UH Seidman Cancer Center researchers present at American Society of Hematology Annual Meeting

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CLEVELAND: Researchers from Seidman Cancer Center at University Hospitals (UH) Case Medical Center and Case Western Reserve University School of Medicine presented new research findings in 25 presentations this weekend at the 53rd Annual Meeting of the American Society of Hematology (ASH) at the San Diego Convention Center.

"The breadth and depth of this innovative cancer research presented at ASH is truly outstanding," says Stan Gerson, MD, Director of the Seidman Cancer Center at UH Case Medical Center and the Case Comprehensive Cancer Center at Case Western Reserve University. "Our faculty members are making tremendous advances in hematology and oncology which is reflected in their being chosen for oral and poster presentations."

Speaking at the ASH "Scientific Symposium on Lymphoid Neoplasia" in a session titled "Autophagy and Metabolism in Lymphoid Malignancies," Clark Distelhorst, MD, provides a synthesis of the latest research indicating that autophagy occurs in lymphoid malignancies and may be a novel therapeutic target for lymphoma and other lymphoid neoplasia. His research suggests that targeting autophagy (a process through which cells eat parts of themselves to generate sufficient energy to stay alive) may be a useful adjunct to the longstanding use of glucocorticoids, such as prednisone, to kill cancer cells.

His session outlines the growing body of evidence that treatments aimed at inducing autophagy have great promise in treating lymphoid malignancies. In his session, Dr. Distelhorst presents important data explaining how glucocorticoids starve tumor cells of glucose and thus induce autophagy. Researchers at UH Case Medical Center and Case Western Reserve University identified the Dexamethasone-induced Gene 2 (dig2) that encodes a protein mediator of autophagy.

"This new cancer-fighting strategy lays the groundwork for further development of autophagy inhibitors to enhance the glucocorticoids properties," says Dr. Distelhorst, who is vice-chair of the ASH subcommittee on Lymphoid Neoplasia. "This is a major step forward in our research efforts to develop new therapies for lymphoid malignancies."

Dr. Distelhorst's session is Saturday, December 10, 4 p.m. - 5:30 p.m. in Room 6A. (San Diego Convention Center).
http://ash.confex.com/ash/2011/webprogram/Paper35836.html [1]

In a poster presentation (Abstract# 1907), Jeffery Auletta, MD, Kenneth Cooke, MD,

and colleagues presented significant findings that mesenchymal stem cells (MSCs) effectively treat graft-versus-host disease (GvHD) while not interfering with bone marrow transplant's efficacy in treating leukemia.

MSCs are non-hematopoietic (not blood-forming cells) adult stem cells found in the bone marrow and were discovered at UH Seidman Cancer Center and Case Western Reserve University. They maintain hematopoietic stem cell (blood-forming cells) development and also differentiate into fat cells, bone cells and cartilage cells. MSCs have been shown to suppress immune responses ex vivo (outside the body in cell culture conditions).

Due to these properties, MSCs have been used to treat GvHD in bone marrow transplant (BMT) patients. However, how MSC immunomodulation works in vivo (inside the body) has not been well studied, and, in fact, could potentially promote leukemia/lymphoma recurrence in transplant patients. That is, the benefit of BMT is that the donor graft kills residual leukemia in the transplant recipient (host), a process called graft-versus-leukemia (GvL).

"We used a pre-clinical mouse model of BMT to study how human MSCs mediate in vivo immune effects," says Dr. Auletta. "Our results show for the first time using an animal model that human MSCs simultaneously attenuate GvHD, but spare GvL activity."

The poster titled "Human Mesenchymal Stem Cells Attenuate Graft-Versus-Host Disease and Maintain Graft-Versus-Leukemia in Murine Allogeneic Bone Marrow Transplantation" is Saturday, December 10, 5:30 p.m.-7:30 p.m. in Hall GH (San Diego Convention Center).
http://ash.confex.com/ash/2011/webprogram/Paper39108.html [2]

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