ABSTRACT

The Prevention Mechanisms of Endothelial Dysfunction by Cacao (Theobroma Cacao) Through Analysis of Plasma F2-Isoprostan Levels, Expression of NF-kB, CD-34, and Flk-1 on Cigarette Smoking Exposed Rat

Dina Helianti

Background: Smoking has known as causative factor of cardiovascular disease that was started with endothelial dysfunction. Polyphenols has known significantly prevent endothelial dysfunction. Cacao is a rich source of polyphenols. This study was designed to evaluate the cardioprotective effects of cocoa that mediated through the anti-oxidant effect, and was measured by plasma F2-isoprostane level, anti-inflammatory effect by expression of NFκB, and Endothelial Progenitor Cell (EPC) activation by expression of CD-34 and Flk-1 in coronary arteries. The condition of endothelial dysfunction was measured by expression of ICAM-1 and VCAM-1 in coronary arteries.

Material and Methods: These research was conducted in 2 phases: the first phase determined the efective dose of cocoa in reducing plasma F2-isoprostane level and the second phase analyzed the preventing mechanism of endothelial dysfunction by cocoa on cigarette smoke exposure. This study using cocoa powder. In the first phase, 3 doses of cocoa were used. This study subjected rats, divided into five groups: the normal control group (2 ml of aquabidest, air exposure); the cigarette control group (2 ml of aquabidest, cigarette smoke); cacao group 1 (1205 mg/kg BW/day, cigarette smoke); cocoa group 2 (2410 mg/kg BW/day, cigarette smoke); cacao group 3 (3615 mg/kg BW/day, cigarette smoke). Each group was treated for 14 days. In the second phase of the study using the optimal dose of cacao, based on the results from the first phase. NFkB, CD34, Flk-1, VCAM-1 and ICAM-1 were measured by immunohistochemistry.

Results: Cocoa 1205 mg/kg/day significantly decreases plasma F2-isoprostane level, NFkB, ICAM-1 and VCAM-1 expression of coronary arteries in cigarette smoking exposed rat ($p<0,05$). There was not a significant increases CD-34 but there was a significant increases in Flk-1 expression ($p<0,05$).

Conclusions: Cocoa in cigarette smoke-exposed rats can prevent endothelial dysfunction in 2 ways: the pathway of increasing EPC and an inflammatory pathway with a decrease in NFκB as an inflammatory mediator. The results of this study can be used as a basis for preventing endothelial dysfunction due to cigarette smoke by using cocoa.

Keywords: cigarette smoke exposure, cacao, F2-isoprostane, EPC, endothelial dsyfunction