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Correlation of the GC-MS-based metabolite profile of *Momordica charantia* fruit and its antioxidant activity

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Abstract

Momordica charantia or bitter melon (Cucurbitaceae) is a widely consumed edible fruit with strong antioxidant properties. Due to these properties, it has been commercialised by the natural product industries as a coadjutant in the treatment of various ailments attributable to the deleterious effects of oxidants. The present work aimed to evaluate the antioxidant activity of *M. charantia* fruit extracts made with different compositions of ethanol:water, and to identify the metabolites that are responsible for this activity. To this end, the fruit samples were extracted using six different concentrations of ethanol in water (0, 20, 40, 60, 80, and 100%). Gas chromatography-mass spectrometry (GC-MS) and multivariate data analysis (MVDA) were used to identify significant antioxidants. The 80% ethanol:water extract showed the most significant ($p < 0.05$) antioxidant activity when tested with the 1, 1-diphenyl-2-picrylhydrazyl (DPPH) and ferric reducing antioxidant power (FRAP) antioxidant assays. The multivariate data analysis revealed that the metabolites related to this antioxidant activity were gentiobiose, glucose, galactonic acid, palmitic acid, galactose, mannose, and fructose.

Introduction

The prevalence of diabetes mellitus (DM) has been escalating. It was estimated that in 2030, Malaysia would have a total number of 2.48 million cases. As a result, the healthcare expenditure on DM accounted for 16% of the total healthcare expenditure in Malaysia; equivalent to USD 1.01 million annually, as DM becomes one of the major problems faced by the country (Ashari *et al.,* 2016).

It is well known that the oxidative stress resulting from hyperglycemia is closely related to the development and progression of DM and its related complications. The aetiology of DM through oxidative stress is assumed to be due to the increased production of free radicals or impaired antioxidant defences (Yaribeygi *et al.,* 2019). Mechanisms of the diabetic complications where increased oxidative stress is involved in are through the activation of transcription factors; advanced glycated end products (AGEs), a mitogen-activated protein kinase (Yang *et al.,* 2019; Dharshini *et al.,* 2020; Kim *et al.,* 2020).

Another mechanism that promotes the production of free radical is through the interaction of glucose with proteins which further increases the production of amadori product, followed by (AGEs). These AGEs, via their receptors (RAGEs), inactivate enzymes and alter their structures and functions.

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