

Assessing the Bone Health of Sick Cell Mice: A Histological Study of Cathepsin K Knockout

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This project is dedicated to enhancing the quality of life for individuals afflicted with sickle cell disease, a hereditary condition characterized by abnormal red blood cell morphology and function, culminating in a spectrum of health complications, notably chronic inflammation and skeletal impairments. Employing a distinctive murine model termed the "humanized sickle mouse" or transgenic mouse model, engineered to simulate the human gene associated with sickle cell disease, I endeavor to elucidate novel therapeutic avenues. In this experiment, I focus on a protein called Cathepsin K, which is known to be involved in bone breakdown and inflammation. By removing Cathepsin K from the sickle mouse model, I hope to reduce these harmful effects of sickle bone disease and improve bone health. To understand the impact of Cathepsin K removal, I have compared the bones of mice without Cathepsin K to those with Cathepsin K, including mice with different genetic backgrounds related to SCD. I study bone structure and composition using Masson's Trichrome, Hematoxylin & Eosin, and Safranin-O stains. Additionally, I observed Micro-CT, a special scanning technique, to examine bone microstructure. This study aims to provide insights into how Cathepsin K affects bone health in SCD and how removing it could potentially benefit individuals with this condition. This research may help develop new treatments to improve the quality of life for people living with SCD.