Louisiana Tech University Louisiana Tech Digital Commons

ANS Research Symposium

ANS Research Symposium 2019

Apr 11th, 8:30 AM - 11:30 AM

The Role of Mediator Subunit MED12 in Adipogenesis

Joseph Straub Louisiana Tech University

Conner Hartupee
Louisiana Tech University

Jamie Newman

Louisiana Tech University

Follow this and additional works at: https://digitalcommons.latech.edu/ans-research-symposium

Recommended Citation

Straub, Joseph; Hartupee, Conner; and Newman, Jamie, "The Role of Mediator Subunit MED12 in Adipogenesis" (2019). ANS Research Symposium. 28.

https://digitalcommons.latech.edu/ans-research-symposium/2019/poster-presentations/28

This Event is brought to you for free and open access by the Conferences and Symposia at Louisiana Tech Digital Commons. It has been accepted for inclusion in ANS Research Symposium by an authorized administrator of Louisiana Tech Digital Commons. For more information, please contact digitalcommons@latech.edu.

The role of Mediator subunit MED12 in adipogenesis

Joseph Straub¹, Conner Hartupee¹, Dr. Jamie J. Newman²

Mediator, a large protein complex exclusive to eukaryotes, is a master regulator of cell-type specific gene expression. Mediator functions as an adapter that connects activators bound at enhancers to the transcription pre-initiation complex (PIC) located at the promoter. Our research focuses on how Mediator influences the differentiation of adipose-derived mesenchymal stem cells (ASCs). ASCs are harvested from adult donors and have the ability to self-renew and can differentiate down chondrogenic, osteogenic, and adipogenic lineages. We recently published findings indicating that knockdown of the Mediator subunit MED31 reduces adipogenesis in bone marrow-derived MSCs. We are now focused on MED12, a subunit of the Mediator complex kinase domain that appears to have a significant role in maintaining cell state. Our goal is to understand MED12's dual role as both a Mediator subunit in the regulatory kinase module and as a coactivator of transcription. We hypothesize that the loss of MED12 disrupts adipogenic differentiation in ASCs. Previously published research indicates MED12 coactivates β-catenin, a driver of adipogenesis, but little research currently links MED12 to the regulation of adipogenesis. We have performed siRNA-mediated knockdowns of MED12 in ASCs prior to inducing adipogenesis. ASCs display reduced adipogenesis as demonstrated by images of cell morphology and lipid vesicle staining. We have also optimized our sonication protocol for chromatin immunoprecipitation (ChIP) experiments and we are currently investigating MED12 genomic occupancy in order to determine the direct gene targets of MED12 during adipogenesis. Overall, this research is important for elucidating the requirements for proper regulation of differentiation of clinically-relevant ASCs, and the broader understanding of Mediator's function in ASCs will help foster their use in clinical applications such as regenerative medicine.

¹ School of Biological Sciences, Louisiana Tech University

² Molecular Sciences and Nanotechnology, Louisiana Tech University