AACR's 2012 Princess Takamatsu Memorial Lectureship Goes to Mary J. C. Hendrix, Ph.D.

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CHICAGO — The AACR will honor Mary J. C. Hendrix, Ph.D., president and scientific director of the Children's Memorial Research Center at Northwestern University's Feinberg School of Medicine in Chicago, with the sixth annual Princess Takamatsu Memorial Lectureship at the AACR Annual Meeting 2012, held here March 31 – April 4.

The AACR Princess Takamatsu Memorial Lecture is presented to a scientist whose novel and significant work had or may have a far-reaching impact on the detection, diagnosis, treatment or prevention of cancer, and who embodies the dedication of the princess to multinational collaborations. Her Imperial Highness Princess Kikuko Takamatsu was instrumental in promoting cancer research and encouraging cancer scientists. She became a champion for these causes following her mother's death from bowel cancer in 1933 at the young age of 43.

"I am honored and humbled to be the recipient of the Princess Takamatsu Memorial Lectureship, especially having had the extraordinary privilege of meeting Princess Takamatsu and admiring her tenacious efforts toward the eradication of cancer," Hendrix said. "Many of our scientific accomplishments were greatly enhanced through international collaborations, which the Princess Takamatsu Lectureship actively promotes."

Hendrix's lecture, "Targeting the Plasticity of Metastatic Tumor Cells," will take place at 4:30 p.m. CT on Monday, April 2 in room S100 at McCormick Place West.

Hendrix, a cell biologist, has led groundbreaking work that has helped change the way scientists understand how cancer grows and spreads, findings that hold promise for the development of new therapeutic strategies.

New blood vessels are capable of being formed from existing vessels through a process called angiogenesis. Tumors exploit this biological process as a means to foster their development and growth and to transport spreading (metastatic) cancer cells to secondary tumor locations throughout the body via the bloodstream.

This concept of tumor growth and spreading was further expanded in 1999, when Hendrix and her colleagues introduced the concept of "vasculogenic mimicry" (VM) in their study published in the *American Journal of Pathology* and highlighted in *Science*. Hendrix's team observed that highly aggressive uveal melanomas, which develop in the eye, exhibit the ability to form vascular networks in the primary tumor, networks which had been generated by the cancer cells themselves. This discovery supported the idea that tumors possess the ability to fuel themselves by

generating complex, vascular networks without the need of endothelial cells or new blood vessel formation. VM has now been reported in over 20 different cancer types, associated with aggressive phenotype.

These results, combined with subsequent studies, may help explain why some cancers often do not respond to conventional chemotherapies or why tumor growth and spread can continue despite treatment with angiogenesis inhibitors in rodent models and in humans. This hypothesis has been supported by Hendrix in her 2004 *Journal of the National Cancer Institute* article, which showed that the drug endostatin, along with other angiogenesis inhibitors, blocked endothelial cell-mediated formation of new blood vessels, but failed to halt the formation of vascular networks produced by treated uveal and cutaneous melanoma cells.

Subsequent studies conducted by Hendrix and her colleagues have focused on understanding the molecular driving forces behind vasculogenic mimicry. More specifically, how cell-to-cell communication, cell identity and fate, and cellular interactions within the tumor microenvironment all contribute to specific gene expression changes that lead to tumorigenesis, tumor-mediated vasculogenesis and metastasis. These studies, in addition to contributing to the understanding of cancer evolution and survival, have further highlighted the “plastic” nature of tumor cells (similar to an embryonic phenotype) and their potential to be reprogrammed, which may ultimately lead to new therapeutic opportunities against cancer.

Hendrix is a past president of the Federation of American Societies for Experimental Biology and is a recipient of a prestigious MERIT Award from the National Cancer Institute. An author of more than 200 scientific publications, Hendrix has received numerous awards and honors.

Press registration for the AACR Annual Meeting 2012 is free to qualified journalists and public information officers: www.aacr.org/PressRegistration [2].

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About the AACR

Founded in 1907, the American Association for Cancer Research (AACR) is the world’s first and largest professional organization dedicated to advancing cancer research and its mission to prevent and cure cancer. AACR’s membership includes 34,000 laboratory, translational and clinical researchers; population scientists; other health care professionals; and cancer advocates residing in more than 90 countries. The AACR marshals the full spectrum of expertise of the cancer community to accelerate progress in the prevention, biology, diagnosis and treatment of cancer by annually convening more than 20 conferences and educational workshops, the largest of which is the AACR Annual Meeting with more than 18,000 attendees. In addition, the AACR publishes seven peer-reviewed scientific journals and a magazine for cancer survivors, patients and their caregivers. The AACR funds meritorious research directly as well as in cooperation with numerous cancer organizations. As the Scientific Partner of Stand Up To Cancer, the AACR provides

expert peer review, grants administration and scientific oversight of individual and team science grants in cancer research that have the potential for patient benefit. The AACR actively communicates with legislators and policymakers about the value of cancer research and related biomedical science in saving lives from cancer.

For more information about the AACR, visit www.AACR.org [6].

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