PEOPLE


The Jackson Laboratory

Edison Liu, MD, who has served as the founding executive director of the Genome Institute of Singapore (GIS) since 2001, will become president and chief executive officer of The Jackson Laboratory in January.

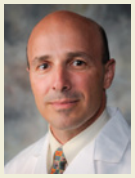
In addition to growing GIS into a major research institute, Liu managed the Singapore Cancer Syndicate, a funding agency to enhance translational oncologic research, and headed the national tissue bank. He also served as chairman of the board for the Health Sciences Authority, the nation's regulatory agency for health and blood banking. Prior to his Singapore stint, he directed the Division of Clinical Sciences at the National Cancer Institute.

Liu primarily studies the functional genomics of breast cancer.


St. Jude Biomedical Communications

St. Jude Children's Research Hospital has named **Richard Gilbertson, MD, PhD,** director of its Comprehensive Cancer Center. He will also serve as an executive vice president in the organization.

Recruited to St. Jude from England in 2000, Gilbertson has led international research efforts to better understand the biology of medulloblastomas and ependymomas and has helped develop and run clinical trials to test therapies for patients with these cancers. He plans to continue his research in his new role.


Juan Pulido, Children's Medical Center

Stephen X. Skapek, MD, has taken over as director of the Center for Cancer and Blood Disorders at Children's Medical Center Dallas. He also became the director of UT Southwestern Medical Center's Division of Pediatric Hematology–Oncology.

Before moving to Texas, Skapek spent 4 years at the University of Chicago Children's Hospital, where he was the director of pediatric oncology. An expert on soft tumors, he has studied the molecular and genetic mechanisms that lead to cellular proliferation in childhood cancers.

George R. Buchanan, MD, who led the cancer center for more than 30 years, will continue to treat patients there.

Asking and Answering Provocative Questions

How does obesity contribute to cancer risk? Why are some disseminated cancers cured by chemotherapy alone? Why do second, independent cancers occur at higher rates in patients who have survived a primary cancer than in a cancer-naïve population?

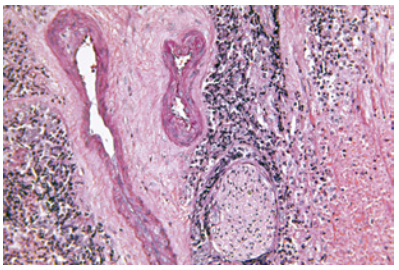
This is just a sampling of 24 vexing questions related to cancer risk, prevention, development, detection, diagnosis, and treatment that the National Cancer Institute (NCI) would like to answer through its Provocative Questions program. The brainchild of the organization's director, Harold E. Varmus, MD, the program will dole out about $15 million in fiscal year 2012 to support innovative research to examine inadequately studied observations and to better understand perplexing or paradoxical findings. The questions were compiled based on website submissions and discussions at several NCI workshops over the past year.

The workshops, which included experts in basic science, population science, and treatment and prevention, "gave everyone a chance to take a step back, survey the cancer landscape, and see what questions remained puzzling to the field," says Jerry S.H. Lee, PhD, deputy director of NCI's Center for Strategic Scientific Initiatives. "They raised questions for which we don't have complete answers and questions that we have neglected or haven't had the technology to study."

Researchers can apply for either a standard R01 grant or an exploratory R21 grant. Proposals, which are due November 14, must address one of the 24 questions to be considered. For more information, log on to http://provocativequestions.nci.nih.gov. ■

A Tumor Suppressor for Prostate Cancer

Prostate cancer usually grows so slowly that only about 1% of patients diagnosed with early-stage disease die from it over the next 15 years. But because physicians have no foolproof method to separate aggressive cancers from indolent ones, most patients opt for treatment, risking complications such as impotence and incontinence.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Pathologists can diagnose prostate cancer, shown here, with a microscope, but they can't precisely determine how it will behave.

The discovery of a new prostate cancer tumor suppressor gene, *PHLPP1,* and studies examining its relationship to the tumor suppressor gene *PTEN* may simplify decision-making for newly diagnosed patients—and may point to the most effective drugs for recurrent cancers.

Studying mice, researchers at Cold Spring Harbor and other laboratories found that Phlpp1 keeps the oncoprotein Akt in check, much like *Pten,* which is mutated in roughly half of prostate cancer patients (Cancer Cell 2011;20:173–86). But in *Pten*-deficient animals lacking both copies of *Phlpp1,* Akt activity kicked into overdrive. Although the master tumor suppressor p53 delayed progression of the disease, tumors eventually overcame that too.

The researchers then examined more than 200 primary and metastatic tumor samples from men. Almost none of the primary cancers were missing both *PTEN* and *PHLPP1,* but both genes were frequently deleted in metastatic tumors, along with *p53.* By measuring expression levels of *PTEN* and *PHLPP1* in biopsy or prostatectomy samples, doctors could determine the likelihood of an aggressive cancer and the chances of disease recurrence.

"Low RNA transcription activity for these genes seems to herald a future problem," says Lloyd C. Trotman, PhD, the study's leader. "About 70% of these patients will have a recurrence within 10 years."

Checking for mutations of *PTEN, PHLPP1,* and the closely related *PHLPP2* in circulating tumor cells in relapsed patients could also help determine which ones would be candidates for clinical trials of drugs that inhibit the PI3-kinase/AKT pathway, when these patients should start taking the drugs (based on *p53* status), and which drugs might be optimal for them. ■

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Cancer Discovery 2011;1:368.