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Hemp Seed Oil Inhibits the Adipogenicity of the Differentiation-Induced Human Mesenchymal Stem Cells through Suppressing the Cannabinoid Type 1 (CB1)

Abstract:

Central and peripheral mechanisms of the endocannabinoid system (ECS) favor energy intake and storage. The ECS, especially cannabidiol (CBD) receptors, controls adipocyte differentiation (hyperplasia) and lipid accumulation (hypertrophy) in adipose tissue. In white adipose tissue, cannabidiol receptor 1 (CB1) stimulation increases lipogenesis and inhibits lipolysis; in brown adipose tissue, it decreases mitochondrial thermogenesis and biogenesis. This study compared the availability of phytocannabinoids [CBD and Δ^9 -tetrahydrocannabinol (THC)] and polyunsaturated fatty acids [omega 3 (ω 3) and omega 6 (ω 6)] in different hemp seed oils (HSO). The study also examined the effect of HSO on adipocyte lipid accumulation by suppressing cannabinoid receptors in adipogenesis-stimulated human mesenchymal stem cells (hMSCs). Most importantly, Oil-Red-O' and Nile red tests showed that HSO induced adipogenic hMSC differentiation without differentiation agents. Additionally, HSO-treated cells showed increased peroxisome proliferator-activated receptor gamma (PPAR γ) mRNA expression compared to controls (hMSC). HSO reduced PPAR γ mRNA expression after differentiation media (DM) treatment. After treatment with HSO, DM-hMSCs had significantly lower CB1 mRNA and protein expressions than normal hMSCs. HSO treatment also decreased transient receptor potential vanilloid 1 (TRPV1), fatty acid amide hydrolase (FAAH), and monoacylglycerol lipase (MGL) mRNAs in hMSC and DM-hMSCs. HSO treatment significantly decreased CB1, CB2, TRPV1, and G-protein-coupled receptor 55 (GPCR55) protein levels in DM-hMSC compared to hMSC in western blot analysis. In this study, HSO initiated adipogenic differentiation in hMSC without DM, but it suppressed CB1 gene and protein expression, potentially decreasing adipocyte lipid accumulation and lipogenic enzymes.