中文題目：新綠原酸藉由抑制 NF-κB 表現達到保護粥狀動脈硬化的效用在高脂飲食所誘導的 ApoE 基因剃除小鼠

英文題目：Protective effects of neochlorogenic acid on atherosclerosis by inhibiting NF-κB expression in high-fat diet-fed ApoE$^{-/-}$ mice

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Background: Atherosclerosis is a chronic inflammatory disease, and it is a global clinical problem. The development of new and effective therapeutic targets for atherosclerosis is necessary. ApoE$^{-/-}$ mice were fed either a high fat diet (HFD) for 12 weeks to induce diabetic atherosclerosis, and the drug group was supplemented with 100 mg/kg neochlorogenic acid (nCGA). The effect of neochlorogenic acid (150 μM) treated for 24 h with high glucose (25 mM) and palmitic acid (200 μM) was evaluated.

Method: MTT and flow cytometry assays were performed to evaluate cell proliferation and cell death, respectively.

Results: Then, western blot was used to quantify the apoptosis-related proteins, including Bax, cleaved caspase-3, cleaved caspase-8, and further establish the NF-κB/TNF-α pathway for high glucose and palmitic acid -induced endothelial damage. The data of animal after 12 weeks of induction, neochlorogenic acid was observed to effectively slow down cardiovascular lipid accumulation, reduce the rate of weight gain, and improve serum levels caused by a high fat diet. The cell experiments simulating a diabetic environment by high glucose and palmitic acid have confirmed that neochlorogenic acid can down-regulate the changes of apoptosis-related proteins. The protein expression of p-IκB/NF-κB/TNF-α signaling pathway was also reduced after neochlorogenic acid treatment. **Conclusion:** Based on the above experimental results, neochlorogenic acid can control the NF-κB signaling, which can reduce the inflammation and apoptosis of endothelial cells caused by high glucose and palmitic acid, and then slow down the course of atherosclerosis.