PEOPLE

The University of Texas System

Cancer geneticist **Ronald A. DePinho, MD,** assumed his role as president of the University of Texas M.D. Anderson Cancer Center on September 1. He formerly served as professor of medicine (genetics) at Harvard Medical School and as director of the Belfer Institute for Applied Cancer Science at Dana-Farber Cancer Institute. He is a member of the Institute of Medicine of the National Academies and a fellow of the American Academy of Arts and Sciences.

DePinho's research focuses on the biological and molecular processes behind aging and the development of cancer and degenerative diseases. For example, he has shown that telomere dysfunction activates p53, leading to age-related disorders, and that dysfunctional telomere-related phenotypes, such as brain atrophy, can be reversed in mice. DePinho has also studied the c-myc and FOXO transcription factors.

DePinho succeeds John Mendelsohn, MD, who stepped down as president of M.D. Anderson after 15 years.

Duke University Medical Center

Michael B. Kastan, MD, PhD, former director of the Comprehensive Cancer Center at St. Jude Children's Research Hospital, started his position as the first executive director of the newly launched Duke Cancer Institute on August 1. He had directed St. Jude's cancer center since 2004.

In the 1990s, Kastan published a series of papers describing p53 and its role in cellular repair and responses to damage. The findings launched discoveries that have provided a greater understanding of cancer's causes and new approaches to its treatment. His other research interests include radiation biology and the determinants of chemosensitivity and radiosensitivity.

Kastan was elected to the Institute of Medicine of the National Academies in 2009.

Mesenchymal Stem Cells May Boost Solid Tumors

Do mesenchymal stem cells (MSC) help to slow solid-tumor growth, or accelerate it? A new study of MSCs in human solid-tumor samples sheds light on the controversy by pinpointing carcinoma-associated MSCs (CA-MSC) that assist in expanding ovarian tumors.

University of Michigan scientists found CA-MSCs in 14 of 15 primary human ovarian cancer tumor samples (J Clin Invest 2011;121:3206–19). These cells were nontumorigenic and, like MSCs from normal cells, could differentiate into adipose tissue, bone, or cartilage. Their gene expression patterns, however, differed from those of MSCs from normal cells, with significantly greater expression of bone marrow protein (BMP) growth factor proteins.

When injected into immunocompromised mice, the CA-MSCs promoted tumor growth more than did control MSCs. Further, both *in vitro* and *in vivo* tests suggested that CA-MSCs boost production of cancer stem cells. Production of cancer stem cells also climbed in separate tests when tumor cells were treated with BMP family member BMP2. Conversely, treating tumor cells with the BMP inhibitor Noggin partially cut the CA-MSC effect on cancer stem cell production, suggesting BMP signaling may be a therapeutic target.

Given the important role of the BMP family in maintaining healthy bones, treating ovarian cancer by systematically delivering BMP inhibitors may not be workable. "But you might be able to target therapy to the tumor vascular niche, where the mesenchymal stem cells and cancer stem cells live, with signaling peptides or nanoparticles or another mechanism," says senior author Ronald Buckanovich, MD, PhD. ■

Mutation Linked to Cancers, Unstable Chromosomes

Most human cancers are aneuploid (containing an abnormal number of chromosomes). But the links between aneuploidy and cancer are not well understood, particularly the mechanisms driving chromosomal instability during tumorigenesis.

Scientists now have identified mutations of the *STAG2* gene, whose protein normally aids in chromosome segregation during cell division, as common in several types of solid human tumors (Science 2011;6045:1039–43). Moreover, the investigators demonstrated that mutations of *STAG2* can kick-start the aneuploid process.

A Georgetown University Medical Center team found that the STAG2 protein was missing in 20% of the glioblastoma, malignant melanoma, and Ewing sarcoma samples they studied. They then showed that knocking out *STAG2* in a chromosomally stable human cell line could trigger aneuploidy. Conversely, when aneuploid glioblastoma cells, in which the endogenous mutant *STAG2* allele had been corrected by homologous recombination, began re-expressing the protein, their collections of chromosomes began returning to normal.

STAG2 is located on the X chromosome—apparently the second cancer-related gene that has been found there. With just one functional copy for each gene on this chromosome, "it only requires a single hit to get complete inactivation, which may help to explain the mutation's prevalence in the solid tumors we examined," notes lead author David Solomon, PhD. The Georgetown scientists are broadening their studies to analyze *STAG2* in breast, colon, lung, and other cancers. ■

New Rules Proposed for Human Studies

Aiming to boost the safety and privacy of patients in clinical trials, cut red tape, and address the evolving nature of medical research, the federal government proposed several modifications to current regulations in July. Known as the Common Rule, the regulations have been in place since 1991.

Back then, studies were usually conducted at one institution. Today, most studies that enroll humans involve multiple centers, each with its own institutional review board (IRB) to