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In Vitro Atheroprotective Effects of *Trigonella Foenum Graecum* (TFG) and its Saponins in LPS-Stimulated Human Coronary Artery Endothelial Cells

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Abstract

There has been a shift towards utilizing natural products as an adjunct therapy to standard treatment in the prevention of coronary artery disease, and *Trigonella foenum graecum* (TFG) is one of the potential natural products of interest. In the present study, we attempted to determine the effects of TFG and its saponins on atherosclerosis related biomarkers *in vitro*. Protein expression of markers of inflammation, endothelial activation and transcription factors were measured by Procarta™ and ELISA assays. Gene expression of the same markers were determined by qPCR and the interaction between monocytes and HCAECs were evaluated through monocyte binding assay following 16 h of treatment with TFG and saponins. Both TFG and its saponins exhibited reducing effects on atherosclerosis-related markers. Based on the area under the curve (AUC) analysis, TFG reduced protein and gene expressions of ICAM-1 and VCAM-1 better than the saponins, while saponins reduced E-selectin expression better than TFG. Saponins showed a reduction of gene and protein expressions of IL-6, IL-8, NF-κB p50 and p65 better than TFG. TFG is more effective in reducing binding of monocytes to endothelial cells than saponins. TFG better reduced endothelial activation but exerted weaker anti-inflammatory effects than saponins, suggesting the possible synergism with other compounds in the crude extract which enhances attenuation of endothelial activation while inhibiting anti-inflammatory properties of saponins in the crude extract.

Keywords: *Trigonella foenum graecum*, Saponins, Atherosclerosis, Inflammation, Endothelial activation

Introduction

The World Health Organization (WHO) reported that about 30 from 71 % of total deaths in Malaysia during 2002 due to chronic diseases is mainly caused by cardiovascular diseases (CVD). In 2008, CVD was the cause of 32 of 67 % of total deaths due to non-communicable disease [1,2]. The number may be increased if no intervention or preventive measures are initiated to control the prevalence of CVD. The majority of CVD are caused by atherosclerosis, a chronic process involving key processes which include endothelial activation, inflammation, oxidative stress and prothrombogenesis, leading to lipid-rich plaque formation in the walls of arteries which can lead to obstruction and ischaemia of major organs. According to the American Heart Association, approximately 75 % of fatal CVD reported are caused by atherosclerosis [1].

The current approach in the management of the atherosclerotic-related complications such as coronary artery disease (CAD) is to prevent them by addressing risk factors such as hypertension,