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The role of Wnt/β-catenin pathway in adipogenic differentiation of mouse preadipocytes induced by S-adenosylmethionine

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S-adenosylmethionine (SAM) is a biological sulfonium compound known as the major biological methyl donor in reactions catalyzed by methyltransferases. In the present study, we found that SAM could promote adipogenesis, but the molecular mechanism of this promotion remains elusive. Three experiments were conducted to investigate the mechanism of SAM inducing adipogenic differentiation and lipid accumulation in mouse embryonic fibroblast-derived 3T3-L1 preadipocytes. Cells were treated with 0, 0.25, 0.50 mmol/L SAM, MDI differentiation medium and MDI differentiation medium containing 0.50 mmol/L SAM respectively. In Exp. 1, observation of cell morphology, Red Oil O staining and measurement of OD value were utilized to examine the effects of SAM on adipogenesis and lipid accumulation in 3T3-L1 cells both in vision and quantity. Cells treated with SAM and (or) MDI differentiation medium had more lipid droplets and higher OD value accompanied with the increasing concentration of SAM, especially with the MDI differentiation medium containing 0.50 mmol/L SAM treatment ($P<0.05$). The results demonstrated that SAM had the potential to promote adipogenic differentiation in adipose. Exp. 2 was set to verify the promotion of differentiation by SAM on transcription and translation levels. According to the result of Real-time PCR and Western blot, we could see that SAM markedly increased the mRNA ($P < 0.05$) and protein expression of ADD1, C/EBPα and PPARγ, transcription factors of adipogenic differentiation, Ap2, a marker of mature adipocytes. To explore the possible mechanisMS by which SAM promoted adipogenic differentiation, we determined the effect of SAM on the expression of genes and proteins related to Wnt signaling in Exp. 3. Upon stimulation of SAM induced 3T3-L1 cell differentiation, the expression of important factors and target genes in Wnt/β-catenin signaling pathway, such as Wnt 10b, Fz2, Lrp5, c-Myc, cyclin D1, were suppressed ($P<0.05$). This result was consistent with Wnt signaling suppression, which lead to differentiation to adipocytes. Lipid accumulation, macrography changes in adipogenic differentiation, transcription and translation levels associated with differentiation, and inhibition of canonical Wnt/β-catenin signaling were all positively correlated with the concentration of SAM. In conclusion, results indicate that SAM has the potential to promote adipogenic differentiation and lipid accumulation in adipose. The mechanism of this phenomenon is associated with Wnt/β-catenin signaling pathway.

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