Review of: "Plakoglobin regulates adipocyte differentiation independently of the Wnt/β-catenin signalling pathway"

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Potential competing interests: The author(s) declared that no potential competing interests exist.

Reviewer report by H KIM

This scientific report led by Abou Azar F et al investigated exploration of key regulator in the adipogenesis and characterization of regulator molecule, plakoglobin which they claim it as new features of adipogenesis (e.g., positive molecule affects to adipogenesis different signaling pathway such as Wnt/β-catenin independent compared to Beta catenin regulator in adipogenesis using mice 3T3-L1 preadipocyte cell and human adipocyte stem cells (hADSC) differentiation.

It is written well solid evidence-based justification the difference and potentiality what plakoglobin exert in cellular and molecular lever utilized two difference mice 3t3-L1 preadipocyte cells and hADSC cells following their working hypothesis and produced solid molecular interaction between key regulator such as PPARg2 and Plakoglobin. Points of research updated systematically and compromised to determine molecular cross talk between scaffold protein 14-3-3zeta which demonstrated engagement on adipogenesis. Following ongoing study, they conducted a new screening system like proteomic level difference between LFD and HFD to depict key regulator difference between Beta catenin and Plakoglobin. They provide details regarding underlying molecular mechanism using molecular interaction using gene level and protein expression level and transcriptional activity focused on Wnt/β-catenin signaling in part included lipid accumulation, as indicator of morphological difference, compared to differentiation condition. It is a valuable findings and insightful approach to illustrate the underlying cellular and molecular mechanism by which new aspects of molecules such as Plakoglobin in the process of developmental difference between human and mice model.

In addition, the idea extends to molecular cross talk between 14-3-3zeta and plakoglobin could be beneficial to understand better sequential processes followed by their strategy utilizing multi-omics platforms for example, transcriptomics and proteomics. I recommend further study considering the cellular difference and localization visualized by either histological approach or staining difference two regulatory molecules interact to PPARg2 in the adipogenesis utilizing FACS analysis. Overall, I agree that it makes a substantial contribution to inspire the research community in obesity and adipogenesis to unfold cellular and molecular mechanisms that go through new molecules like plakoglobin as a counterpart of 14-3-3zeta. Future study could validate the potential target motif in the regulatory and compensatory role of Plakoglobin in mice and human models as well.