Augmenting Stress-Induced Erythropoiesis

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Technology description

Description:
Anemia, defined as a diminished number of red blood cells (RBCs) or less than normal hemoglobin levels, is the most common hematological disorder. Major causes of anemia include blood loss, excessive blood cell destruction, or deficient RBC production. Depending on the severity and duration of the anemia, symptoms can range from tiredness and shortness of breath to lasting organ damage and death. Erythropoiesis, the process of making new RBCs, or erythrocytes, occurs continuously in vivo to offset the turnover of erythrocytes in circulation and is stimulated by erythropoietin (EPO), a hormone produced in the kidney. In response to hypoxic stress, EPO production and release is upregulated to increase RBC production in the bone marrow and "extramedullary hematopoiesis sites," most notably the spleen.
Researchers at the University of Virginia have identified a mechanism to stimulate an individual's own stress-induced erythropoiesis response to increase RBC production. The process involves activating CD24, a glycoprotein expressed at the surface of a variety of cells, including dendritic cells (DCs). Engagement of CD24 on a distinct subset of splenic DCs using a unique monoclonal antibody (αCD24) leads to stimulation of extramedullary hematopoiesis. Injection of αCD24 in mice led to splenomegaly and dramatic increases in endogenous EPO production, proliferation of RBC precursor cells, and the number of circulating reticulocytes (immature RBCs). This novel discovery has significant therapeutic potential to complement existing recombinant EPO therapy for treatment of anemia, or possibly as a stand-alone treatment. Safety and efficacy of repeated injections of αCD24 to generate several waves of erythropoiesis in mice have been established.

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