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LYSINE METHYLATION SIGNALING IN MUSCLE STEM CELL FATE

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Protein lysine methylation is an important post-translational modification that regulates protein stability, activity, and localization. It is controlled by lysine methyltransferases, which add methyl groups, and lysine demethylases, which remove them. Although its role in epigenetic regulation has been extensively studied, the contribution of lysine methylation to stem cell fate remains largely unexplored. The main objective of my research is to understand how lysine methylation signaling regulates stem cell fate during development, aging, and tissue repair in both sexes. My group focuses on muscle stem cells, which are essential for muscle growth and regeneration. Their function depends on the coordinated regulation of epigenetic mechanisms, extracellular signals, and cellular metabolism. By combining these approaches, we aim to uncover how lysine demethylases coordinate epigenetic regulation, signaling, and cellular metabolism to control muscle stem cell function.

One of our projects investigates the relationship between the Wnt/ β -catenin signaling pathway and the lysine demethylase LSD1. We found that LSD1 stabilizes β -catenin by demethylating it, thereby maintaining its nuclear levels and transcriptional activity. This mechanism contributes to the regulation of mitotic spindle orientation and reveal a novel link between extracellular signaling, protein methylation, and stem cell fate.

A second project explores the role of the lysine demethylase PHF2 in lipid droplet metabolism during muscle regeneration. We demonstrated that PHF2 promotes interactions between lipid droplets and mitochondria, supporting lipid utilization and efficient muscle regeneration. We further identified a molecular mechanism through which PHF2 regulates lipid droplet turnover by controlling gene expression and communication between cellular organelles.

Together, these projects highlight how lysine demethylases integrate epigenetic regulation, signaling, and cellular metabolism to control stem cell function.

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