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Hydroxyl Safflower Yellow Pigment A (HSYA) Improves Vascular Endothelial Injury Induced by LPS: *In Vitro* Study

Yan LI, Jingping GE, Yuanyuan YIN, Xiaomei LI, Boxiang ZHAO & Jianping GU *

Department of Vascular and Interventional Radiology, Nanjing First Hospital, Nanjing Medical University, Nanjing 210006, China

***SUMMARY*. The purpose of this study was to investigate the effects and mechanisms of Hydroxyl Safflower Yellow Pigment A (HSYA) in vascular endothelial injury by *in vitro* study. Human umbilical vein endothelial cells (HUVECs) were divided into 5 groups as normal control (NC), model (LPS treated) group, HSYA-L, HSYA-M, and HSYA-H groups. Cells in the HSYA treatment groups were treated with LPS, followed by 40, 80, and 120 mg/mL HSYA intervention (HSYA-L, HSYA-M, and HSYA -H groups), respectively. The cell proliferation, apoptosis, relative proteins, and mRNA (TLR4, MyD88 and NF-κB(p65)) were measured, by MTT, flow cytometry, WB, and RT-qPCR assay. Cellular immunofluorescence was used to evaluate NF-κB(p65) nuclear volume of difference groups. With HSYA supplement, the cell proliferation rates were significantly up-regulation with cell apoptosis and significantly down-regulation with TLR4, MyD88 and NF-κB mRNA and proteins expression and NF-κB(p65) nuclear significantly depressed with dose-dependent ($P < 0.05$, respectively). In conclusion, HSYA improved vascular endothelial injury induced by LPS via TLR4/MyD88/NF-κB(p65) pathway by *in vitro* study.**

RESUMEN. El propósito de este estudio fue investigar los efectos y mecanismos del pigmento amarillo hidroxilo de cártamo A (HSYA) en la lesión endotelial vascular mediante un estudio *in vitro*. Las células endoteliales de la vena umbilical humana (HUVEC) se dividieron en 5 grupos: grupos de control normal (NC), modelo (tratado con LPS), y grupos HSYA-L, HSYA-M y HSYA-H. Las células en los grupos de tratamiento HSYA fueron tratadas con LPS, seguido de 40, 80 y 120 mg/mL de HSYA (grupos HSYA-L, HSYA-M y HSYA-H), respectivamente. La proliferación celular, la apoptosis, las proteínas relativas y el ARNm (TLR4, MyD88 y NF-κB (p65)) se midieron mediante MTT, citometría de flujo, WB y ensayo RT-qPCR. La inmunofluorescencia celular se utilizó para evaluar el volumen nuclear de NF-κB (p65) de los grupos de diferencia. Con el suplemento HSYA, las tasas de proliferación celular aumentaron significativamente con apoptosis celular y una disminución significativa con el ARNm de TLR4, MyD88 y NF-κB y la expresión de proteínas y NF-κB (p65) nuclear significativamente deprimido con dosis dependiente ($P < 0,05$, respectivamente). En conclusión, HSYA mejoró la lesión endotelial vascular inducida por LPS a través de la vía TLR4/MyD88/NF-κB (p65) mediante estudio *in vitro*.

KEY WORDS: cell apoptosis, HUVECs, HSYA, MyD88, NF-κB(p65), TLR4.

* Author to whom correspondence should be addressed. *E-mail:* gujianping@tom.com

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