PEOPLE


Aaron Washington

William C. Hahn, MD, PhD, deputy chief scientific officer at Dana-Farber Cancer Institute (DFCI) in Boston, MA, began his 1-year term as president of the American Society for Clinical Investigation (ASCI) in April. With more than 2,800 elected members representing all specialties, the ASCI is dedicated to advancing research to improve treatment of human diseases. The organization also publishes the peer-reviewed *Journal of Clinical Investigation.*

Hahn, who is also chief of molecular and cellular oncology at DFCI and director of its Center for Cancer Genome Discovery, studies the genetic interactions that lead to malignant transformation of cells. His laboratory has developed experimental models for the study of normal and malignant epithelial cell biology and helped develop genome-scale tools that permit the manipulation of genes in a comprehensive manner. Using these tools, he is identifying and validating new targets for translational cancer research.

Robert A. Bradway, MBA, assumed the role of chief executive officer of Amgen (Thousand Oaks, CA) in May, replacing Kevin W. Sharer, who will retire at the end of the year. Amgen identifies, isolates, produces, and uses human proteins as therapeutic agents in the treatment of various cancers and other serious illnesses. The company boasts 17,000 employees at facilities in dozens of countries. Its 10 approved drugs generated $15.3 billion in sales in 2011.

Bradway joined Amgen in 2006 as a vice president in operations and worked his way up to chief financial officer in 2007 and then president and chief operating officer in 2010. Before joining Amgen, he spent 19 years at Morgan Stanley.

A Moving Picture of Cancer Metabolism

Metabolic analysis of 60 human cancer cell lines has produced a large set of data about what nutrients cancer cells take up and release over time. Researchers affiliated with Harvard Medical School and their colleagues who put together the dataset have used it to identify an important role for the amino acid glycine in the proliferation of cancers.

Most previous studies of cancer metabolism have provided a single snapshot of the nutrients and byproducts present in cells at one moment in time. These studies can provide information about the presence or absence of tens of thousands of metabolites within the cell but trying to understand them may be like trying to determine traffic flow in a city by looking at an aerial photograph.

To get a moving picture of cancer metabolism, the scientists created a database of metabolites consumed and released by cancer cells over time (Science 2012;336:1040–4.) The result is one of the first kinetic portraits of cancer-cell metabolism.

The group took measurements on the NCI-60, a collection of 60 human cancer cell lines from 9 tumor types, which let them leverage the large body of research already carried out on these cells, including an atlas of NCI-60's gene-expression profiles. They took samples from the growth media supporting the cells and employed mass spectrometry to measure levels of 219 metabolites over time.

Vamsi Mootha, MD, professor of systems biology at Harvard Medical School and senior author on the study, hopes that other researchers will use the metabolomics data to find trends in the metabolic uptake and release data.

The association between glycine uptake and rapid proliferation of cancer cells was striking, says Mootha—particularly because noncancerous cells with comparable rates of proliferation were found to *release* glycine.

To identify the pathways that underlie the cancer cells' reliance on glycine, scientists examined the gene-expression patterns of 1,425 metabolic enzymes in a previous NCI-60 study. Two pathways related to the amino acid stood out. When they silenced key enzymes in these pathways in 2 cancer cell lines, the proliferation rate slowed.

The researchers then turned to databases of gene expression in 1,300 patients with early-stage breast cancer. They found that above-median expression of 3 enzymes in the glycine-synthesis pathway were as strongly associated with mortality as are lymph node status and tumor grade. ■

Sequencing Spots Residual Leukemia

T-lineage acute lymphoblastic leukemia/lymphoma (T-ALL) is an aggressive blood cancer that accounts for 20% to 30% of all acute leukemia. Choosing the correct treatment for a patient is difficult, with some suffering needless side effects from overtreatment and others facing the possibility of relapse because of insufficient treatment.

Harlan Robins, PhD, a computational biologist at Fred Hutchinson Cancer Research Center (Seattle, WA), and colleagues evaluated high-throughput sequencing (HTS) of lymphoid receptor genes as a way to diagnose T-ALL and assess patients for minimal residual disease (MRD), in which leukemia cells can still be detected in the bone marrow using sensitive tests (Sci Transl Med 2012;4:134ra63).

The current standard in the United States for assessing MRD is multiparametric flow cytometry; real-time quantitative PCR is another option. However, interpretation of flow cytometry data is highly subjective, the antibodies used are expensive, and the method is not very sensitive. Quantitative PCR, though highly sensitive, is expensive and labor intensive due to the need to develop patient-specific primers.

Robins and his colleagues used an Illumina Hi-Seq platform and proprietary analytic tools developed at the University of Washington and licensed exclusively to Adaptive Biotechnologies of Seattle, WA, to sequence the variable regions of the *TCRB* and