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phisms may be related to dampness-phlegm pattern of Korean standard PI types in stroke patients.

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The Effect of Do-Hong-Sa-Mul-Tang on 3T3-L1 Adipocyte Differentiation



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Purpose: The aim of this study is to determine the antiadipogenesis effect of Do-Hong-Sa-Mul-Tang (DHSMT) in vitro

Methods: We adopted Oil red O staining that observed the formation of fat droplets to determine the anti-adipogenesis effects of DHSMT. And triglyceride (TG) production, leptin level and the protein expressions of peroxisome proliferator-activated receptor gamma (PPAR $\gamma$ ), CCAAT/enhancer binding proteins alpha (C/EBP $\alpha$ ) in 3T3-L1 adipocytes.

Results: We adopted Oil red O staining that observed the formation of fat droplets to determine the anti-adipogenesis effects of DHSMT. The TG level was suppressed about 3 fold as compare to differentiation group. Leptin was inhibited the production in supernatant about 2-10 fold as compare to differentiation group. We confirmed that protein expressions inhibited peroxisome proliferator-activated receptor gamma (PPAR- $\gamma$ ), CCAAT/enhancer binding proteins alpha (C/EBP- $\alpha$ ) as dose dependent significantly.

Conclusion: Our results showed that DHSMT suppressed lipogenesis effectively. Therefore we suggested that DHSMT will be treatment of disease related on lipid as like obesity, arteriosclerosis, hyperlipidemia and stroke.

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Persicarin isolated from the Oenanthe javanica attenuates diabetes-induced liver injury through the hyperglycemia-upregulated NADPH oxidase activation



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**Purpose:** This study was conducted to examine whether persicarin isolated from the Oenanthe javanica has an protective effect on hyperglycemia-induced alterations, such as oxidative stress and inflammation in the liver of streptozotocin-induced type 1 diabetic mice.

**Methods:** Persicarin (2.5 or 5 mg/kg body weight/day) was administered orally to diabetic groups of mice for 10 days, and its effect was compared with the vehicle-treated diabetic and non-diabetic mice

Results: The administration of persicarin (both 2.5 and 5 mg/kg body weight/day) caused a significantly increase in the body weight gain and liver weight. The increased glucose, hepatic functional parameter levels in serum, and glucose and glucose transporter type 4 (GLUT4) protein expression levels in the liver of diabetic mice were significantly decreased by persicarin. Moreover, the liver of diabetic mice exhibited the higher values of oxidative stress parameter (reactive oxygen species, peroxinitrite, and thiobarbituric acid-reactive substance); however, persicarin administration acts as a regulator in oxidative stress caused by overexpression of nicotinamide adenine dinucleotide phosphate (NADPH) oxidase subunit, such as Nox-4 and P47phox. In addition, persicarin altered the abnormal expressions of pro- inflammatory transcription factors and inflammatory protein expressions. Taken together, these results suggest that that persicarin suppress diabetes-induced pro-inflammatory factors by reducing oxidative stress through down- regulation of hyperglycemiaupregulated NADPH oxidase activation.

Conclusion: The present study demonstrated that the administration of persicarin isolated from the Oenanthe javanica had a hepatoprotective effect against inflammatory response under type 1 diabetes through regulations of hyperglycemia- upregulated NADPH oxidase activation.

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