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**Objectives:** Neuronal differentiation is involved in brain development. Stimulation of all-trans-retinoic acid (RA) in neuroblastoma cells results in growth inhibition, increased neuron-specific enolase (NSE) activity, and promoted axonal growth. Mitochondrial uncoupling protein-4 (UCP4) is specifically expressed in brain. UCP4 overexpression is neuroprotective against mitochondrial dysfunction by increasing ATP supply. We aimed to determine changes of UCP4 expression, and its role in modulating energy supply during neuronal differentiation.

**Methods:** We stably overexpressed human UCP4 in SH-SY5Y cells. Neuronal differentiation was induced by RA for 14 days in both UCP4 overexpressing and vector control cells. Neurite growth was visualised by immunostaining of neuronal tubulin (Tuj1), and changes in UCP4 expression were determined by Western blot. Number of neurite-bearing cells and neurite length were determined by confocal microscopy. The total ATP levels were measured by luciferase bioluminescent assay.

**Results:** Treatment of RA for 14 days induced neuronal differentiation in SH-SY5Y cells, as visualised by morphological changes and induction of neurite projection. The lengths of neurites in UCP4 overexpressing cells were significant longer compared with the controls. During the time course, UCP4 expression was gradually increased in parallel with Tuj1. The pattern of changes of ATP levels in both UCP4 overexpressing and vector cells were similar. However, the ATP level in UCP4 overexpressing cells was consistently higher than the controls.

**Conclusions:** This study revealed potential role of UCP4 in promoting neuronal differentiation, possibly via increasing energy supply. Higher ATP level in UCP4 overexpressing cells is likely rendering the cells better energy supply for the differentiation processes. Knowledge in UCP4 which modulate neuronal energy supply and differentiation has shed light on potential cell therapies against various neurodegenerative diseases.

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**Background:** Obesity is a major risk factor for non-alcoholic fatty liver disease (NAFLD). Our recent study showed that circulating level of adipocyte fatty acid binding protein (A-FABP) is elevated in patients with NAFLD and correlated with hepatic inflammation and fibrosis. We also demonstrated that both acute and chronic liver injuries are accompanied by elevated A-FABP expression in Kupffer cells. This study aimed to examine whether pharmacological inhibition of A-FABP acts as a promising strategy for the treatment of NAFLD using a diet-induced obese mouse model.

**Methods:** C57 male mice were fed with a high-fat high-cholesterol (HFHC) diet to induce chronic liver injury and administered with an A-FABP selective inhibitor, BMS309403 (BMS), for 16 weeks. Serum alanine aminotransferase (ALT), aspartate transaminase (AST), triglyceride and cholesterol levels, and hepatic fat content, were measured by biochemical methods. Quantitative-PCR was performed to determine the hepatic expression levels of A-FABP and several pro-inflammatory cytokines, as well as endoplasmic reticulum (ER) stress and fibrotic markers. H&E staining and oil red O staining were performed to determine the inflammatory status and necrosis in the liver.

**Results:** Oral administration of BMS alleviated glucose intolerance, insulin resistance and liver injury in HFHC-diet-induced obese mice. Treatment with BMS markedly reduced the hepatic expression of pro-inflammatory cytokines (TNF- $\alpha$ , MCP-1, IL-6), endogenous A-FABP, fibrotic (pro-collagen and TIMP-1) and ER stress (CHOP, GRP78 and XBP-1) markers. H&E staining and oil red O staining indicated the alleviation of inflammatory status and steatosis.

**Conclusion:** A-FABP may play an aetiological role in obesity-induced NAFLD by inducing ER stress and inflammation. The selective inhibitor of A-FABP may represent a promising drug candidate for the treatment of NAFLD.

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