Special Issue

Sickle Cell Disease: Pathogenesis and Therapies

Message from the Guest Editors

Since its emergence in prehistoric times in countries where malaria is endemic, sickle cell disease (SCD) has spread throughout the world, becoming the most common genetic disease globally.

SCD is caused by the homozygosity for the beta globin gene mutation (a substitution of valine for glutamic acid at position 6: □6 Glu→Val) or by compound heterozygosity in which one HbS allele is inherited together with another □-globin variant (e.g., HbSC, HbS/□^{0}-thalassemia, HbS/□□-thalassemia,etc). Under physiological stress conditions such as hypoxia, infection, acidosis, or dehydration, HbS polymerizes within the red blood cells, resulting in erythrocytes deformation, a process known as sickling.

SCD merely as red blood cell disorder□ its pathophysiology also involves endothelial dysfunction, platelet activation, leukocyte adhesion, a procoagulant state, and chronic inflammation.

Current research aims to better define these interconnected pathways, which both shape clinical outcomes and represent promising targets for innovative therapies.

Allogeneic hematopoietic stem cell transplantation (HSCT) from HLA-matched sibling donors remains the only established curative option.

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Cells has become a solid international scientific journal that is now indexed on SCIE and in other databases. We have successfully introduced a special issues format so that these issues serve as mini-forums in specific areas of cell science. *Cells* encourages researchers to suggest new special issues, serve as special issues editors, and volunteer to be reviewers. Our main focus will remain on cell anatomy and physiology, the structure and function of organelles, cell adhesion and motility, and the regulation of intracellular signaling, growth, differentiation, and aging. We are open to both original research papers and reviews.

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