Research collaboration was the watchword at the 2010 Albert Einstein Cancer Center Advances retreat, held in May. Jeffrey Segall, Ph.D., professor of anatomy and structural biology and the Betty and Sheldon Feinberg Senior Faculty Scholar in Cancer Research, encouraged the 110 attendees from many different Einstein departments to look for opportunities to collaborate with their colleagues. Affirming the importance of teamwork in research, AECC director Dr. I. David Goldman reminded center members of the importance the National Cancer Institute assigns to the synergy of collaborative research when evaluating cancer centers. Dr. Goldman thanked the AECC’s generous supporters, acknowledging a recent $500,000 gift from Myles P. Dempsey Sr. and his wife, Jane, that will help fund pilot projects aimed at developing new approaches to diagnosing and treating breast cancer.

Among the attendees at the AECC Advances meeting were, from left: Rose Dempsey Dahlman; John S. Condeelis, Ph.D., the Judith and Burton P. Resnick Chair in Translational Research and coleader of the AECC’s Breast Cancer Working Group; Jeffrey W. Pollard, Ph.D., the Louis Goldstein Swan Chair in Women’s Cancer Research; Dr. Goldman; Jane A. Dempsey; Myles P. Dempsey Sr., founder and chairman of Tech Air, a leading regional provider of industrial, medical, and specialty gases; Myles P. Dempsey Jr., president of Tech Air; and Joseph A. Sparano, M.D., coleader of the Breast Cancer Working Group. (Dempsey family members not pictured: Marilyn Dempsey, Craig Dalhman, Katy Dempsey Haley, Kevin Haley, Kelly Dempsey Connolly, Mark Connolly, Jennifer Dempsey Torre and Edward Torre.)

Blood Boosters

Patients with MDS and acute myeloid leukemia (AML) often develop thrombocytopenia, a deficiency of blood platelets. Platelets are necessary for the prevention of bleeding, and thrombocytopenia can be life-threatening in these patients. A new drug was recently approved for treating thrombocytopenia in people with hepatitis C. The drug works by stimulating the bone marrow to produce new blood cells. Researchers led by Ulrich Steidl, M.D., Ph.D., assistant professor in the department of cell biology and the Diane and Arthur B. Belfer Faculty Scholar in Cancer Research, have now shown that the drug effectively and safely boosts platelets in patients with MDS and AML. The study was published in the October 2009 issue of Blood. A clinical trial with this drug will be initiated soon.

New, Improved Inflammation Model

In one in five patients with inflammatory bowel disease (IBD), the condition progresses to colorectal cancer. Chronic inflammation is thought to be the main culprit when IBD morphs into cancer. But just how inflammation leads to tumor development hasn’t been clear. Now, AECC researchers led by Elaine Lin, Ph.D., assistant professor in the department of medicine (oncology), have created a mouse model in which chronic inflammation spontaneously develops in the colon, followed by cancer in the inflamed regions. The novel mouse model has revealed a previously unrecognized molecular pathway that plays a key role when chronic inflammation gives rise to tumors, according to a study Dr. Lin and her colleagues published in the February 2010 issue of the American Journal of Pathology.

Reining In Runaway Cell Growth

In normal cells, a tumor-suppressing protein called the retinoblastoma protein (pRb) silences certain genes, preventing uncontrolled growth and division of cells. But when pRb is inactivated, the genes it normally silences turn on—resulting in cell proliferation and tumors. One way to halt cell growth would be to silence pRb’s target genes when pRb is inactivated. Liang Zhu, M.D., Ph.D., and his colleagues have done just that; by disabling Skp2, a cancer gene normally kept in check by pRB, they were able to prevent tumors from developing in mice that lacked pRB. Their findings, published in the January 2010 issue of Nature Genetics, suggest that Skp2 might be a good target for drugs that could treat or prevent tumors when pRB can’t do its cancer-control job.