$7.8 million) each over 2 years on top of existing annual funding of between £3 million and £4 million (between about $4.7 million and $6.2 million) each. The centers will act as hubs of research in areas of strategic importance to CRUK, and will facilitate collaboration among investigators across CRUK's 15 centers.

"We are substantially increasing funding to develop more comprehensive centers with the expertise, infrastructure, and technology required to deliver translational research at the highest international level," says David Scott, PhD, CRUK's director of Discovery Research and Centers. "We hope to increase the level of investment in these locations over the next few years as research activity ramps up."

At Cambridge, the new funding will support development of early-detection techniques for different cancers, says Scott. Investigators are also building a brain cancer program under the direction of Richard Gilbertson, MD, PhD, who most recently served as director of the Comprehensive Cancer Center at St. Jude Children's Research Hospital in Memphis, TN.

In Manchester, researchers will establish a national center to discover biomarkers for early detection, clinical decision-making, and monitoring treatment, says Scott. The center already has expertise in circulating tumor cells (CTC), and investigators there published findings last year showing that CTCs could be used to monitor small-cell lung cancer and predict response to treatment (Nat Med 2014;20:897–903). In addition, the Manchester center has partnered with the CRUK center at University College London to establish a Center of Excellence in lung cancer.

Researchers at Oxford's Clinical Imaging and Radiation Oncology Hub are exploiting insights from basic biology to improve radiotherapy. For example, they recently published findings suggesting that giving AKT inhibitors in combination with radiotherapy might improve the response to radiotherapy in p53-deficient tumors (J Clin Invest 2015;125:2385–98).

"We think of this new funding as a way to bridge the gap between the lab and the clinic," says Scott. "It's providing the infrastructure, such as data managers, biobanking, and platforms, to profile tumors at the molecular level—the things that enable translational research."

Such discovery is critical given that the 10-year survival rate in the UK for all cancers is about 50%. "We'd like to see that increase to at least 75% over the next 20 years," Scott says. ■

New Compound Targets Warburg Effect

Researchers have developed a drug that kills cancer cells by inhibiting lipid production and the Warburg effect, the tendency for tumors to rely heavily on glycolysis even when a more efficient way of metabolizing glucose is available. The drug induces cell death in multiple cancer types and does not cause the side effects that have derailed previous attempts to target these processes.

"It's hitting two of the major metabolic pathways that cancer cells like to use," says senior author Tom Burris, PhD, chair of the pharmacology and physiology department at the St. Louis University School of Medicine in Missouri.

Researchers do not fully understand how the Warburg effect helps cancer cells, but it may aid cellular proliferation—and thus give tumors a growth advantage—by increasing production of metabolites that can be converted to lipids, amino acids, and nucleotides. Scientists have tried to design drugs to target specific enzymes in glycolysis or lipogenesis. However, most glycolysis inhibitors have been ineffective or killed normal cells, and lipogenesis inhibitors have caused side effects, such as anorexia and weight loss.

The agent developed by Burris's team, SR9243, targets the nuclear receptors LXRα and LXRβ. These receptors activate expression of glycolysis and lipogenesis enzymes. The drug prompts the receptors to bind co-repressor proteins that reduce expression of those enzymes instead.

The drug killed colorectal, lung, and prostate cancer cells in cell culture but did not affect healthy cells, the team reports (Cancer Cell 2015;28:42–56). Expression of glycolysis and lipogenesis genes dropped, as did levels of glycolysis metabolites and lipids. In mice with xenografts of these cancers, the drug slowed tumor growth and did not cause weight loss, liver toxicity, or inflammation of normal tissue.

That the drug worked in different types of cancer cells is encouraging, says Ralph DeBerardinis, MD, PhD, chief of the division of pediatric genetics and metabolism at The University of Texas Southwestern Medical Center in Dallas, who wasn't involved in the study. "It means that if the therapy turned out to be effective, it wouldn't be tied to a small subset of human cancers. It could potentially be fairly general."

Burris's team found some limitations, however. SR9243 was less potent in pancreatic and ovarian cancers and had no effect on breast tumor cell lines, says Burris. He and his colleagues are investigating why these cancers don't respond well to the treatment.

DeBerardinis also cautions that scientists still know little about which metabolic pathways are active in a human tumor. "The metabolism of an actual tumor in a person is a black box," he says. He would like to see the drug tested in mice with tumors that arise spontaneously rather than from an injected cell line, because their metabolism may be more similar to that of a human patient's tumor than tumors established from xenografts. ■

Device Captures CTC Clusters in Blood

Individual blood-borne tumor cells have been linked to metastasis in patients with cancer, and clusters of these circulating tumor cells (CTC) spell an even worse prognosis. To facilitate the study of these aggregates, researchers have created Cluster-Chip, a microfluidic device that captures CTC clusters directly from the blood without using antibodies or chemical labels.

Isolating CTCs is tricky because they are uncommon—about one per billion cells in blood—and CTC clusters are rarer still. Many methods use antibodies, which bind CTCs in order to capture them directly or bind other blood cells in order to separate them from CTCs. However, because antibody strength and specificity often vary and CTCs can be heterogeneous in their expression of surface markers, antibody-based techniques are not ideal.

Cluster-Chip, on the other hand, is "antigen-agnostic," says A. Fatih