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MUSC/Hollings Cancer Center

Andrew S. Kraft, MD, a nationally recognized prostate cancer physician and researcher, begins work this month as the director of the University of Arizona Cancer Center and associate vice president for oncology programs for the University of Arizona Health Sciences Center. He replaces Anne E. Cress, PhD, who has been interim director since July 2013. For the past decade, Kraft led the Hollings Cancer Center at the Medical University of South Carolina in Charleston, which earned distinction as an NCI-designated cancer center in 2009.


MD Anderson Cancer Center

Bruce D. Minsky, MD, a professor of radiation oncology and the deputy division head and director of clinical research in the Division of Radiation Oncology at The University of Texas MD Anderson Cancer Center in Houston, will begin a 1-year term as president of the American Society for Radiation Oncology on September 16. A graduate of the University of Massachusetts Medical School in Worcester, Minsky has made significant contributions to the clinical management of esophageal and rectal cancer. Widely published, he serves on the editorial boards of several journals.


Michigan State University

Joseph R. Haywood, PhD, a professor of pharmacology and toxicology and assistant vice president for regulatory affairs at Michigan State University in East Lansing, began a 1-year term as president of the Federation of American Societies for Experimental Biology in July. He has served on the Council of the Association for the Assessment and Accreditation of Laboratory Animal Care International and the Board of Governors of the International Council for Laboratory Animal Science.

Beleodaq Approved for Rare Lymphomas

The FDA approved Beleodaq (belinostat; Spectrum Pharmaceuticals) on July 3 for the treatment of patients with relapsed or refractory peripheral T-cell lymphomas (PTCL), a rare and aggressive group of diseases that accounts for 10% to 15% of all non-Hodgkin lymphomas.

PTCL is difficult to treat, and patients often relapse after first-line treatment, usually combination chemotherapy such as CHOP (cyclophosphamide, doxorubicin, vincristine, and prednisolone). "Anything we can add to the armamentarium is helpful," says Eric D. Jacobsen, MD, an oncologist at Dana-Farber Cancer Institute in Boston, MA.

Beleodaq inhibits histone deacetylases (HDAC), enzymes involved in the regulation of genes linked to tumorigenesis and cancer progression. It is the third drug to receive FDA approval for PTCL since 2009. Folotyn (pralatrexate; Spectrum Pharmaceuticals) received accelerated approval in 2009 for patients with relapsed or refractory PTCL, and Istodax (romidepsin; Celgene), another HDAC inhibitor, was approved in 2011 for patients who had received at least one prior therapy. Beleodaq received accelerated approval, a designation given to drugs that fill an unmet medical need.

The FDA based its approval on results from a multicenter, phase II trial of 120 evaluable patients with relapsed or refractory PTCL. In the trial, 10.8% of patients experienced a complete response and 15% had a partial response. The most common adverse effects, which occurred in more than a quarter of study participants, included nausea, fatigue, pyrexia, anemia, and vomiting.

Jacobsen, an investigator on an earlier phase II trial of Beleodaq, notes that in the BELIEF trial, which led to the FDA approval, the overall response rates were comparable to those of Folotyn and Istodax. The response rate was even higher—45.5%—in patients with angioimmunoblastic T-cell lymphoma, a common subtype of PTCL. Jacobsen called those results "very encouraging."

As part of the approval process, the drug's sponsor, Spectrum Pharmaceuticals, is required to conduct a phase III trial to compare the efficacy of Beleodaq combined with CHOP versus CHOP alone, to establish whether the drug would be effective in first-line treatment as well. ■

Analysis Finds Value in Pseudogenes

In a large, systematic analysis of the role of pseudogenes across seven types of cancer, researchers from The University of Texas MD Anderson Cancer Center in Houston have found that pseudogenes alone and in combination with other clinical and molecular markers may help define prognostic subgroups.

The findings point to new avenues of research into the roles pseudogenes may be playing in driving the growth of tumors, says principal investigator Han Liang, PhD, assistant professor of bioinformatics and computational biology (Nat Commun 2014 July 7 [Epub ahead of print]).

Pseudogenes, sometimes labeled as "junk" DNA, exist in the human genome in about the same abundance as protein-coding genes. Accumulated sequence changes prevent pseudogenes from encoding proteins, yet they are still transcribed into RNA sequences and, in turn, may play regulatory roles in cancer.

For instance, recent smaller studies have identified cases of transcribed pseudogenes acting as decoys that attract microRNAs away from tumor suppressor genes, such as *PTEN*, increasing their activity. Pseudogene transcription may also result in gene silencing, says Liang.

"People are beginning to realize that pseudogenes have functional effects," says Zhaolei Zhang, PhD, professor of molecular genetics at the University of Toronto, who did not participate in the study, but who helped characterize the abundance of pseudogenes in 2003.

To assess the role of pseudogenes in cancer more broadly, Liang analyzed data from The Cancer Genome Atlas (TCGA), which provided a snapshot of transcript levels in over 2,600 tumor samples from patients with seven types of cancer—breast, brain,

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