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Chondrocyte Identity and Function are Controlled by Glutamine Metabolism

Claire-Sophie Devignes*

Laboratory of Clinical and Experimental Endocrinology, Department of Chronic Diseases and Metabolism, Belgium

Abstract

Correct functioning of chondrocytes is crucial for bone growth and fracture repair. These cells square measure extremely associateabolic however survive and performance in an avascular setting, implying specifc metabolic necessities that square measure, however, poorly characterised. Here, we tend to show that chondrocyte identity and performance square measure closely coupled with amino acid metabolism during a feed forward method. The master chondrogenic transcription issue SOX9 stimulates amino acid metabolism by increasing amino acid consumption and levels of glutaminase one (GLS1), a rate-controlling catalyst during this pathway. Consecutively, GLS1 action is important for chondrocyte properties and performance via a triangular mechanism. First, amino acid controls chondrogenic organic phenomenon epigenetically through salt dehydrogenase-dependent acetyl-CoA synthesis, necessary for simple protein acylation. Second, transaminase-mediated aspartate synthesis supports chondrocyte proliferation and matrix synthesis. Third, glutamine-derived glutathione synthesis avoids harmful reactive element species accumulation and permits chondrocyte survival within the avascular growth plate. Together, our study identifes amino acid as a metabolic regulator of gristle ftness throughout bone development.

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Introduction

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***Corresponding author:** Claire-Sophie Devignes*, Laboratory of Clinical and Experimental Endocrinology, Department of Chronic Diseases and Metabolism, Belgium, E-mail: Sophiecd@hotmail.co.in

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