Luke's-Roosevelt Hospital Center and University Hospital of Columbia University, both in New York City.

Dr. Falkowski has served as an assistant professor at New York University School of Medicine, Hofstra University, and the Albert Einstein College of Medicine. She is currently a member of the College of American Pathologists and the United States and Canadian Academy of Pathology.

Dr. Falkowski is an author and lecturer in breast pathology and frequently talks with the media on issues concerning breast cancer diagnostics. See [www.acupath.com](http://www.acupath.com) for more information.