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Opinion Article

Significance of Fatty Acid Metabolism in Terminal Red Blood Cell Formation

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DESCRIPTION

Metabolism is essential for terminal erythropoiesis. The PHOSPHO1 gene product, a phosphocholine phosphatase, has been proposed to play an important role. PHOSPHO1 knockouts (KOs) showed decreased erythroblast proliferation and enucleation in both mouse and human erythroid tissues, presumably due to energy depletion mediated by oxidative stress inhibition. This study emphasizes the importance of altered expression of lipid metabolism genes during maturation of red blood cells [1].

Despite being highly specialized for gas transport, it is much more than an inert hemoglobin receptacle, with a plethora of surprisingly sophisticated properties. Among these, oxygen tension has recently been shown to control glucose metabolism, cytoskeletal integrity, and membrane permeability. During maturation, the developing red cell must proliferate as well as undergo significant changes in order to acquire the necessary properties to survive in the circulatory system, where it lacks the ability to synthesize proteins *de novo* and faces significant challenges such as repeated segment of shear and oxidative stress.

During later erythrogenesis, many significant changes occur, including the loss of the nucleus. During erythropoiesis is still limited. There is a lot of information out there about globin gene switching, which is important for a number of common hemoglobinopathies. Other nonglobin protein changes have also been thoroughly researched. These include the accumulation of cytoskeletal elements as a result of spectrin condensation, increased expression of band, and the acquisition of other membrane transporters. Although mutations in these proteins are uncommon, they are occasionally linked to hemolytic anemia and irregularities in red cell shape or volume (such as stomatocytes and spherocytosis). Understanding of red cell physiology is improving as we learn more about their molecular causes [2,3].

Diseases involving abnormal lipid metabolism may be less well understood. Protein transporter interactions can be secondary. The loss of aminophospholipid asymmetry in a variety of hemoglobinopathies, such as sickle cell disease, is an obvious example. Although primary disturbances involving specific gene mutations directly involved in lipid metabolism have been described, they are much more uncommon. Neuroacanthocytosis and phytosterolemia are two examples of disorders that cause erythropoiesis abnormalities as well as acanthocytosis and stomatocytic hemolysis.

Their study identifies a new lipid pathway that is essential for normal red cell development. Individually analyzed subpopulations of developing mouse erythroblasts revealed significant variation in lipid content and metabolism.

During terminal differentiation, phosphocholine and its precursor phosphatidylcholine were downregulated, while sphingomyelin and choline were upregulated, and catabolic end products of phosphatidylcholine metabolism were enriched. PHOSPHO1 expression was reduced in mouse foetal erythroid progenitors and deficiencies in terminal erythroblast differentiation were discovered by analyzing genes upregulated during terminal differentiation.

Cell proliferation and enucleation decreased, foetal livers had fewer mature red cells, late embryos were smaller and paler and reticulocytosis increased [4]. When early red cells from KO mice foetal livers were induced to differentiate into erythrocytes *in vitro*, genes that were normally upregulated showed decreased expression levels. The total number of enucleation events was lower.

Red cell count and volume were mostly normal in postpartum mice. However, reticulocytosis indicated that this was most likely the result of a compensatory stress erythropoiesis. PHOSPHO1 knockout mice were also less able to respond to phenylhydrazine-induced stress erythropoiesis. The final series investigated the behaviour of human CD 34^+ stem/progenitor cells induced to proliferate and differentiate into enucleated erythrocytes.

The decrease in phosphocholine and increase in choline during terminal differentiation mirrored the situation in mouse cells [5]. PHOSPHO1 gene expression was increased concurrently, whereas PHOSPHO1 knockdown resulted in a significant decrease in cell proliferation and enucleation. Finally, glycine or serine supplementation reduced proliferation rate. These findings

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point to a conserved function of PHOSPHO1 in terminal erythropoiesis from mice to humans.

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