Iron Transporter Protein Slc22a17 Regulates Myelin Formation and Oligodendrocyte Development

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Solute carriers (SLCs) are key to cellular transport of growth substances and their dysfunction is often associated with disease. Studies have focused on the solute carrier protein Slc22a17, which is involved in iron transport. Previous studies shown that Slc22a17 is highly expressed in oligodendrocytes of the central nervous system (CNS), but its specific physiological function in oligodendrocytes and its involvement in the mechanism of myelin sheath formation are not well understood. The study determined the expression pattern of Slc22a17 gene in oligodendrocytes using Gene Set Enrichment Analysis (GSEA) and single-cell sequencing. Confocal microscopy, electron microscopy and live cell imaging techniques helped to reveal the function of Slc22a17 in oligodendrocyte proliferation, differentiation and migration. Iron metabolism in oligodendrocytes was assessed using Prussian blue staining, ICP-MS and fluorescent probes. Finally, employing a primary cell iron supplementation model, the role of promoting cell differentiation was revealed. The results highlight the critical role of the gene Slc22a17 in the early stages of oligodendrocyte differentiation, whose deletion leads to oligodendrocyte differentiation arrest and iron deficiency. Interestingly, Slc22a17 deletion did not affect oligodendrocyte migration and FAC iron treatment promoted oligodendrocyte differentiation. The study unveiled that Slc22a17 deficiency is one of the causes of oligodendrocyte differentiation failure and myelin deficiency, and that iron supplementation offers new perspectives for the treatment of demyelinating diseases.