Protective effect of anthocyanin on lung ischemia-reperfusion injury

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Background and Goal of Study

Cyanidin-3-O- β -glucoside (C3G), a typical anthocyanin pigment that exists in the human diet, has been reported to protect the heart [1], brain [2], liver [3] and other organs [4] from ischemia/reperfusion (I/R)-induced inflammatory and apoptosis. However, unclear is the potential effect of C3G on Lung ischemia-reperfusion injury (LIRI). Thus, we hypothesized that C3G treatment plays a vital role in relieving LIRI.

Materials and Methods

Forty eight C57BL/6J adult male wild-type mice (n = 12/group, 20-25g), were randomly and evenly divided to I/R+C3G (I/R+C) group, I/R+saline (I/R) group, sham+C3G (sham+C) group and sham+saline (sham) group. LIRI mouse model was built via one-hour occlusion of the left pulmonary hilum and two-hour reperfusion. C3G(40mg/kg) and same dose of 0.9% sterile saline were administered to I/R+C group and other groups in advance, respectively. Blood and lung tissues of all mice were harvested after lung I/R for subsequent analysis. Arterial blood gas (ABG) analysis, Lung Wet/dry (w/d) weight ratio analysis, and Histological analysis were performed. The levels of inflammatory cytokines, including tumor necrosis factor- α (TNF- α), interleukin-6 (IL-6) and nuclear factor- κ B p65 (NF- κ B p65) activation in the serum and lung tissue, were measured by ELISA, Western blot (WB) and Quantitative polymerase chain reaction (qPCR), respectively. Also, toll-like receptor 3 (TLR3) levels were measured by the same methods. The apoptosis assays, including cleaved caspase-3 and TUNEL, were assessed.

Results and Discussion: The results exhibited significantly increased lung injury scores based on the outcomes of ABG, w/d weight ratio and lung Histological analysis after I/R. Moreover, the results of ELISA, WB and PCR showed the cytokine production including TNF- α , IL-6 and NF- κ B 65 activation, and TLR3 levels were substantially raised in I/R group. Pulmonary apoptosis after I/R was elevated as shown in TUNEL staining and activated caspase-3. C3G treatment, in contrast, markedly reduced the and pulmonary apoptosis in I/R+C group. In addition, pulmonary and circulating TLR3 levels in I/R + C group decreased compared with I/R group. But the expressions of the inflammatory cytokines in both lung tissue and serum did not show a difference.

Conflicts of interest: The authors have no conflicts of interest

Figure 1. Effect of C3G on lung ischemia/reperfusion injury. Left lung tissues were stained with HE. Representative photomicrographs are at 400 magnification.

Conclusion(s): Injection of C3G during preoperative period relieved pulmonary apoptosis induced by LIRI. The potential mechanism may be related to the decrease of TLR3 level and the inhibition of inflammation and apoptosis.

References

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