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**Charles S. Abrams, MD,** a professor of medicine, pathology, and laboratory medicine; vice chair for research and chief scientific officer of the Department of Medicine at the University of Pennsylvania; and director of the Blood Center for Patient Care and Discovery at the University of Pennsylvania and Children's Hospital of Philadelphia, will become president of the American Society of Hematology on December 8. Serving a 1-year term, he will succeed David A. Williams, MD.

A graduate of Johns Hopkins University in Baltimore, MD, and Yale University School of Medicine in New Haven, CT, Abrams's research interests include platelet activation, platelet disorders, and production of megakaryocytes. In particular, his lab focuses on phospholipid signaling in platelets and its contribution to inappropriate platelet activation.

MRC Laboratory of Molecular Biology

Nobel laureate **Venkatraman "Venki" Ramakrishnan, PhD,** became president of the Royal Society, the United Kingdom's preeminent scientific institution, on December 1. He replaces geneticist Paul Nurse, PhD, who held the position for the past 5 years. Nurse will continue his role as director and chief executive of the London, UK–based Francis Crick Institute, a biomedical research center.

Most recently, Ramakrishnan served as deputy director of the Medical Research Council Laboratory for Molecular Biology in Cambridge, UK. He was awarded a share of the Nobel Prize in Chemistry in 2009 for his work on the structure and function of ribosomes.

The Royal Society was founded in 1660. As the organization's president, Ramakrishnan will wield influence over and be able to speak out on science policy, as well as the government's research budget, which has flagged significantly in purchasing power since 2010.

## First Immunotherapy Combo Approved for Cancer

The FDA has granted accelerated approval to the combination of Bristol-Myers Squibb's PD-1 inhibitor nivolumab (Opdivo) and CTLA-4 inhibitor ipilimumab (Yervoy) to treat advanced melanoma, the first approval of an immunotherapy combination to treat cancer.

The agency approved the combination for patients with BRAF V600 wild-type unresectable or metastatic melanoma, based on the pivotal phase II CheckMate 069 trial (N Engl J Med 2015;372:2006–17). In that study, patients with BRAF wild-type melanoma had objective response rates (ORR) of 61% with the combination therapy versus 11% with ipilimumab alone.

Later results from the phase III CheckMate 067 study, which will be taken into account for final approval, suggest that the combination may be an option for all melanoma patients with advanced disease (N Engl J Med 2015;373:23–34). Researchers enrolled 945 patients with untreated advanced melanoma regardless of mutation status and found that the nivolumab–ipilimumab combination extended progression-free survival (PFS) and improved ORR compared with either drug alone.

"This is a very exciting development for melanoma patients," says Patrick Ott, MD, PhD, clinical director of the Melanoma Center and the Center for Immuno-Oncology at Dana-Farber Cancer Institute in Boston, MA. "Although the response rate of 60% with the combination compared to 40% with nivolumab alone seen in both the phase II and phase III trials comes at a price for these patients because the toxicity is substantially higher with the combination compared to anti–PD-1 therapy alone."

In the phase III study, about 36% of patients in the combination therapy group dropped out due to side effects, compared with about 8% in the nivolumab group and 15% in the ipilimumab group. The most common adverse events were diarrhea and colitis.

When looking at response based on PD-L1 status, researchers in the phase III study noted that the greatest benefit with the combination of nivolumab and ipilimumab versus nivolumab alone may occur in the context of negative PD-L1 tumor expression, as the PFS was similar between combination therapy and monotherapy in patients with PD-L1–positive tumors. Expression of PD-L1 has been associated with increased response rates in previous studies using PD-1 inhibition alone.

"At Dana-Farber, we are offering the combination to most patients who we think can tolerate the toxicity, regardless of PD-L1 status," says Ott, who has been treating patients for the past 8 months on an extended-access protocol. "Patients face a choice between taking a PD-1 inhibitor alone with lower toxicity or the combination with higher toxicity, but a better overall chance of response." ■

## HPV Vaccine Triggers Regression of Precancers

A DNA vaccine that targets strains of the human papillomavirus (HPV) causes cervical lesions to regress or disappear, a phase IIb trial shows.

In most women who are infected with HPV, the immune system attacks and eliminates the virus. The two approved HPV vaccines, Gardasil and Cervarix, help protect against new infections with HPV-16 and HPV-18, the strains responsible for 70% of cervical cancers, but they don't clear the virus from already-infected patients. HPV infection can trigger a precancerous lesion known as cervical intraepithelial neoplasia (CIN).

Doctors can't predict which lesions will progress to cervical cancer, so the standard treatment is to have them excised. However, more than one procedure is often necessary, and deep excisions can increase the risk of premature birth. Researchers are working on several less invasive alternatives, including therapeutic vaccines, to induce immune responses that would eliminate HPV.

Cornelia Trimble, MD, of the Johns Hopkins School of Medicine in Baltimore, MD, and colleagues tested an investigational therapeutic DNA vaccine, VGX-3100, developed by Inovio Pharmaceuticals (Plymouth Meeting, PA). VGX-3100 contains plasmids that target two key HPV proteins, E6 and

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*Cancer Discov* 2015;5:1228.