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New findings may help overcome hurdle to successful bone marrow transplantation

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Blood diseases such as leukemia, multiple myeloma, and myelodysplasia can develop from abnormal bone marrow cells and a dysfunctional bone marrow microenvironment that surrounds these cells. Until now, researchers have been unable to replace the cells that make up the bone marrow microenvironment. Researchers reporting in the May 28 issue of the Cell Press journal *Developmental Cell* have found that eliminating a gene in the cells found in this microenvironment causes them to die, therefore enabling donor cells to replace them. In addition to providing a better understanding of the bone marrow microenvironment, the findings could help improve bone marrow transplant therapy for patients who need it.

Scientists led by Dr. Hongbo Luo of Harvard Medical School and Boston Children's Hospital found that mice lacking a gene that codes for an enzyme called SH2-containing inositol-5'-phosphatase-1 (SHIP) could not form a normal bone marrow microenvironment but that transplanted cells from normal mice could reconstitute the microenvironment in the mutant animals.

This microenvironment reconstitution normalized blood cell production in the bone marrow and also corrected defects due to abnormal blood cell production in the spleens and lungs of the SHIP-deficient mice.

"We believe our finding is a major breakthrough in the field," says Dr. Luo. "Long-term reconstitution of the bone marrow environment can be achieved by treatment with a specific SHIP inhibitor, although it is

currently not available yet."

More information: *Developmental Cell*, Liang et al.: "Deficiency of Lipid Phosphatase SHIP Enables Long-Term Reconstitution of Hematopoietic Inductive Bone Marrow Microenvironment." dx.doi.org/10.1016/j.devcel.2013.04.016

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