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Review Article

The Therapeutic Potentials of Malaysian Medicinal Plants for Cardiovascular Diseases: A Review

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ABSTRACT

Cardiovascular diseases are among the most common diseases around the world with a considerably high rate of mortality. In view of this, multiple approaches have been proposed, especially in exploiting the potential of herbal plants due to their availability, accessibility and medicinal values. This review was conducted based on 16 medicinal herbs available in Malaysia as listed in the Malaysian Herbal Monograph. The aim was to study their effects on the alleviation of cardiovascular diseases, to analyse the relationship between the phytochemical compounds of herbal plants in relation to cardiovascular diseases and to review their underlying mechanism of action. The medicinal plants are organised based on their effects on alleviating cardiovascular diseases either directly or indirectly through reduction of cardiovascular disease risk factors, such as antihyperlipidemic, vasodilatory effects, cardioprotective and anticoagulant effects. Both *in vivo* and *in vitro* studies are included in this review to demonstrate the scientific value of related plants in cardiovascular diseases. In conclusion, many studies have identified a diversity of herbal plants in Malaysia to possess cardiovascular-relieving activities. Further studies can be conducted to develop these plants for cardiovascular disease treatment or prevention.

Key words: Antihyperlipidaemia, anticoagulant, cardioprotective, cardiovascular diseases, Malaysian plants, vasorelaxant

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INTRODUCTION

Cardiovascular diseases (CVDs) have been recognised as a major contributor to death worldwide. CVDs include a vast range of disorders which can be grouped into coronary heart disease, cerebrovascular disease, peripheral arterial disease as well as deep vein thrombosis and pulmonary embolism. Generally, CVDs are diseases that mainly affect the heart and blood vessels. Common CVDs experienced by individuals include heart attack, heart failure, stroke, coronary artery disease and thromboembolism. The World Health Organisation (WHO) estimated 19.8 million deaths from CVDs in 2022, representing about 32% of global deaths, and 85% of these were due to heart attack and stroke (WHO, 2025). A similar trend is seen in Malaysia, where the second leading cause of death in the country is ischaemic heart disease in 2023 (15.1%), and the leading cause of death in 2023 is pneumonia (15.2%) (DOSM, 2024). The prevalence might be due to several risk factors, including hypertension, high blood cholesterol, obesity, smoking and physical inactivity. They were shown to be higher, especially among middle- to low-income countries due to poor healthcare approaches and a lack of awareness of a healthy lifestyle (WHO, 2025).

One of the most common CVDs suffered by the population is heart failure, which affects more than 64 million people worldwide (Savarese *et al.*, 2023). Heart failure is diagnosed when there is any structural or functional damage to the cardiac components such as the valves, endocardium and heart rhythm. These result in the pooling of blood in the ventricle and thus reduced cardiac output. The risk of heart failure increases with age and other diseases such as hypertension, obesity and coronary artery disease. These diseases cause a progressive deterioration of the heart structures and functions. Moreover, direct cardiac damage is also possible with the use of cardiotoxic agents such as anthracycline-containing therapy (Schwinger, 2021).

In relation to heart failure, myocardial infarction (MI) is an acute CVD that is characterised by sudden death of the myocardial tissues. This is due to insufficient blood and oxygen supplies to the heart. Multiple aspects have been demonstrated to cause a reduction in blood supply to the heart. Occlusion of a coronary artery by plaques from atherosclerotic ruptures contributes to 70% of this reduction (Mechanic *et al.*, 2022). Immediate reperfusion of the heart remains the mainstay treatment of MI. The time taken to receive treatment determines the severity of the cardiac damage. However, reperfusion treatment predisposes the patient to ischaemia-reperfusion injury. This is characterised by a contradictory exacerbation of cardiomyocyte dysfunction and cell death after restoration of

blood supply to the ischaemic heart tissues. It has been shown that reactive oxygen species (ROS) such as superoxide anion, hydrogen peroxide and hydroxyl radical formed during the ischaemic state are responsible for the post-ischaemic-reperfusion damage. This is due to the release of proinflammatory markers and activation of endothelial cells. These compositely disrupts the permeability of cells and ultimately results in cell death (Mandalaneni *et al.*, 2024).

Secondary to ischaemic heart disease, stroke is another cause of death worldwide, including Malaysia. It accounts for approximately 8.7% of total mortality in the country (Kuriakose & Xiao, 2020). Stroke is a multifactorial neurological disorder which can be categorised based on the pathophysiological aspects: haemorrhagic and ischaemic stroke. Ischaemic stroke is more prevalent, where it makes up roughly 85% of stroke incidences. Haemorrhagic stroke, on the other hand, contributes to around 15% of the cases (Michel *et al.*, 2020). Generally, the prevalence of stroke is seen to be higher among older populations (more than 55 years old) and in low- to middle-income countries. Ischaemic stroke is caused by the reduction of blood flow to the brain due to the formation of a thrombus in the blood vessels or an embolic event. This happens when clots from other parts of the body, usually from the heart, travel to the affected vessels, causing blockage (Kuriakose & Xiao, 2020). Therefore, prevention of blood clot formation is important in reducing the occurrence of both thrombotic and embolic ischaemic stroke. Antiplatelets such as aspirin and clopidogrel remain the mainstay treatment in preventing the recurrence of stroke. Anticoagulants such as warfarin are used in patients with atrial fibrillation (Hackam & Spence, 2019).

Risk factors for the incidence of CVDs can be categorised into non-modifiable and modifiable. Non-modifiable risk factors, which include age, gender, ethnic background and genetic predisposition, increase the prevalence of CVDs in a distinct way and are not reversible (WHO, 2025). On the contrary, modifiable risk factors, which include hypertension, high blood cholesterol, smoking, diabetes, obesity, unhealthy diet and sedentary lifestyle, can be improved through pharmacological and non-pharmacological therapies (Yusuf *et al.*, 2020). According to the National Health and Morbidity Survey 2023, about 29.2% of Malaysians are suffering from hypertension, but only 17.3% of that number are diagnosed. It was also reported that among the hypertensive patients who are taking medication, about 52% still have inadequately controlled blood pressure (MOH, 2024). In addition, long-term elevated levels of low-density lipoprotein cholesterol (LDL-c) and non-high-density lipoprotein (HDL) cholesterol have been demonstrated to increase the risk of CVDs (Gidding & Allen, 2019). Excessive LDL-c circulating in the blood vessels will be taken up by the activated pro-atherogenic endothelial cells and adhere to the vessel wall. This process is facilitated by the increased expression of the surface adhesion molecules, for instance vascular cell adhesion molecule 1 (VCAM-1) and intracellular cell adhesion molecule 1 (ICAM-1). Over time, fatty streaks will be formed along the affected area, and this will promote inflammation, which then facilitates phagocytosis. After ingesting LDL-c, macrophages lose their function and become foam cells. These foam cells reside in the vessel wall and promote a positive feedback loop in recruiting additional leukocytes. Subsequently, fibrous cells, which block the blood vessels, causing atherosclerosis, will be formed (Kaski, 2024) (Figure 1). In short, the incidence of CVDs can be reduced by targeting factors which contribute to hypertension and high blood cholesterol.

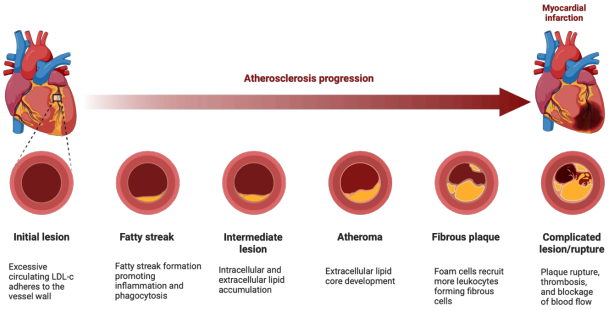

Fig. 1. Atherosclerosis formation leading to MI. (Created in BioRender. Chua, E. (2026) https://BioRender.com/80vm5s2)

Herbal medicines have been used to treat minor and major illnesses since ancient times. Written evidence ranges from the Chinese book "Pen TS'ao", which describes treatment properties of a total of 237 medicinal plants, to the Indian holy books, the Vedas. Vedas illustrate the treatment modalities of various plants originating from the botanical-rich land of India (Hou *et al.*, 2016). The presence of multiethnic cultures and biodiversity in Malaysia encourages the use of traditional Malay medicines, traditional Chinese medicines and Ayurvedic medicines. Herbal medicines are mainly used in the prevention and treatment of minor ailments or chronic diseases. They are also used for improvement of general health despite the availability of conventional therapies (Joachimdass *et al.*, 2021). A survey by Aziz and Tey (2009) reported that the usage of herbal medicines among Malaysians is more prevalent among women, Malay communities, a higher income group and those who are suffering from health issues, with a positive perception of the beneficial effects of herbal medicines. Herbal medicines are also used among the elderly population due to their accessibility and affordability as well as recommendations from family and friends. The immense potential of several Malaysian medicinal plants has been thoroughly investigated and characterised. For instance, root extracts from *Eurycoma longifolia* Jack (tongkat ali) and its bioactive compound eurycomanone have been extensively studied to improve male fertility. This is achieved by enhancing sexual desire and performance as well as by improving testosterone levels and sperm quality (Leisegang *et al.*, 2022). Apart from traditional plants, common vegetables and spices such as *Momordica charantia* L. (bitter melon) and *Zingiber officinale* Roscoe (ginger) have also been studied in-depth. They demonstrated antidiabetic and anti-inflammatory activities, respectively (Bekkouch *et al.*, 2023; Liu *et al.*, 2023). Considering the association between cardiovascular diseases and medicinal plants, this review aimed to summarise the relationship between the phytochemical compounds of herbal plants in relation to cardiovascular diseases. Their underlying mechanism of action are also discussed.

MATERIALS AND METHODS

An in-depth review on the phytochemical compositions and the pharmacological properties of medicinal plants in Malaysia in relation towards cardiovascular diseases was carried out through search engines such as PubMed, Google Scholar, ScienceDirect and Scopus from the year 2000 to 2023. The keywords used were "cardiovascular disease," "hypertension," "dyslipidaemia", "antioxidant," "antithrombotic," "anticoagulant," "heart failure," "myocardial infarction," "herbal plants," "traditional plants," "herbs," "Malaysia," "in vivo," "in vitro," and "human studies'' in different combinations possible. This review generally focuses on recognising and gather-

ing information on Malaysian medicinal plants that have been reported to possess medicinal effects on cardiovascular diseases or intermediate diseases linked to CVDs such as hypertension and dyslipidaemia through in vitro, in vivo, or clinical studies.

RESULTS AND DISCUSSION

Potential mechanisms of cardiovascular disease agents

Anticoagulant

The normal coagulation pathway is mediated by a balance between the initiation of the pro-coagulant pathway and the mechanism to inhibit this pathway. The pro-coagulant pathway is responsible for the initiation of blood coagulation during injury. Therefore, the role of endogenous antithrombotic substances is essential in limiting clot formation and preventing the propagation of thrombus. During injury or inflammation, inflammatory cytokines, specifically thromboxane A2, are produced and secreted by the activated platelets. This is to promote mass platelet aggregation and conversion of fibrinogen into fibrin, which adds stability to the clot, resulting in the formation of a platelet plaque. However, the formation of clots or platelet plaques in the blood vessels will lead to occlusion and progressively attenuates blood flow to the respective organs. Blockage within blood vessels supplying blood to the brain and heart will cause stroke or myocardial ischemia, respectively. Therefore, anticoagulants are essential to prevent unnecessary blood clots by suppressing the function or synthesis of coagulation factors or co-factors such as Vitamin K (Rishavy *et al.*, 2018).

Antihyperlipidaemic activity

Hyperlipidaemia is a medical condition in which there are elevated blood cholesterol and triglyceride (TG) levels subjected to hereditary factors or acquired through secondary factors such as unhealthy diet, poor lifestyle or medication side effects (AHA, 2020). Ingested lipids circulate in the blood vessels in the form of lipoproteins such as low-density lipoprotein (LDL), very low-density lipoprotein (VLDL), high-density lipoprotein (HDL) and chylomicrons. They constitute varying proportions of cholesterol, TG, and phospholipids in association with proteins (Feingold, 2021). Many studies have correlated the elevated levels of LDL cholesterol with the increased prevalence of atherosclerosis. This suggests that excessive lipids accumulating around the innermost layer of endothelial cells trigger the downstream pathology of atherosclerotic plaque formation through inflammatory mechanisms. In addition, blockage of the blood vessels at the affected area or distant blood vessels could lead to the development of hypertension or MI and stroke for the latter. A lower atherogenic index can be achieved by using antihyperlipidaemic agents. These agents will lower lipid parameters such as TG and LDL levels while increasing levels of HDL in blood serum (Kaski, 2024).

Cardioprotective and cardiomyocyte preservation post MI

MI, also known as a heart attack, is an acute medical condition when the balance between oxygen demand and supply of cardiac tissues is disrupted. This is due to blood vessel blockage and/or insufficient blood flow, resulting in ischaemic death of myocardial tissues. Ischaemia results in inhibition of adenosine triphosphate (ATP) generation from oxidative phosphorylation. This initiates an alternative pathway through anaerobic glycolysis, which causes the accumulation of lactate, leading to intracellular acidosis (Frangogiannis, 2015). Therefore, reperfusion through percutaneous coronary intervention (PCI) or thrombolytic therapy remains the mainstay of MI treatment. This is to restore sufficient oxygen supply and prevent cardiomyocyte death (Mandalaneni *et al.*, 2024). However, it is hypothesised that reperfusion post MI induces severe myocardial damage along with structural alterations. This is mediated through downstream apoptotic or necrotic pathways induced by oxidative stress and inflammatory markers. These can act as potential targets for cardioprotective compounds (Duan *et al.*, 2024).

Vasorelaxant and vasodilatory activities

Normal blood pressure can be maintained through preservation of cardiac output and peripheral vascular resistance. It has been hypothesised that early hypertension is caused by the raised of cardiac output in relation to sympathetic overactivity. This is followed by compensatory development of peripheral resistance elevation to block the flow of high-pressure blood into the capillary bed and disrupt cell homeostasis (Harrison *et al.*, 2021). Therefore, reduction of peripheral resistance through relaxation of vascular smooth muscle remains the centre of the target for treatment and prevention of hypertension. Vasorelaxant activity can be achieved through several possible mechanisms: the inhibition of alpha-adrenergic-stimulated vasoconstriction, the inhibition of potassium-induced depolarisation vasoconstriction, the attenuation of intracellular calcium and the stimulation of nitric oxide (NO) release (Trammel & Sapra, 2023).

Malaysian plants with cardiovascular activities

Acorus calamus L.

Acorus calamus L. (sweet flag, local name - jerangau) has long been known as a herbal plant for the therapy of various minor and major illnesses including neurological diseases, gastrointestinal or digestive problems and vascular disorders (Rajput *et al.*, 2014). Based on the preliminary documentation, both hypotensive and vascular modulating effects were obtained from crude aqueous-methanolic extract, ethyl acetate and hexane fractions. Both the crude extract and the ethyl acetate fraction inhibited phenylephrine- and high-potassium-concentration-induced vasoconstriction to a different extent. This is in addition to a dose-dependent prevention of phenylephrine peak formation in calcium-free medium. Hexane fractions produced an equal potency in phenylephrine- and K-induced contractions. The hexane fraction also demonstrated additional vasodilatory activity specifically in endothelium-intact aortic rings. This can be explained by the NO mechanism. The hypotensive activity of *A. calamus* might be due to a synergistic effect of restricting the entrance of extracellular calcium through voltage-dependent channels and receptor-operated channels. This is in addition to preventing the release of calcium from internal storage. It was also reported that the crude extract and the hexane fraction of *A. calamus* promote vasodilatory effect through mediation of endothelium-derived hyperpolarising factor (EDHF) release (Shah & Gilani, 2012).

The hypolipidaemic activity of *A. calamus* was initially studied in the atherogenic diet-induced hyperlipidaemic rats. The serum cholesterol and TG were significantly reduced while the HDL levels were significantly elevated with the administration of 100 mg/kg of ethanolic extract and 200 mg/kg of ethanolic and aqueous extract. Nevertheless, the aqueous extract demonstrated a lower hypolipidaemic effect compared with the ethanolic extract (Parab & Mengi, 2002). The potent activity of the ethanolic extract might be due to phytochemicals such as alkaloids and tannins. Previous studies reported that tannins cause an elevation in faecal bile acid excretion, thus lowering cholesterol levels (Issac *et al.*, 2018). However, literature on a specific tannin which demonstrate this effect in *A. calamus* is limited.

The cardioprotective effect of *A. calamus* was evaluated in isoproterenol-induced cardiac damage treated with 70% ethanolic rhizome extract. A lower serum calcineurin level was observed. Calcineurin induces cardiomyopathy by the downstream dephosphorylation of nuclear factor of activated T-cells (NFAT). The study also suggested that the antioxidant effect of *A. calamus* possibly plays a role in the cardioprotective effect. Moreover, 100 mg/kg and 200 mg/kg of *A. calamus* supplementation causes reversal of myofibril thickness and disorganised muscle fibres and pyknotic cells. This is in addition to

the maintenance of heart weight to body weight ratio, which further supports its cardioprotective effect (Singh *et al.*, 2011).

***Anacardium occidentale* L.**

Anacardium occidentale L. (cashew, local name - gajus) is an oleaginous nut that is rich in lipid content composed mainly of unsaturated fatty acids such as oleic and linoleic acid. This is in addition to other phytochemicals including flavonoids, anthocyanins, tannins and fibres (Baptista *et al.*, 2018). An in vivo study conducted on dyslipidaemic rats with co-administration of *A. occidentale* successfully lowers body weight and reduces visceral and retroperitoneal fat deposition. It also causes elevation of HDL level and excretion of faecal fat (Dias *et al.*, 2019). Fibre and unsaturated fatty acids in the extract might contribute to these effects (Bhaskaran *et al.*, 2017).

***Areca catechu* L.**

Areca catechu (betel nut, local name – pinang) is an indigenous herbal plant that is cultivated and used widely throughout Southeast Asia. Many studies have suggested potential medicinal properties of *A. catechu*, including antihypertensive, cariostatic, and antiparasitic. It is also used to treat minor ailments such as diarrhoea, beriberi and pharyngitis (Ali & Khuwaja, 2011). It was reported that the tannin fraction of *A. catechu* seeds (areca tannin) demonstrated in vivo blood pressure-lowering activity. This is possibly attributed to angiotensin-converting enzyme (ACE) inhibition and angiotensin blocker activity in reference to a reduction in levels of both angiotensin I and II. A heptapeptide isolated from the areca nut kernel globulin also showed ACE inhibitory activity (Liu *et al.*, 2021).

***Carica papaya* L.**

Carica papaya L. (papaya, local name – betik) is a medicinal plant that is widely cultivated in tropical and sub-tropical regions. It has been traditionally used in the treatment of hypertension using extracts from its fruits and leaves. Deoxycorticosterone (DOCA)-salt and renal-induced hypertensive rats treated with *C. papaya* ethanolic extract demonstrated promising hypotensive activity. This is possibly due to the suppression of adrenomedullary catecholamine release. Moreover, *C. papaya* extract also causes dose-dependent vasorelaxation in isolated rabbit arterial strips. However, was attenuated by a non-selective alpha blocker, proposing alpha adrenoceptor activity (Eno *et al.*, 2000). *C. papaya* extract also showed inhibitory activity of the angiotensin-converting enzyme (ACE) (Brasil *et al.*, 2014).

C. papaya seed ethanolic, ether and aqueous extracts were shown to decrease the levels of serum TC, TG and LDL while increasing the HDL level in a dose-dependent manner. The aqueous extract was the most potent. Moreover, the ethanolic and aqueous extracts preserved hepatic architecture and prevented the accumulation of lipids in the liver (Iyer *et al.*, 2011). In addition, the chloroform extract of *C. papaya* potently lowered levels of serum LDL, liver cholesterol and atherogenic index and increased the level of HDL. This was confirmed by the histological representation of a lesser extent of hepatic steatosis and minimal accumulation of lipid droplets in hepatocytes (Zetina-Esquivel *et al.*, 2015). *C. papaya* fruit juice also significantly reduced serum TG, TC and LDL levels. This is in addition to body and adipose tissue weight loss possibly mediated through the inhibition of pancreatic lipase activity in facilitating intestinal absorption of dietary fats (Od-ek *et al.*, 2020).

***Centella asiatica* (L.) Urb**

Centella asiatica (L.) Urb (gotu kola, local name – pegaga) has been traditionally used in the treatment of urinary tract infection, bladder inactivity, and venous disorders (Kant *et al.*, 2019). Previous studies reported the hypotensive effect of the ethyl acetate fraction. The vasodilatory effect observed might be partially contributed to by its high content of triterpenoids such as asiaticoside, madecosside, asiatic acid and madecassic acid. These compounds were shown to gradually lower the blood pressure in phenylephrine-induced contraction with an ED_{50} ranging from 9 to 11 mg/kg for systolic, diastolic and mean arterial pressure (Harwoko *et al.*, 2014). The hypotensive activity observed also might be due to its protection and preservation of vascular endothelial cells (Bian *et al.*, 2012). This is supported by another study in hypertensive rats. The ethanolic extract of *C. asiatica* was shown to maintain the normal serum level of NO and the downstream process of elevated endothelial nitric oxide synthase (eNOS) expression. This causes vasodilation and reduction of reactive oxygen species (ROS) induced oxidative damage to the endothelial cells (Bunaim *et al.*, 2021).

The hypolipidaemic activity of *C. asiatica* was first presented in vivo in rats fed a high-cholesterol diet treated with the aqueous extract. The lipid parameters, including LDL, VLDL, TC and TG levels, were significantly reduced in a dose-dependent manner while the HDL was significantly elevated. In addition, the extract demonstrated in vivo antioxidant activity through a reduction of oxidative stress markers such as NO and thiobarbituric acid reactive substance (TBARS) and increased the levels of antioxidant enzymes superoxide dismutase (SOD) and reduced glutathione (GSH). The extract also inhibited lipid peroxidation, which is an essential step in atherosclerosis development. Multiple phenolic antioxidant compounds were identified in the extract, including gluconic acids, ferulic acid, kaempferol, and asiatic acid (Kumari *et al.*, 2016).

The cardioprotective effect of *C. asiatica* was investigated using ischaemic-reperfusion rat models which were pretreated with 2, 10 and 50 mg/kg/day of madecassoside. It was observed that the levels of the myocardial injury biomarkers creatine kinase (CK) and lactate dehydrogenase (LDH) decreased post-treatment. The post-ischaemic ventricular function was restored, and the infarct size was minimised with higher doses (10 & 50 mg/kg/day). In addition, free radicals associated with subsequent inflammation post-ischemia were reduced, and a protective effect from cell apoptosis was observed (Bian *et al.*, 2008). Moreover, the cardioprotective effect of asiatic acid was demonstrated in oxygen-glucose deprivation-induced ischaemic and reoxygenation H9c2 cells through several mechanisms. Those include the regulation of ROS and calcium feedback loop, the phosphorylation of Akt-glycogen synthase and kinase-3β (Akt/GSK-3β) downstream mechanism. In addition, the elevation of hypoxia-inducible factor 1-α (HIF-1α), the reduction of caspase-3 and caspase-9, and the restoration of B-cell lymphoma protein 2 (Bcl2)/Bcl2-associated X (Bax) ratio. These result in a cumulative protective effect through suppression of apoptotic activity, inhibition of spike in intracellular ROS as well as preservation of mitochondrial function (Huang *et al.*, 2016). It was also proposed that the protective mechanism of asiatic acid is through the increment of glucose transporter type 4 (GLUT4) in the heart plasma membrane. This promotes the movement of glucose into cardiomyocytes, preventing glycogenolysis and gluconeogenesis. Subsequently, improving the mRNA expression and protein levels of peroxisome proliferator-activated receptor γ (PPARγ) (Dai *et al.*, 2018). Besides cell necrosis and apoptosis, autophagy of cardiomyocytes is also a common destructive mechanism that occurs post MI and reperfusion. Asiatic acid was also shown to stabilise the Bcl-2/beclin-1 complex and to depress p-38 phosphorylation and subsequently preserve cells from apoptosis or autophagy (Liu *et al.*, 2020).

***Cucumis sativus* L.**

Cucumis sativus L. (cucumber, local name – timun) is from the Cucurbitaceae family and commonly used as food among the populations of Africa, Asia and South America. It is rich in macro and micronutrients. It is also used to treat a wide range of ailments, namely inflammation (Agatemor *et al.*, 2015) and hyperlipidaemia (Soltani *et al.*, 2017). A detailed study on the hypotensive activity of *C. sativus* was only reported recently. The ethanolic seed extract of *C. sativus* was shown to cause vasorelaxation in both endothelial-

intact and denuded aortic rings contracted by phenylephrine and potassium. The phenylephrine-induced contraction was partially or completely blocked by atropine and L-N^G-nitroarginine methyl ester (L-NAME). This indicates a possible involvement of calcium antagonistic activity in addition to the NO-promoted endothelium-derived relaxing factor mechanism (Fu *et al.*, 2012).

The seeds of *C. sativus* have been traditionally used as a health supplement in lowering lipid levels. However, studies on the hypolipidaemic activity of *C. sativus* both in vitro and in vivo are lacking. Lipid-lowering activity of *C. sativus* was demonstrated in a randomised double-blind placebo-controlled clinical trial. A single oral administration of 500 mg of *C. sativus* seed ethanolic extract for 6 weeks potently lowered serum TC, TG and LDL levels while raising HDL levels significantly (Wahid *et al.*, 2022). Literature discussing the hypolipidaemic mechanism of action of *C. sativus* is scarce. However, it was proposed that the unsaturated fatty acids such as linoleic acid (Fu *et al.*, 2012), resins (Azadmehr *et al.*, 2014) and phytosterols (Kelishadi *et al.*, 2016) present in *C. sativus* seeds are responsible for the lipid-lowering effect.

***Cymbopogon citratus* (DC.) Stapf**

Cymbopogon citratus (DC.) Stapf (lemongrass, local name – serai) is traditionally used among the Asian and Brazilian communities to treat rheumatic disease (Meenapriya & Priya, 2017), inflammatory conditions (Figueirinha *et al.*, 2010) and hypertension (Bastos *et al.*, 2010). Citronellol, an essential oil constituent of *C. citratus*, was shown to possess hypotensive activity in vivo. It also has a potent vasorelaxant effect in phenylephrine-, potassium depolarisation, and calcium chloride-induced contractions. This is indicative of a hypotensive mechanism through the inhibition of calcium influx through voltage-gated calcium channels and intracellular calcium release (Bastos *et al.*, 2010). Studies also suggest that citral, a monoterpenoid bioactive compound in *C. citratus*, produces relaxation in phenylephrine- and potassium depolarisation-induced vasoconstriction (Devi *et al.*, 2012). Methanolic extract of *C. citratus* leaves and roots also demonstrated similar hypotensive and vasodilatory activities (Simões *et al.*, 2020).

Supplementation of aqueous-ethanolic extract of *C. citratus* at doses of 100 and 200 mg/kg to isoproterenol-induced MI rat models significantly lowered the level of lipid peroxidation. This was shown by the reduced level of TBARS while upregulating the levels and activities of endogenous antioxidants such as SOD, GSH, glutathione S-transferase (GST) and glutathione peroxidase (GPx). This study also suggested the role of trace elements in the antioxidant activity of *C. citratus* extract. The reduction of Fe and Cu levels lowered the prevalence of lipid peroxidation while the elevation of Zn levels promoted the SOD activity (Gayathri *et al.*, 2011).

***Elaeis guineensis* Jacq.**

Elaeis guineensis Jacq. (oil palm, local name – kelapa sawit) originated from the tropical rainforest area of Africa. It was brought into and subsequently cultivated widely in South America (Brazil) and Southeast Asia (Malaysia & Indonesia) (Reddy *et al.*, 2019). *E. guineensis* possesses many medicinal benefits, including antibacterial (Chong *et al.*, 2007), antioxidant (Varatharajan *et al.*, 2013), anti-inflammatory (Anyanji *et al.*, 2013), anti-diabetic (Sharif *et al.*, 2015), and anti-cancer activities (Vijayarathna & Sasidharan, 2012). The potential antihypertensive activity of *E. guineensis* was demonstrated with its potent vasorelaxant effect on noradrenaline-induced vasoconstriction. The vasoconstriction is partly inhibited through removal of endothelium and co-administration of L-NAME and indomethacin. This suggests the role of NO and cyclooxygenase for this effect (Abeywardena *et al.*, 2002). This was further confirmed through the increased NO production and eNOS phosphorylation involving redox and PI3-kinase-dependent mechanisms (Ndiaye *et al.*, 2009). Catechins isolated from the methanolic leaf extract might be responsible for this effect (Jaffri *et al.*, 2011).

***Ficus deltoidea* Jack**

Ficus deltoidea Jack (mistletoe fig, local name – mas cotek) is a herbal plant possessing many medicinal benefits. It has been used in Malay traditional medicine for the treatment of diabetes (Farsi *et al.*, 2014), ulcer (Zahra *et al.*, 2009), and inflammatory disorders (Abdullah *et al.*, 2009) and less commonly in the treatment of hypertension. Among the limited evidence reported for the antihypertensive activity is the in vivo blood pressure-lowering effect in spontaneously hypertensive rats. This effect is possibly mediated through decrement of endothelin-1 without the involvement of the renin-angiotensin-aldosterone system (RAAS) (Ahmad Kamal *et al.*, 2019). In contrast, Abdul Azis *et al.* (2019) presented a dose-dependent blood pressure-lowering effect of aqueous-ethanolic extract of *F. deltoidea* probably due to the mechanism related to RAAS.

***Gynura procumbens* (Lour.) Merr.**

Gynura procumbens (Lour.) Merr. (longevity spinach, local name – sambung nyawa) is originated from the Compositae family. It is an essential herbal plant in Southeast Asia for the treatment of various diseases. The antihypertensive activity of *G. procumbens* was demonstrated in spontaneously hypertensive rats through reduction of systolic blood pressure. This is possibly mediated through nitric oxide production (Kim *et al.*, 2006). Moreover, multiple studies linked the vasorelaxant activity of *G. procumbens* to the RAAS. Hoe *et al.* (2007) demonstrated the in vitro ACE inhibition by *G. procumbens* fraction. It was also reported that *G. procumbens* fraction demonstrated potent vasodilatory activity in angiotensin II-induced vasoconstriction both in vivo and in vitro. This was mediated through the increased production of NO and prostaglandin I2 (PGI2) in addition to the enhancement of bradykinin effect through indirect action on bradykinin receptor 2 (Poh *et al.*, 2013). Interestingly, Iqbal *et al.* (2017) demonstrated the potent vasorelaxant effect of the *G. procumbens* petroleum ether leaf extract. The activity is possibly mediated through the inhibitory activity on calcium channels. This is in agreement with the study conducted by Hoe *et al.* (2011). Recent studies also suggested potential cholinergic pathway-mediated vasodilatory action of aqueous and methanolic extracts of *G. procumbens* leaves. The activity might be due to the isolated compound kaempferol 3-*O*-rutinoside, which inhibits the M3 receptor subtype (Shahlehi & Petalcorin, 2021).

Studies have shown that co-administration of ethanolic extract of *G. procumbens* at 250 and 500 mg/kg of body weight significantly reduced serum levels of LDL, TC and TG and increased HDL levels. In addition, *G. procumbens* extract has also been shown to reduce the lipid droplet accumulation within tissues. The major phenolic contents identified in the 80% ethanolic extract of *G. procumbens* were chlorogenic acid, gallic acid, kaempferol, quercetin, and rutin (Ahmad Nazri *et al.*, 2021). The hypolipidaemic effect of *G. procumbens* is probably attributed to the chlorogenic acid content. It was shown to lower the cholesterol and TG levels in hyperlipidaemic and insulin-resistant Zucker rats. In addition, it also inhibited fat absorption and enhanced fat metabolism (Shimoda *et al.*, 2006).

***Kaempferia galanga* L.**

Kaempferia galanga L. (aromatic ginger, local name – kencur), which originated from the Zingiberaceae family, is commonly grown in Southeast Asia, especially Indonesia, Malaysia, Singapore and India (Wang *et al.*, 2021). Its rhizome is usually utilised as a food flavouring or culinary spice. Medicinally, it is used to treat hypertension, rheumatism, asthma and bronchitis (Khare, 2007). Vasorelaxant effect of *K. galanga* was demonstrated in rat aortic smooth muscle by inhibition of high potassium and phenylephrine-induced contractions. This might be mediated through blockage of both voltage- and receptor-operated channels, hence hindering calcium influx. Further studies identified that ethyl cinnamate

and ethyl-*p*-methoxycinnamate, bioactive compounds in the *K. galanga* rhizome extract, are responsible for the vasorelaxant effect (Srivastava *et al.*, 2021). Ethyl cinnamate inhibited both high potassium and phenylephrine-induced vasoconstriction, suggesting the involvement of calcium channel blockage with additional factors related to NO and prostacyclin pathways (Othman *et al.*, 2002). This is supported by Srivastava *et al.* (2021), where ethyl-*p*-methoxycinnamate demonstrated a vasorelaxant effect through the opening of large conductance calcium-dependent potassium channels while the endothelium does not participate in the response.

***Labisia pumila* (Blume) Benth. & Hook.f. ex Fern.**

Labisia pumila (Blume) Benth. & Hook.f. ex Fern. (quicksilver, local name - kacip Fatimah) belongs to the Myrsinaceae family. It is a herbal plant that is used traditionally to promote female fertility as well as to aid childbirth and improve postpartum health (Abdullah *et al.*, 2013). Studies evaluating the antihypertensive activity of *L. pumila* are lacking until Manshor *et al.* (2020) presented a potent in vivo blood pressure-lowering activity of aqueous *L. pumila* extract. This was followed by the investigation of possible mechanisms of action through the NO-independent sGC/cyclic GMP pathway, decreased intracellular calcium release, escalation of myosin light chain phosphatase activity, inhibition of receptor-operated calcium channels and mildly through blockage of the voltage-gated calcium channels.

The hypolipidaemic potential of *L. pumila* was evaluated in hyperlipidaemic-induced rats and treated with 80% ethanolic extract and aqueous extract at 100, 200 and 400 mg/kg doses. Both aqueous and ethanolic extracts successfully lowered the TC, TG and LDL levels while raising the HDL level. In addition, both extracts also reduced the atherogenic index of plasma (AIP) and Castelli's risk index-I (CRI-I), with the ethanolic extract demonstrating a superior effect compared to the aqueous extract. Interestingly, at 400 mg/kg, the ethanolic extract showed comparable serum TG, HDL, AIP and CRI-I levels to those reported in the normal diet control and atorvastatin groups (Dianita *et al.*, 2016).

The cardioprotective effect of *Labisia pumila* was identified in the isoproterenol-induced MI rat models, where pretreated rats showed reduced levels of cardiac markers including alanine transaminase (ALT), aspartate aminotransferase (AST), creatine kinase MB (CK-MB), lactate dehydrogenase (LDH) and Troponin I. Moreover, the extract also significantly upregulated endogenous antioxidants GPx and SOD levels, protecting against ROS-induced myocardial damage. The severity of myocardial structural damage and cell necrosis was also reduced at lower doses of extract, while preservation of the cardiac structure and cell integrity near to normal was observed at higher doses (Dianita *et al.*, 2015).

***Melastoma malabathricum* L.**

Melastoma malabathricum L. (Malabar melastome, local name – senduduk) belongs to the Melastomataceae family. It is a perennial herb which is used traditionally among Malays, Chinese, Indian and Indonesian communities. Manicam *et al.* (2010) demonstrated strong novel in vitro anticoagulant activity in *M. malabathricum* water extract. The potential bioactive constituents and mechanisms of action involved were further investigated. Different compositions of phenolic acids, carbohydrates, and uronic acids present in the active extracts suggest that the anticoagulant effect is positively correlated to the concentration of polysaccharides and hexuronic acids (Khoo *et al.*, 2014). This is in agreement with studies reported on other plants (Pawlaczyk *et al.*, 2010). Further study revealed that cinnamic acid in combination with its derivative is one of many bioactive compounds contributing to the anticoagulant effect of *M. malabathricum* (Khoo *et al.*, 2015).

***Momordica charantia* L.**

Momordica charantia L. (bitter melon, local name – peria katak) has been long studied for its antidiabetic (Jiang *et al.*, 2020) and hypocholesterolaemic or dyslipidaemia activities (Saad *et al.*, 2017), which presented promising results. Senanayake *et al.* (2004) demonstrated that *M. charantia* methanolic fraction lowers liver TG level, suggesting possible hypocholesterolemic bioactive compounds present in this fraction. Moreover, the methanolic fraction lowers liver cholesterol without affecting serum cholesterol, suggesting an involvement of cholesterol ester reduction. In contrast, Matsui *et al.* (2013) demonstrated a reduction in serum total cholesterol (TC), LDL and HDL levels in hypercholesterolemic rats supplemented with *M. charantia* extract. In addition, the elevation of faecal bile acid indicated a reduction in bile acid reabsorption in the intestine. This led to the downstream process of blood cholesterol conversion into bile acid in the liver. In agreement with Matsui *et al.* (2013), Saad *et al.* (2017) suggested the involvement of farnesoid X receptor (FXR)/short heterodimer partner (SHP) mediated hepatic cytochrome P450 (CYP)7A1 in bile acid homeostasis, lowering the serum TC. It was also shown that *M. charantia* increased the expression of 3-hydroxy-3-methyl-glutaryl (HMG) messenger ribonucleic acid (mRNA) level, denoting that the reduction of serum cholesterol is mediated by downregulation of hepatic cholesterol synthesis. An additional mechanism proposed for the hypolipidaemic activity is related to the mediation of fatty acid synthase (FAS) and sterol regulatory element binding protein 1 (SREBP-1)c gene expression. The downregulation of these regulators resulted in the reduction of lipogenesis and sterol biosynthesis. The hypolipidaemic activity of different *M. charantia* parts; skin, flesh of fruit, whole fruit, and leaves was further established in hyperlipidaemic rat models through the attenuation of TC, TG and LDL levels while increasing the level of HDL (Mahwish *et al.*, 2017).

The role of *M. charantia* polysaccharide extract in MI was studied in isoproterenol-induced rat models supplemented with 50 mg and 100 mg/kg of extract for 25 days. The extract significantly reduced the prevalence of cardiac dysfunction in association with a drop in cardiac function markers, including AST, CK-MB and LDH and prevented the elevation in heart weight as well as heart-to-body weight ratio. The extract was also shown to downregulate lipid peroxidation (LPO), NO, inducible nitric oxide synthase (iNOS) expression and lower the levels of proinflammatory cytokines tumour necrosis factor α (TNF-α) and interleukin 6 (IL-6). Therefore, alleviating the inflammatory response of cardiac tissues such as oedema and infiltration of neutrophils. In addition, the study also suggested that the extract possesses antiapoptotic activity through prevention of Bcl-2 degradation in the damaged region (Raish, 2017).

***Morinda citrifolia* L.**

Morinda citrifolia L. (noni, local name – mengkudu) is a member of the Rubiaceae family. It is used traditionally for the treatment of various illnesses such as hypertension, dyslipidaemia (Shoeb *et al.*, 2016) and pain (Basar *et al.*, 2010). *M. citrifolia* juice was shown to lower systolic blood pressure in spontaneously hypertensive rats and to have in vitro ACE inhibitory activity (Yamaguchi *et al.*, 2002). *M. citrifolia* root extract also was shown to inhibit high potassium- and phenylephrine-induced contraction. This indicates the possible involvement of receptor blockage and voltage-operated calcium channels in addition to the inhibition of the release of intracellular calcium stores (Yamaguchi *et al.*, 2002; Gilani *et al.*, 2010). Another in vivo study demonstrated that the combination of *M. citrifolia* leaves and fruits exhibited a greater hypotensive effect than *M. citrifolia* leaves or fruits alone. This suggests the synergistic activity of rutin and scopoletin, which were isolated from leaves and fruit extract, respectively (Wigati *et al.*, 2017). In contrast to Gilani *et al.* (2010), Yoshitomi *et al.* (2020) suggested that *M. citrifolia* fruit juice potentially mediates blood pressure by increasing the production of NO through the regulation of glucagon-like peptide-1 receptor (GLP1R)-induced Ca(2+)/calmodulin-dependent protein kinase kinase-adenosine monophosphate-activated protein kinase

(CaMKKβ-AMPK-eNOS) pathway with subsequent endothelium-dependent vasodilation.

Various parts of *M. citrifolia*, including fruits, leaves, root and seed extracts, demonstrated significant inhibition of TC and TG levels at higher doses in tyloxapol-induced hyperlipidaemic rats. This indicates the inhibition of HMG-CoA, which results in the prevention of cholesterol biosynthesis (Mandukhail *et al.*, 2010). Studies also demonstrated that fruits, leaf, root and seed significantly lower levels of mean serum TC and LDL while increasing the level of HDL and a reduction in the atherogenic index (Mahadeva Rao & Shanmuga Sundaram, 2017). The main constituents identified in *M. citrifolia* seed oils include linoleic, oleic, palmitic, palmitoleic and stearic acids, with linoleic acid being the principal component, suggesting the possibility of its involvement in hypolipidaemic mechanism (Pazos *et al.*, 2011).

***Parkia speciosa* Hassk.**

Parkia speciosa Hassk. (bitter bean, local name – petai) is a herbal plant which is grown locally in Southeast Asia. It contains extensive concentrations of polyphenols and flavonoids (Siow & Gan, 2013). According to traditional records, the roots and seeds in the presence or absence of pods are used in the treatment of hypertension. This might be attributed to the ACE inhibitory activity of low molecular weight protein hydrolysate extracted from *P. speciosa* seeds (Lim, 2012). It was also reported that the methanolic extract of *P. speciosa* pods showed potential blood pressure-lowering activity in L-NAME-induced hypertension, possibly through preservation of NO (Kamisah *et al.*, 2017).

The possible cardioprotective effect of *P. speciosa* was conducted in L-NAME-induced rat models treated with methanolic empty pods extract. The extract inhibited the elevation of cardiac ACE activity and prevented the rise in nicotinamide adenine dinucleotide phosphate (NADPH) oxidase activity and lipid peroxidation. This resulted in the inhibition of angiotensin II (Ang II) production and its downstream transforming growth factor-β (TGF-β) and endothelin signalling pathway-induced cardiac hypertrophy. Major phytoconstituents present in the methanolic extract of *P. speciosa* empty pods include catechin, epicatechin, epigallocatechin gallate, quercetin, coutaric acid, tangeretin and apigenin (Kamisah *et al.*, 2017). Previously, polyphenols and quercetin were reported to possess ACE inhibitory and antioxidant activities, suggesting these activities in *P. speciosa* (Huang *et al.*, 2017). Studies on a specific cardioprotective mechanism demonstrated that *P. speciosa* extract significantly reduced the expression of nuclear factor kappa-light-chain-enhancer of activated B cells (NFkB) and p38 mitogen-activated protein kinase (MAPK) by activating MAPK phosphatases and decreasing the phosphorylation of nuclear factor of kappa light polypeptide gene enhancer in B-cells inhibitor, alpha (IkBα). This subsequently lowers the expression of pro-inflammatory markers including iNOS, vascular cell adhesion protein 1 (VCAM-1), cyclooxygenase 2 (COX-2), NO and ROS. Ultimately, lessen the occurrence of cardiovascular diseases associated with inflammation. The anti-inflammatory properties demonstrated by *P. speciosa* might be mediated by quercetin, which is a predominant phytochemical identified in the ethyl acetate fraction of *P. speciosa* (Gui *et al.*, 2019). Furthermore, another study reported that cardiac cell hypertrophy induced by the imbalance of the ROS/NO axis caused by Ang II was reduced in size when supplemented with *P. speciosa* extract. This is possibly mediated through the inhibition of extracellular signal-regulated protein kinase (ERK1/2), c-Jun N-terminal kinases (JNK) and p38 phosphorylation. This inhibits the downstream activation of transcriptional factors including certain hypertrophic gene products such as brain natriuretic peptide 32 (BNP) (Siti *et al.*, 2021).

Bioactive compounds of herbal plants discussed above and their underlying mechanism of action are summarised in Table 1. Chemical structures of those compounds are illustrated in Figure 2.

CONCLUSION

The utilisation of traditional plants for medicinal purposes has evolved drastically, and increasing studies were conducted to identify novel medicinal properties on a wide range of herbal plants. This is in addition to the potential bioactive constituents and possible mechanisms of action of both existing and newly identified pharmacological activities. However, the vast number of herbal plants around the world is associated with scattered evidence on their medicinal properties, resulting in the decline of knowledge and unreliable use of traditional herbs. Therefore, it is of importance to gather and document all the available studies in relation to the medicinal properties of herbal plants in order to maintain their quality and preservation. According to this review, Malaysian herbal plants are rich in polyphenols, flavonoids, terpenoids and catechins in addition to other phytochemicals acting individually or in combination for the treatment of cardiovascular diseases. The plants exhibited vasodilation, antihypertensive, antihyperlipidaemic, cardioprotective, and anticoagulant effects. These are mechanisms that are closely related to cardiovascular diseases such as coronary artery disease, atherosclerosis, hypertension and MI.

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ETHICAL STATEMENT

Not applicable.

CONFLICT OF INTEREST

The authors declare no conflict of interest.

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Table 1. Malaysian plants with potential cardiovascular mechanisms.

No.	Plants	Phytochemicals	Vasorelaxant mechanisms	References
1.	*A. calamus* (sweet flag)	Alkaloids and tannins	Inhibition of potassium-induced depolarisation vasoconstriction EDHF release Antihyperlipidaemic Cardioprotective effect	(Parab & Mengi, 2002) (Shah & Gilani, 2012) (Parab & Mengi, 2002) (Agila & Barringer, 2011)
2.	*A. occidentale* (cashew)	Flavonoids, anthocyanins, tannins and fibres	Antihyperlipidaemic	(Ali & Khuwaja, 2011)
3.	*A. catechu* (betel nut)	Heptapeptide	ACE inhibition	(Eno *et al.*, 2000; Liu *et al.*, 2021)
4.	*C. papaya* (papaya)	Kaempferol, quercetin, ferulic acid, caffeic acid, gallic acid, nicotiflorin, clitorin and manghaslin	Alpha-adrenergic stimulated vasoconstriction ACE inhibition Antihyperlipidaemic	(Ravikant *et al.*, 2012) (Brasil *et al.*, 2014; Iyer *et al.*, 2011) (Kant *et al.*, 2019; Zetina-Esquivel *et al.*, 2015)
5.	*C. asiatica* (gotu kola)	Asiatic acid, asiaticoside, ferulic acid, gluconic acid, kaempferol, madecassic acid and madecosside	Preservation of vascular endothelial cells Antihyperlipidaemic Cardioprotective effect	(Kant *et al.* 2019; Zetina-Esquivel *et al.*, 2015) (Bian *et al.*, 2008) (Dai *et al.*, 2018; Huang *et al.*, 2016; Liu *et al.*, 2020)
6.	*C. sativus* (cucumber)	Linoleic acid, resins and phytosterols	Attenuation of intracellular calcium Stimulation of NO release Antihyperlipidaemic	(Fu *et al.*, 2012) (Fu *et al.*, 2012) (Minaiyan *et al.*, 2011)
7.	*C. citratus* (lemon grass)	Citronellol and citral	Inhibition of phenylephrine- and potassium-induced depolarisation vasoconstriction Cardioprotective effect	(Devi *et al.*, 2012; Simões *et al.*, 2020) (Gayathri *et al.*, 2011)
8.	*E. guineensis* (oil palm)	Catechin	Inhibition of noradrenaline-induced vasoconstriction	(Abeywardena *et al.*, 2002)
9.	*F. deltoidea* (mistletoe fig)	Flavonoids, saponins, alkaloids, triterpenoids and terpenes	RAAS	(Abdul Azis *et al.*, 2019)
10.	*G. procumbens* (longevity spinach)	Kaempferol 3-*O*-rutinoside	ACE inhibition Calcium channel inhibition Antihyperlipidaemic	(Hoe *et al.*, 2007) (Iqbal *et al.*, 2017) (Ahmad Nazri *et al.*, 2021)
11.	*K. galanga* (aromatic ginger)	Ethyl cinnamate and ethyl-*p*-methoxycinnamate	Inhibition of phenylephrine- and potassium-induced depolarisation vasoconstriction	(Othman *et al.*, 2002)
12.	*L. pumila* (quicksilver)	Gallic acid, catechin, rutin, myricetin, 5-(Z-nonadec-14-enyl)resorcinol and demethylbelamcandaquinone ß	Inhibition of receptor-operated calcium channels Antihyperlipidaemic Cardioprotective effect	(Manshor *et al.*, 2020) (Dianita *et al.*, 2016) (Dianita *et al.*, 2015)
13.	*M. malabathricum* (Malabar melastome)	Polysaccharide, hexuronic acids and cinnamic acid	Anticoagulant	(Khoo *et al.*, 2015; Manicam *et al.*, 2010)
14.	*M. charantia* (bitter melon)	Momordicoside P, momordicoside G and Momordicoside F1	Antihyperlipidaemic Cardioprotective effect	(Matsui *et al.*, 2013; Saad *et al.*, 2017) (Raish, 2017)
15.	*M. citrifolia* (noni)	Linoleic, oleic, palmitic, palmitoleic and stearic acids	Stimulation of NO release Antihyperlipidaemic	(Othman *et al.*, 2002) (Mahadeva Rao & Shanmuga Sundaram, 2017; Mandukhail *et al.*, 2010; Pazos *et al.*, 2011)
16.	*P. speciosa* (bitter bean)	Polyphenols and flavonoids	Preservation of NO Cardioprotective effect	(Kamisah *et al.*, 2017) (Gui *et al.*, 2019; Huang *et al.*, 2017; Siti *et al.*, 2021)

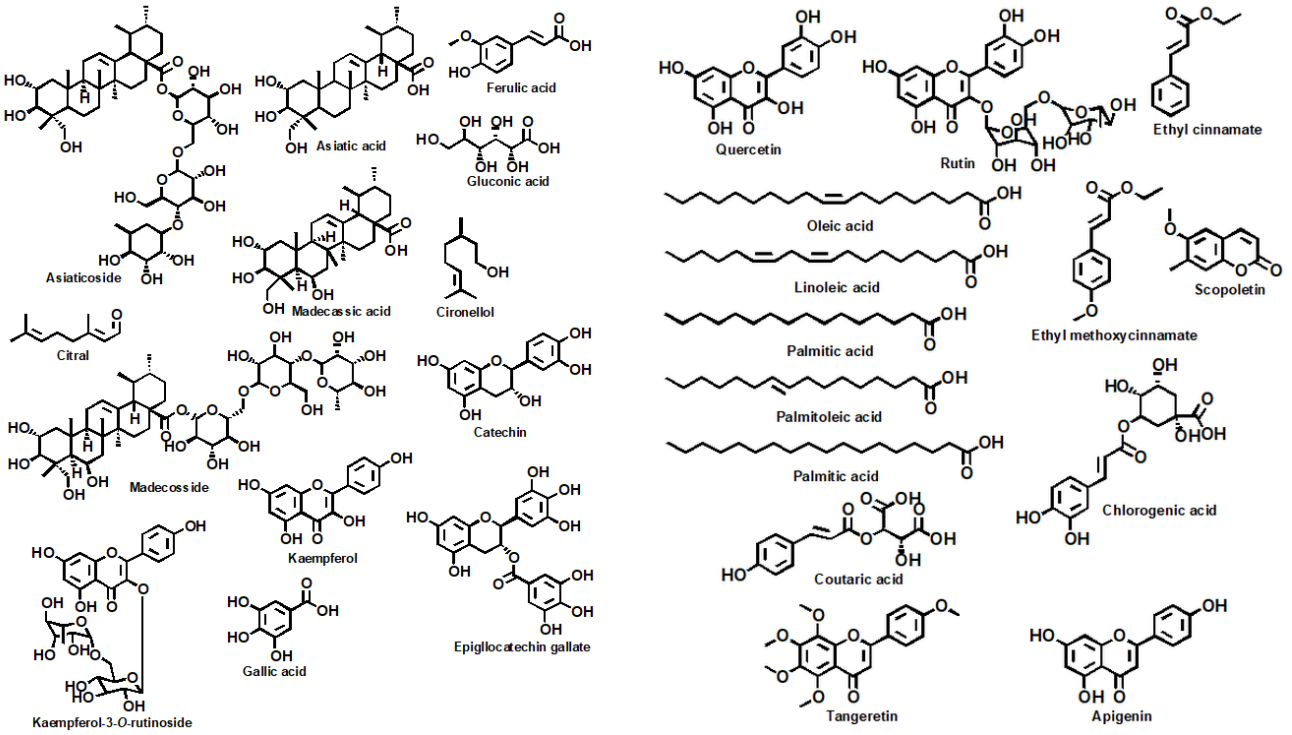

Fig. 2. Chemical structures of bioactive compounds identified in the reviewed Malaysian medicinal plants.

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