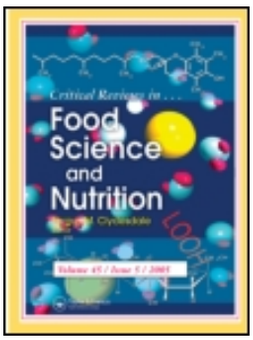

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The Health Benefits of Selected Culinary Herbs and Spices Found in the Traditional Mediterranean Diet

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The Mediterranean diet is considered one of the healthiest diets in the world. This is often attributed to low saturated fat consumption, moderate wine consumption, and high vegetable consumption. However, herbs and spices associated with these diets may also play an important role in the quality of this diet. This review summarizes the most recent research regarding the anti-diabetic, anti-inflammatory, anti-hyperlipidemic and anti-hypertensive properties of this collection of culinary species. Additionally, this review briefly summarizes studies performed on lesser known herbs from around the world, with the goal of identifying new culinary species that may be useful in the treatment or prevention of diseases.

Keywords Diabetes, inflammation, hyperlipidemia, metabolic syndrome, herbs, spices

INTRODUCTION

Since the release of the results from the Seven Countries Study (Keys et al., 1984), interest in the Mediterranean diet has grown exponentially. Recent epidemiological studies on individuals from the Mediterranean region indicate that adherence to a traditional Mediterranean diet is inversely associated with inflammation (Azzini et al., 2011) and all-cause mortality (Trichopoulou et al., 2003; Martinez-Gonzalez et al., 2008). Furthermore, the island of Ikaria in Greece is of special interest to the scientific community due to its disproportionate number of individuals over the age of 80 years old.

The Mediterranean diet refers to the dietary characteristics of the populations in Crete, Greece, and southern Italy. It consists of an abundance of plant foods with low to moderate amounts of dairy products. Olive oil is the principal fat and low to moderate amounts of poultry, red meat, fish and eggs are consumed. It is also characterized by moderate consumption of wine (Willett et al., 1995).

The PREDIMED study, a 4-year randomized controlled trial assessing the use of the Mediterranean diet for primary prevention of cardiovascular disease, showed that consuming this diet high in olive oil reduced levels of circulating oxidized LDL as well as reduced blood pressure (Fito et al., 2007). In another study, consuming a Mediterranean type diet was not associated with lower incidences of type II diabetes, however, it was associated with lower insulin levels among non-diabetics and with lower blood glucose before adjustment for obesity in the study population (Abiemo et al., 2013).

These observations may be attributed to several components of the Mediterranean diet, since this dietary pattern provides a nutrient profile that is high in fiber, vitamins and natural antioxidants and provides a high monounsaturated fatty acid to saturated fatty acid ratio (Martinez-Gonzalez et al., 2012). In fact, high consumption of the main components of the Mediterranean diet: vegetables, fruit, nuts, olive oil, cereals and legumes, and moderate red wine were predictors of lower mortality (Trichopoulou et al., 2009).

There may be, however, non-nutrient components to this dietary pattern that also contribute to the observed health benefits of the Mediterranean diet. The traditional Mediterranean diet is also high in wild greens and herbs (Nestle, 1995) and the current dietary recommendations from the Hellenic ministry of health suggest replacing salt with herbs and spices (Ministry of Health and Welfare, 1999). Analysis of flavonoid content of tradition Greek diets also suggests that herbs and spices are significantly responsible for the higher level of antioxidants consumed in this population (Vasilopoulou et al., 2005).

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Herbs and spices have long been used for both culinary and medicinal purposes. This review focuses on the herbs and spices of the Mediterranean diet and the benefits they may convey to conditions associated with metabolic syndrome, such as diabetes, inflammation, hyperlipidemia and hypertension. Additionally, the potential health benefits of selected herbs and spices from other cultures are presented in order to expand the knowledge of the therapeutic potential of the herb and spice cabinet.

The collections of herbs and spices that are reviewed in this paper have been drawn from ethnopharmacological studies from various regions of Europe, while taking into consideration their role as culinary seasonings. Parsley, oregano, basil, thyme have been used for medicinal and culinary purposes throughout Italy (Guarrera, 2005; Guarrera et al., 2005a,b; Cornara et al., 2009while all of these spices in addition to dill, fennel, marjoram, rosemary, lavender, bay leaf, sage, savory and hyssop are also used in areas of Spain, Greece and Portugal (Heinrich, 2005; Neves et al., 2009). Additionally, cumin and coriander were used in ancient Greece (Bown, 2001; Brussel, 2004).

The herbs and spices mentioned above are reviewed in the following pages. Table 1 provides the total polyphenol content of the herbs and spices discussed when the data were available. For convenience, summaries of in vivo studies addressing these herbs and spices and their effect on diabetes and hyperlipidemia are summarized in Tables 2 and 3, respectively. Figure 1 presents chemical structures of representatives of some bioactive compounds that are found in herbs and spices.

Lastly, since the herbs and spices discussed in this paper are of a culinary nature, it is important to consider the effect of cooking on the chemical properties of these plant products. Cooking methods that involve steeping herbs in a warm liquid, such as in the process of making soup, increase the antioxidant capacity of the herb extract (i.e., the soup), while extracts taken after grilling had a lower antioxidant capacity compared to the uncooked herb extract (Chohan et al., 2008). Steaming and sautéing have been reported to increase the antioxidant capacity and phenolic content of extracts taken after cooking (Trakoontivakorn et al., 2012).

Table 1 Total polyphenol content (mg GAE/100g) of selected herbs and spices*

Common Name	Botanical Name	Family	Total Polyphenols (Dried)	Total Polyphenols (Fresh)
Mediterranean				
Basil	*O. basilicum*	Lamiaceae	4318	232
Bay	*Laurus nobilis*	Lauraceae	4170	402
Cumin (seed)	*Cuminum cyminum*	Apiaceae	2038	N.A.
Coriander (seed)	*Coriandrum sativum*	Apiaceae	357	N.A.
Dill	*Anethum graveolens*	Apiaceae	1250	208
Fennel (leaf)	*Foeniculum vulgare*	Apiaceae	3949[a]	284
Fennel (seed)	*Foeniculum vulgare*	Apiaceae	N.A.	N.A.
Fennel (bulb)	*Foeniculum vulgare*	Apiaceae	N.A.	N.A.
Marjoram	*Origanum majorana*	Lamiaceae	3846	854
Oregano	*Origanum vulgare*	Lamiaceae	6367	935
Parsley	*Petroselinum crispum*	Apiaceae	1584	89
Rosemary	*Rosmarinus officinalis*	Lamiaceae	2519	1082
Sage	*Salvia officinalis*	Lamiaceae	2920	185
Tarragon	*Artemisia dracunculus*	Asteraceae	N.A.	570
Thyme	*Thymus vulgaris*	Lamiaceae	1815	1173
Lavender	*Lavendula angustifolia*	Lamiaceae	75[b]	N.A.
Hyssop	*Hyssopus officinalis*	Lamiaceae	83[c]	N.A.
Summer Savory	*Satureja hortensis*	Lamiaceae	6500[d]	N.A.
Latin				
Annatto (seed)	*Bixa orellana*	Bixaceae	N.A.	N.A.
Coriander (leaf)	*Coriandrum sativum*	Apiaceae	2260	159
Hoja santa	*Piper auritum*	Piperaceae	N.A.	N.A.
Tilia	*Tilia platyphyllos*	Melvaceae	N.A.	N.A.
Asian				
Allspice	*Pimenta officinalis*	Myrtaceae	N.A.	N.A.
Cardamom	*Elettaria cardamomum*	Zingiberaceae	603	N.A.
Star anise	*Illicium verum*	Schisandraceae	1810	N.A.

*Unless otherwise noted, all values are from the Phenol-Explorer Database (Neveu et al., 2010).
GAE – gallic acid equivalents; N.A. - not available.
[a]Barros et al. (2009).
[b]Robu et al. (2012).
[c]Loizzo et al. (2008).
[d]Rodov et al. (2010).

Table 2 Summary of in vivo research on the effect of selected herbs and spices on diabetes

Extract	Details	Solvent	Model	Type	Period	Dose	Results	Reference
Coriander	Seed	EtOH	STZ Diabetic Rats	T	Single Dose	200–250 mg/kg/d, i.p.	↓serum glucose ↑β-cell activity	(Eidi et al., 2009)
	Seed	Water	STZ Diabetic Rats	T	21 days	250–500 mg/kg/d, orally	↓serum glucose ↑serum HDL	(Naquvi et al., 2012)
	Seed	None	HFD induced Obese Mice	P	12 wks	1%, 3% wt:wt in diet	↓plasma glucose ↓serum insulin ↓serum glucose post OGTT	(Patel et al., 2011)
	Seed	None	Humans TIIDM 40–60 yo	T	60 days	5 g/d, orally	↓blood glucose ↓blood LPO ↓blood protein oxidation ↑blood antioxidants	(Rajeshwari and Andallu, 2011)
Cumin	Seed	None	Alloxan Diabetic Rats	T	6 wks	250 mg/kg/d, orally	↓plasma glucose ↓plasma TG ↓glycosylated Hgb ↓tissue cholesterol	(Dhandapani et al., 2002)
	Seed	MeOH	STZ Diabetic Rats	T	4 wks	250 mg/kg/d, orally	↓serum glucose; ↑serum insulin; ↓glycosylated Hgb; ↑hepatic glycogen; ↑skeletal muscle glycogen	(Jagtap and Patil, 2010)
	Seed	EtOH	STZ Diabetic Rats	T	Single Dose	250 mg/kg, orally	↓serum glucose post OGTT	(Srivsatava et al., 2010)
					14 days	250 mg/kg/d, orally	↓serum glucose post OGTT ↓serum glucose ↑serum insulin	
Dill	Leaves Dried	EtOH	Dexamethasone Diabetic Rats	P	15 days	100 mg/kg/d, orally	↓serum glucose ↓serum insulin	(Panda, 2008)
Marjoram	Leaves Dried	MeOH	Nicotinamide +STZ Diabetic Rats	T	21 days	100 – 400 mg/kg/d, orally	↓serum glucose; ↓glycosylated Hgb; ↓serum glucose post OGTT; ↑hepatic glycogen	(Pimple et al., 2011)
Oregano	Leaves Dried	Water	STZ Diabetic Rats	T	14 days	20 mg/kg/d, orally	↓blood glucose	(Lemhadri et al., 2004)
Parsley	Leaves Dried	Water	STZ Diabetic Rats	T	42 days	2 g/kg/d, orally	↓blood glucose ↓hepatocyte damage	(Bolkent et al., 2004)
	Leaves Dried	Water	STZ Diabetic Rats	T	28 days	2 g/kg/d, orally	↓blood glucose ↓hepatic AGEs ↑hepatic GSH	(Ozsoy-Sacan et al., 2006)
	Leaves Dried	Water	STZ Diabetic Rats	T	28 days	2 g/kg/d, orally	↓blood glucose; ↓aorta LPO; ↓heart LPO; ↑aorta GSH; ↑heart GSH	(Sener et al., 2003)
	Leaves Dried	Water	STZ Diabetic Rats	T	28 days	2 g/kg/d, orally	↓blood glucose	(Ozcelik et al., 2001)
Rosemary	Leaves Fresh	Water	Alloxan Diabetic Mice	T	12 days	0.2 ml/d, i.p.	↓serum glucose ↑serum insulin	(Abu-Al-Basal, 2010)
	Leaves Dried	EtOH	Alloxan Diabetic Rabbits	T	8 days	50–200 mg/kg/d, orally	↓serum glucose	(Bakirel et al., 2008)
			Alloxan Diabetic Rabbits and Healthy Rats		Single Dose	50–200 mg/kg, orally	↓serum glucose	

(Continued on next page)

Table 2 Summary of in vivo research on the effect of selected herbs and spices on diabetes *(Continued)*

Extract	Details	Solvent	Model	Type	Period	Dose	Results	Reference
	Leaves Dried	EtOH	STZ Diabetic Mice	P	4 days	0.01%wt/vol in drinking water	↓serum glucose	(Koga et al., 2006)
			Healthy Mice	T	Single Dose	20 mg, orally	↓serum glucose post OGTT	
Sage	Leaves Dried	MeOH	STZ Diabetic Rats	T	Single Dose	100–500 mg/kg, i.p.	↓serum glucose	(Eidi et al., 2005)
			Healthy Rats				= serum insulin = serum glucose	
	Leaves Dried	EtOH	STZ Diabetic Rats	T	14 days	100–400 mg/kg/d, orally	↓serum glucose ↑serum insulin ↓serum TG ↓serum TC	(Eidi and Eidi, 2009)
			Healthy Rats				↓serum glucose	
	Leaves N.S.	EtOH, Water	STZ Diabetic Rats	T	6 days	430 mg/kg,/d i.p.	= serum glucose (both solvents)	(Hajzadeh et al., 2011)
	Leaves Freeze-dried	Water	Healthy Mice	T	14 days	2 g/150 ml infusion as drinking water	↓serum glucose = ipGTT	(Lima et al., 2007)
Tarragon	Leaves frozen	EtOH	Diabetic KK-Aγ Mice	T	7 days	500 mg/kg/d, orally	↓serum glucose ↓serum insulin	(Ribnicky et al., 2006)
			STZ Diabetic Mice	T	7 days	500 mg/kg/d, orally	↓serum glucose, ↓hepatic PEPCK mRNA	
			Healthy Mice	T	7 days	500 mg/kg/d, orally	= hepatic PEPCK mRNA = serum glucose = serum insulin	
	Leaves frozen	EtOH	HFD-Induced Obese Mice	T	Single Dose	500 mg/kg, orally	↓plasma glucose	(Ribnicky et al., 2009)
	Leaves frozen	EtOH	HFD-Induced Obese Mice	T	7 wks	500 mg/kg/d, orally	↓plasma glucose ↑nerve conduction ↑sensory neuropathy ↓PNS 12/15-lipoxygenase ↓PNS nitrated protein	(Watcho et al., 2010)
	Leaves frozen	EtOH	STZ Diabetic Mice	T	7 wks	500 mg/kg/d, orally	↑nerve conduction ↓PNS 12/15-lipoxygenase ↓PNS oxidative-nitrosative stress; = plasma glucose = sciatic nerve sorbitol pathway intermediates	(Watcho et al., 2011)
Thyme	Leaves Dried	MeOH	STZ Diabetic Rats	T	28 days	100 mg/kg/d, orally	= serum glucose ↓serum TC; ↓serum TG; ↓serum LDL; ↓serum VLDL; ↑serum HDL; ↑retinal GSH; ↑lens GSH; ↑erythrocytes GSH; ↓erythrocyte MDA; ↑erythrocyte CAT; = plasma MDA; = plasma GSH	(Ozkol et al., 2012)

= no change, AGEs – advanced glycation end products; CAT – catalase; EtOH – ethanol; GSH – glutathione; Hgb – hemoglobin; HDL – high density lipoprotein; HFD – high fat diet; i.p. – intraperitoneal; ipGTT – intraperitoneal glucose tolerance test; LDL – low density lipoprotein; MeOH - methanol; MDA – malondialdehyde; OGTT – oral glucose tolerance test; P – preventions; PEPCK - phosphoenolpyruvate carboxykinase; PNS – peripheral nervous system; STZ - Streptozotocin; T- treatment; TC – total cholesterol; TG – triglycerides; TIIDM – Type II Diabetes Mellitus; VLDL – very low density lipoprotein.

Table 3 Summary of in vivo research on the effect of selected herbs and spices on hyperlipidemia

Extract	Details	Solvent	Model	Type	Period	Dose	Results	Reference
Basil	Leaves, Dried	Water	Triton WR-1339 Hyperlipidemic Rats	P	Single Dose	5 g/kg, orally	↓plasma TC ↓plasma LDL ↓plasma TG	(Amrani et al., 2006)
	Leaves, Dried	Ethyl Acetate, MeOH, Water	Triton WR-1339 Hyperlipidemic Mice	P	Single Dose	2 g/kg, orally	↓plasma TC* ↓plasma LDL* ↓plasma TG*	(Harnafi et al., 2008)
Bay	Leaves, N.S.	None	High Cholesterol Diet Fed Zebrafish	P	5 wks	10% wt/wt, orally	↓plasma TC ↓plasma TG ↓plasma glucose ↓plasma activity CETP	(Jin et al., 2011)
	Leaves, N.S.	None	Humans with TIIDM, > 40 years old	T	30 days	1–3 g/d, orally	↓serum glucose[a] ↓serum TC[a] ↓serum LDL[a] ↓serum TG[a]	(Khan et al., 2009)
	Leaves, N.S.	None	Humans with TIDM, > 40 years old	T	4 wks	3 g/d, orally	↓serum glucose[b] ↓serum TC[b] ↓serum LDL[b] ↓serum TG[b] ↑serum HDL[b]	(Aljamal, 2010)
Coriander	Seed	Water	Hypercaloric Diet induced Obese Rats	T	30 days	20 mg/kg/d, orally	↓plasma TC ↓plasma LDL ↓plasma TG	(Aissaoui et al., 2011)
	Seed	None	HFD-induced Obese Rats	P	75 days	10% wt/wt/d, in diet	↓tissue cholesterol ↓tissue TG ↓hepatic HMG-CoA ↑reductase activity ↑plasma LCAT ↓serum LDL ↓serum VLDL ↑serum HDL ↑hepatic & fecal BA ↑hepatic & fecal sterols	(Dhanapakiam et al., 2008)
	Seed	MeOH	High Cholesterol Diet Fed Rabbits	P	120 days	500 mg/kg/d, orally	↓serum TG ↓serum LDL ↓serum VLDL ↓serum TC ↓excreted TC ↓aorta cholesterol	(Joshi et al., 2012)
Dill	Leaves, Dried	Fractions	HFD-induced Obese Rats	T	2 wks	50 mg/kg/d, orally	↓plasma TC* ↓plasma TG* ↓plasma LDL*	(Bahramikia and Yazdanparast, 2009)
	N.S. N.S.	None	Hyperlipidemic Humans, 31–78 years old	T	6 wks	1.3 g/d, orally	= plasma TG = plasma TC = plasma LDL	(Kojuri et al., 2007)
Fennel	Bulb, Dried	Water	Triton WR-1339 Hyperlipidemic Mice	P	Single Dose	200 mg/kg, orally	↓plasma TC; ↓plasma TG; ↓plasma LDL; ↓plasma ApoB; ↑plasma HDL; ↑plasma ApoA1; ↓coronary artery lipids; ↓hepatic TC; ↓hepatic TG	(Oulmouden et al., 2011)
	Seed	MeOH	Healthy Rats	T	21 days	200 mg/kg/d, orally Oral	= plasma TG = plasma TC = plasma LDL ↑plasma HDL	(Choi and Hwang, 2004)
Oregano	Leaves, N.S.	Water	Non-Smoking Men	T	4 wks	300, 600 mg GAE/d, orally	↑urinary phenolic acids = serum lipid profile = plasma homocysteine	(Nurmi et al., 2006)

(Continued on next page)

Table 3 Summary of in vivo research on the effect of selected herbs and spices on hyperlipidemia *(Continued)*

Extract	Details	Solvent	Model	Type	Period	Dose	Results	Reference
Rosemary	Leaves, N.S.	EtOH	Lean and Zucker Rats	T	64 days	0.5% wt/wt, in diet	= serum TG‡ = serum TC‡ ↓Stomach HSL activity‡ ↓SI HSL activity‡ ↓serum TG† ↓serum TC† ↓stomach HSL activity†	(Romo Vaquero et al., 2012)
	Leaves, N.S.	N.S.	HFD-induced Obese Rats		16 weeks	500 mg/kg/d in diet	↓body weight ↑fecal lipids ↓plasma glucose ↓plasma TC = plasma TG = Plasma NEFA	(Ibarra et al., 2011)
Sage	Leaves, Dried	EtOH	Hyperlipidemic Humans, 20–60 years old	T	2 mos	500 mg/8 hs, orally	↓serum TC[a b] ↓ serum TG[a b] ↓ serum LDL[a b] ↓ serum VLD[a b] ↑ serum HDL[a b]	(Kianbakht et al., 2011)
	Leaves, Lyophilized	Water	Healthy Humans, 40–50 years old	T	2 wks	4 g in 300 ml water/2x day, orally	↓plasma LDL ↓plasma TC ↑plasma HDL ↑RBC SOD activity ↑RBC CAT activity = plasma glucose = post-prandial glucose = blood pressure	(Sa et al., 2009)
Thyme	Leaves, Dried	Water	Triton WR-1339 Hyperlipidemic Rats	P	Single Dose	2 g/kg, orally	↑plasma TG = plasma TC = plasma LDL = plasma HDL	(Ramchoun et al., 2012)

[a]compared to placebo [b]compared to baseline †compared to lean group ‡compared to obese group *All solvents/fractions
= no change, BA – bile acids; CAT – catalase; CETP – cholesterol ester transfer protein; EtOH – ethanol; HFD – high fat diet; HDL – high density lipoprotein; HMG CoA - 3-hydroxy-3-methylglutaryl-coenzyme A; i.p. – intraperitoneal; LCAT – lecithin-cholesterol acyltransferase; LDL – low density lipoprotein; MeOH – methanol; NEFA – non-esterified fatty acids; N.S. – not specified; P – prevention; RBC – red blood cell; SI – small intestines; SOD – superoxide dismutase; T- treatment; TC – total cholesterol; TG – triglycerides; TIIDM – Type II Diabetes Mellitus; VLDL – very low density lipoprotein.

HERBS AND SPICES

Basil (Ocimum basilicum)

Ocimum basilicum is found in cuisines from all over the world, but it is a staple in the Mediterranean diet (Hill, 2004). Basil leaves are used in salads, tomato based dishes and in pasta sauces, possibly the most notably in Italian "pesto" (Bown, 2001). It is a component of the traditional French collection of herbs referred to as "*herbes de Provence*." Extract from basil contains significant levels of rosmarinic acid and catechin (Shan et al., 2005). Research on the health benefits of extract from basil suggest that basil may protect against or treat several components of the metabolic syndrome.

Studies indicate that extract from basil can inhibit α-glucosidase (Cazzola et al., 2011; El-Beshbishy and Bahashwan, 2012; Koga et al., 2006) and α-amylase (Cazzola et al., 2011; El-Beshbishy and Bahashwan, 2012) and may thus alleviate hyperglycemia associated with diabetes. Basil extract has also been shown to inhibit the activity of aldose reductase (Saraswat et al., 2008) which may then prevent intracellular sorbitol accumulation that leads to osmotic and oxidative stress on vasculature that is associated with diabetic complications (Brownlee, 2001). The anti-diabetic effects of basil extract may be due in part to its rosmarinic acid content, since rosmarinic acid has been found to inhibit α-glucosidase, α-amylase and aldose reductase in vitro (McCue et al., 2004; Ha et al., 2012; Kwon et al., 2006). Basil also contains catechin that can inhibit both α-glucosidase and α-amylase (Yilmazer-Musa et al., 2012). Basil extract has also the potential to inhibit albumin glycation (Cazzola et al., 2011). Advanced glycation end products (AGEs) are a consequence of oxidative stress (Giardino et al., 1996) and polyphenols are potent antioxidants (Stankevicius et al., 2011), thus the anti-AGE effect of basil may be associated with its relatively high polyphenol content.

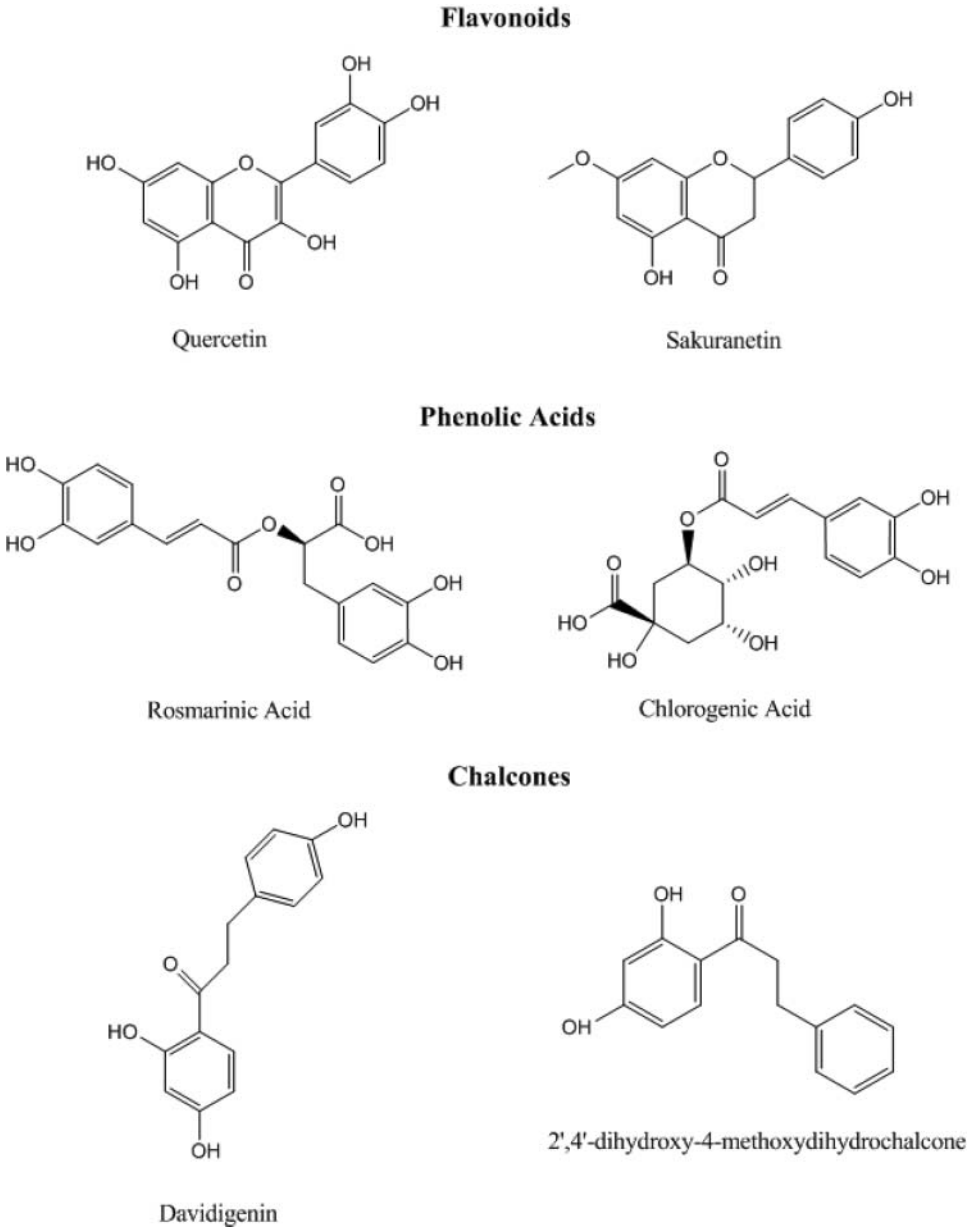

Figure 1 Representative Bioactive Compounds. A. The flavonoids quercetin (bay leaf, dill, fennel, oregano) and sakuranetin (tarragon); B. the phenolic acids rosmarinic acid (basil, marjoram, oregano, rosemary, sage, thyme) and chlorogenic acid (cumin); C. and the chalcones davidigenin (tarragon) and 2′,4′-dihydroxy-4-methoxydihydrochalcone (tarragon) represent three major classes of bioactive compounds found in herbs and spices.

In addition to being anti-diabetic, basil may also be anti-inflammatory. When lipopolysaccharide (LPS)-stimulated blood mononuclear cells were incubated with basil extract, there was reduced transcription of the proinflammatory cytokines interleukin (IL)-2, IL-1β and tumor necrosis factor-α (TNF-α) compared to the LPS-stimulated control (Selvakkumar et al., 2007). LPS-stimulated macrophages also released less IL-6 into the extracellular environment, and more IL-10 (Mueller et al., 2010). Basil extract can also inhibit inducible nitric oxide synthase (iNOS) transcription and protein expression (Selvakkumar et al., 2007; Tsai et al., 2007; Mueller et al., 2010) but does not exhibit nitric oxide (NO) scavenging activity (Tsai et al., 2007). Together, these studies suggest that extract of basil may inhibit nuclear factor-κB (NFκB) binding activity, since genes for many of these cytokines are downstream of this transcription factor (Pahl, 1999). However, the work by (Paur et al., 2008) indicates that treatment of LPS-stimulated monocytes with basil extract had no effect of NFκB binding activity. It may be possible that extract-mediated upregulation of IL-10 is responsible for the decreased inflammatory response, since LPS-stimulated macrophages treated with IL-10 inhibits expression of TNF-α and IL-6 (Fiorentino et al., 1991).

Increased basil consumption may also protect against or treat hyperlipidemia. Hyperlipidemic rodents intragastrically

provided basil extract had lower total cholesterol, LDL and triglycerides compared to rodents without the extract treatment (Amrani et al., 2006; Harnafi et al., 2008). Additionally, macrophages incubated with LDL and basil extract accumulated less lipids and cholesterol esters and synthesized less unesterified cholesterol compared to cells incubated with LDL alone (Bravo et al., 2008). Lower cholesterol and reduced macrophage uptake of lipids may prevent or reduce plaque formation and perhaps protect against atherosclerosis.

Basil may also have favorable effects on blood pressure. Oral administration of basil extract to hypertensive rats reduced blood pressure, reduced circulating endothelin and prevented cardiac hypertrophy more effectively than the positive control drug, captopril. Basil extract may also improve renal function in hypertensive rats, since animals in the basil treatment group also had lower blood urea nitrogen concentration, creatinine and angiotensin II compared to the hypertensive control (Umar et al., 2010).

Bay Leaf (Laurus nobilis)

Laurus nobilis is common to southern Europe and is often used in cooking both sweet and savory dishes in European cuisine (Bown, 2001). Bay leaves are also a component of the French collection of herbs referred to as "*bouquet garni*" (Hill, 2004). Extract of bay leaves contains various classes of flavonoids (Shan et al., 2005) notably quercetin and kaempferol (Agricultural Research Service, 2011) as well as various sesquiterpenes (Matsuda et al., 2000).

Bay leaf has been shown to exhibit a wide array of anti-diabetic effects. In vitro, bay leaf extract was shown to be a PPAR-γ antagonist, binding to PPAR-γ without recruiting coactivator proteins (Mueller and Jungbauer, 2009). Bay leaf extract also inhibited protein glycation (Dearlove et al., 2008) and α-glucosidase activity in vitro (Koga et al., 2006). Bay leaf extract also increased insulin-like glucose uptake by rat epididymal adipocytes (Broadhurst et al., 2000). This ability was lost when polyphenols were removed from the extract, suggesting that the insulin-like activity of bay leaf extract is dependent on its polyphenol content.

Bay leaf also has anti-inflammatory potential. Extract from bay leaf almost completely inhibited IL-6 production and reduced cyclooxygenase (COX)-2 protein expression in LPS stimulated macrophages (Mueller et al., 2010). Additionally, extract from bay leaf prevented NO release in LPS stimulate macrophages (Matsuda et al., 2000). Sesquiterpene constituents of these extracts may be responsible for the anti-inflammatory effect of bay leaf. Incubation of LPS-stimulated macrophages with costunolide dose-dependently inhibited iNOS induction while dose-dependently increasing HSP-72 expression (Matsuda et al., 2000). Induction of HSP-72 prevents nuclear translocation of the transcription factor NKκB (Gabai et al., 1998), thus bay leaf sesquiterpenes may protect against inflammation by indirectly inhibiting NKκB activity.

Several in vivo studies support the fact that bay leaf may also be hypolipidemic. Zebrafish consuming bay leaf extract in addition to a hypercholesterolemic diet had significantly lower plasma total cholesterol, triglycerides and glucose compared to the fish fed the hypercholesterolemic diet alone (Jin et al., 2011). Bay leaf also reduced plasma activity of cholesterol ester transfer protein in the treated zebrafish. Consistent with these observations, the extract treatment also inhibited the transfer of cholesteryl oleate from high-density lipoprotein (HDL) particles to LDL particles. In humans, males and females over 40 years old with controlled Type II diabetes consumed 1, 2 or 3 g of ground bay leaves per day for 30 days. At the end of the study period, subjects had reduced fasting serum glucose, total cholesterol, LDL and triglycerides compared to the placebo group (Khan et al., 2009). Similarly, males and females over 40 years old with controlled type I diabetes consuming 3 g of bay leaf per day for 4 weeks had reduced serum total cholesterol, LDL, triglycerides and fasting glucose and had greater serum HDL compared to baseline (Aljamal, 2010). In vitro, bay leaf inhibited ApoA1 glycation, oxidation of LDL particles and macrophage uptake of oxidized LDL particles (Jin et al., 2011). Taken together, consumption of bay leaf may help manage hyperlipidemia.

Coriander (Coriandrum sativum)

Both coriander leaves and seeds are edible and used in cuisine. The coriander plant is native to eastern Mediterranean and central Asia and has naturalized to North America (Bown, 2001). Coriander seeds are added to oil and lemon to form a dressing used in French cuisine and are also used in pickling and to flavor sausages (Bown, 2001). Coriander leaves, also known as cilantro are used in bean dishes, stews and salsas (Hill, 2004). The seeds are high in caffeoyl derivatives and p-coumaric acid while the leaves are high in quercetin-3-*O*-rutinoside (Barros et al., 2012). Below is a summary of the studies performed on coriander seed. Studies on coriander leaves are summarized in Table 4.

In streptozotocin-induced diabetic rats, intraperitoneal injection of coriander seed extract significantly reduced serum glucose and increased insulin secretion by pancreatic β-cells compared to diabetic controls (Eidi et al., 2009). Oral administration of coriander seed extract also reduced serum glucose and total cholesterol and increased HDL in diabetic rats (Naquvi et al., 2012). In high-fat diet induced obese rats, coriander seed extract prevented diet-induced increases in blood glucose and plasma insulin. The extract treatment also improved intraperitoneal glucose tolerance test results. Results in this study were comparable to the effect of rosiglitazone (Patel et al., 2011). Similar results were observed in humans. Humans between 40 and 60 years old with type II diabetes were provided 5 g of

Table 4 Summary of research on health benefits of other selected herbs and spices

Plant	Details	Solvent	Model	Type	Period	Dose	Results	Reference
Hyssop	Dried, Leaves	Multiple	In vitro	—	—	—	↓α-glucosidase activity (chloroform) ↓ACE activity (hexane)	(Loizzo et al., 2008)
	Dried, Leaves	MeOH	Healthy Mice	P	Single Dose	100, 300 mg/kg, orally	↓blood glucose post OGTT	(Miyazaki et al., 2003)
Lavender	Dried, Flowers	Water	Healthy Wistar Rats	T	Single Dose	3% body weight of lavender infusion, orally	↓urinary osmolarity ↑plasma osmolarity ↑serum sodium ↑water clearance = GFR = osmotic clearance = serum aldosterone = urinary sodium	(Elhajili et al., 2001)
	Dried, Flowers	DMSO	In vitro	—	—	—	No effect on PPAR-γ	(Mueller and Jungbauer, 2009)
Savory	Dried, Leaves	Water	Healthy Rats	T	3 wks	100 mg/kg, orally	↓blood glucose	(Kemertelidze et al., 2004)
			Alloxan Diabetic Rats	T	7 days		↓blood glucose	
			Healthy rats	P	Single Dose	100 mg/kg, orally	↓blood glucose post ipGTT	
Annatto	Seeds	Water	Triton WR1339, Fructose or EtOH Hyperlipidemic Mice	P	3x over 48 hrs	400, 800 mg/kg, N.S.	↓serum TG (all models)	(Ferreira et al., 2013)
	Seeds	N.S.	Neutrophils from Alloxan Diabetic Rats	T	7 days	1% extract in diet	↓ROS production ↓NADPH-Ox mRNA ↑SOD, CAT mRNA	(Rossoni Junior et al., 2012)
	Seed,	Series	Healthy dogs given OGTT one hour post treatment	T	Single Dose	80 mg/ kg, orally	↓plasma glucose ↑plasma insulin ↑insulin receptor binding in isolated mononuclear leukocytes and RBCs	(Russell et al., 2005)
	Arial, dried	EtOH	In vitro	—	—	—	↓AGE formation	(Gutierrez et al., 2010)
Coriander	Leaves	None	Alloxan Diabetic Rats	T	15 days	60 g/kg/d, powdered in diet	= histopathology of pancreas = blood glucose	(Jelodar et al., 2007)
	Arial, fresh	EtOH	Alloxan Diabetic Rats	T	7 days	100–750 mg/kg/d, orally	↓blood glucose levels ↓serum TC, TG, LDL ↑serum HDL	(Sreelatha and Inbavalli, 2012)
	Leaves, dried	EtOH	In vitro	—	—	—	= α-amylase activity	(Narkhede, 2012)
	Arial, fresh	EtOH	LPS-stimulated RAW 264.7 macrophages	—	—	—	↓NO, PGE2 expression ↓iNOS, COX-2, pro-IL-1β mRNA ↓nuclear p65 protein ↓NF-κB nuclear activity ↓phosphorylated MAPK protein	(Wu et al., 2010)
Hoja Santa	Leaves, dried	Series	In vitro	—	—	—	↓glycosylated protein, HgB ↓LDL oxidation, glycation	(Perez Gutierrez et al., 2012)
			STZ Diabetic Rats	T	28 days	200, 400 mg/kg/d	↓renal glucose, AGEs ↓TBARS (liver, kidney, pancreas) ↑CAT, SOD, GSH, GPx (liver, kidney, pancreas)	
	Arial, dried	EtOH	In vitro	—	—	—	↓AGE formation	(Gutierrez et al., 2010)

(Continued on next page)

Table 4 Summary of research on health benefits of other selected herbs and spices *(Continued)*

Plant	Details	Solvent	Model	Type	Period	Dose	Results	Reference
Tilia	Flower, dried	MeOH	In vitro	—	—	—	↓LPL activity	(Slanc et al., 2009)
Allspice	Seeds	NH_4OH	Rat epididymal adipocytes	—	—	—	↓insulin dependent glucose metabolism	(Broadhurst et al., 2000)
Cardamom	Seeds	None	Humans with Stage 1 Hypertension	T	12 wks	3 g/d	↓blood pressure ↑blood fibrinolytic activity ↑blood antioxidant status = blood fibrinogen = serum lipids	(Verma et al., 2009)
	Seeds	Water	LPS-stimulated mouse peritoneal macrophages	—	—	—	↓NO production	(Majdalawieh and Carr, 2010)
Star Anise	Seeds	EtOH	TNF-α/IFN-γ-stimulated human keratinocytes	—	—	—	↓IL-6, IL-1β mRNA and protein ↓I-CAM mRNA ↓NFκB nuclear activity	(Sung et al., 2012)

= no change; ACE – angiotensin converting enzyme; AGE – advanced glycation end products; CAT – catalase; COX-2 – cyclooxygenase 2; EtOH – ethanol; GFR – glomerular filtration rate; GPx – glutathione peroxidase; GSH – glutathione; HDL – high density lipoprotein; HgB – hemoglobin; IFN-γ – interferon-γ; IL – interleukin; ICAM – intercellular adhesion molecule; iNOS – inducible nitric oxide synthase; ipGTT – intraperitoneal glucose tolerance test; LDL – low density lipoprotein; LPL – lipoprotein lipase; LPS- lipopolysaccharide; MAPK – mitogen-activated protein kinase; MeOH – methanol; NADPH-ox – nicotinamide adenine dinucleotide phosphate-oxidase; NF-κB – nuclear factor κB; NO – nitric oxide; N.S. – not specified; OGTT – oral glucose tolerance test; P – prevention; PGE2 – prostaglandin E2; PPAR-γ - peroxisome proliferator-activated receptor-γ; RBC – red blood cell; ROS – reactive oxygen species; SOD – superoxide dismutase; STZ – streptozotocin; T – treatment; TBARS - thiobarbituric acid reactive substances; TC – total cholesterol; TG – triglycerides; TNF-α – tumor necrosis factor-α.

ground coriander seed per day for 60 days. The treatment group had lower fasting blood glucose, blood lipid peroxidation and blood protein oxidation compared to their initial measurements. They also had increased serum and erythrocyte antioxidant levels (Rajeshwari and Andallu, 2011).

Coriander seeds may also be hypolipidemic. Obese rats with hyperlipidemia administered a coriander seed extract orally for 30 days had reduced total cholesterol, LDL, and triglycerides (Aissaoui et al., 2011). When fed coriander seed extract in addition to a high-fat diet for 75 days, rats had lower tissue cholesterol and tissue triglycerides. They also had decreased hepatic HMG-CoA reductase activity and plasma lecithin cholesterol acyl-transferase activity. LDL and very low-density lipoprotein (VLDL) were decreased and HDL was increased as well. There were also increased hepatic and fecal bile acids and neutral sterols (Dhanapakiam et al., 2008). Similar results were observed in rabbits and was accompanied by decreased cholesterol deposition in the aorta (Joshi et al., 2012). This suggests that coriander seed extract has the potential to mitigate atherosclerosis development by reducing circulating cholesterol, cholesterol synthesis and facilitating cholesterol excretion.

Coriander seeds can also facilitate diuresis. When administered coriander seed extract continuously through IV, healthy rats had increase urine production, electrolyte secretion and glomerular filtration rate (Aissaoui et al., 2008). When administered coriander seeds intraperitoneally, healthy rats experienced reduced blood pressure (Jabeen et al., 2009).

Cumin (Cuminum cyminum)

Cumin can be found growing wild from the Mediterranean region through Europe and is widely used in many cuisines from around the world (Bown, 2001; Hill, 2004). Seeds are often ground and used in herb rubs for meat dishes or are used to flavor sauces, pickles and breads (Bown, 2001). Cumin seeds contain phenolic acids including chlorogenic acid and flavonoids, particularly apigenin (Pandey et al., 2012).

Cumin seeds have anti-diabetic effects in vivo. Cumin extract or ground cumin administered orally both reduced blood glucose and glycosylated hemoglobin in diabetic rats (Dhandapani et al., 2002; Jagtap and Patil, 2010). Ground cumin seeds also reduced triglycerides and total cholesterol in plasma, liver, kidney, intestines and pancreas of the diabetic animals (Dhandapani et al., 2002). It also improved serum insulin and glycogen content in the liver and skeletal muscle of diabetic rats (Jagtap and Patil, 2010). Additionally, single and repeated dosing of cumin extract improved oral glucose tolerance in diabetic rats (Srivsatava et al., 2010). Cumin may also protect eye health in diabetic animals, as it has been shown to inhibit aldose reductase activity in vitro (Saraswat et al., 2008) and in the lens of diabetic and healthy rats (Srivsatava et al., 2010). In vitro, cumin seed extracted with 95% ethanol has an inhibitory effect on α-glucosidase (Srivsatava et al., 2010) whereas cumin seed extracted in 50% ethanol did not (Koga et al., 2006). Together, these studies suggest that cumin can mitigate both the direct effects and the secondary consequences of diabetes.

Dill (Anethum graveolens)

Anethum graveolens originated in the southwestern Asia but has naturalized in the Mediterranean and in parts of North America. It is often consumed with seafood and is used to flavor vinegars and in pickling (Bown, 2001). Fresh dill is especially high in quercetin and isorhamnetin (Neveu et al., 2010).

Extract from dill may have anti-diabetic potential. Rats with dexamethasone induced diabetes orally administered dill extract for 15 days had reduced serum glucose and insulin (Panda, 2008). This improvement of the diabetic condition may be due in part to the antagonistic effect of dill on PPAR-γ (Mueller and Jungbauer, 2009). Dill may also reduce carbohydrate influx, since it has been shown to inhibit α-glucosidase activity (Koga et al., 2006).

Dill may also be hypolipidemic. Rats fed a high-fat diet for 3 weeks to induce hyperlipidemia were given dill extract orally every day for the following 2 weeks. Animals fed dill extract had lower circulating total cholesterol, triglycerides and LDL compared to animals eating the high-fat diet for 5 weeks (Bahramikia and Yazdanparast, 2009). In humans, the results are less conclusive. Hyperlipidemic subjects aged 31-78 years old that consumed 1.3 g of dill daily for 6 weeks had no significant reduction or elevation in circulating triglycerides, total cholesterol and LDL (Kojuri et al., 2007). This may be a result of dosage, however, since rats were provided with more dill than the humans on a per-kilogram basis. Additionally, rats were provided basil fractions while humans consumed tablets containing whole dill.

Fennel (Foeniculum Vulgare)

Varieties of fennel are found throughout northern and central Europe. The bulb, leaves, flowers and seeds are all considered edible and occur in many dishes consumed in Mediterranean diet. Bulbs are used raw in salads, while seeds are used to flavor sausages and breads and leaves are cooked with fish or used to make herbal teas (Bown, 2001). The most prevalent flavonoid in polar extract of fennel leaves is quercetin (Agricultural Research Service, 2011).

Regarding the potential benefit of fennel consumption on complications associated with diabetes, extract from the edible portion of fennel has been shown to inhibit aldose reductase, and it does so with a high specificity over aldehyde reductase (Saraswat et al., 2008). Thus, fennel may be able to prevent diabetic complications associated with induction of the polyol pathway without inhibiting aldehyde reductase, a detoxification enzyme in the same family.

Additionally, consumption of fennel seed extract may be anti-inflammatory. Oral administration of fennel seed extract prevented carrageenan-induced paw edema and arachidonic acid-induced ear edema in mice. Extract of fennel leaves also decreased thermal nociception in mice, suggesting an analgesic effect of fennel (Choi and Hwang, 2004).

Fennel bulb but not fennel seed may be antihyperlipidemic. Fennel bulb extract administered total cholesterol, triglycerides, LDL and ApoB in mice after 24 hours. Fennel extract also increased plasma HDL and ApoA1 to levels higher than the hyperlipidemic control. In addition to managing hyperlipidemia, fennel bulb extract prevented lipid accumulation in the coronary artery and decreased hepatic total cholesterol and triglycerides compared to the hyperlipidemic control (Oulmouden et al., 2011). However, when healthy rats were given fennel seed extract orally for 21 days, they experienced no change in blood triglycerides, total cholesterol and LDL. Healthy rats did, however, have greater circulating HDL (Choi and Hwang, 2004).

Marjoram (Origanum majorana)

Origanum majorana is native to southern Europe and northern Africa and leaves are commonly used to flavor Mediterranean dishes near the end of the cooking process or used in flavoring vinegars and oils (Bown, 2001). It is also a component of the herb combinations "herbes de Provence", "fines herbes" and "bouquet garni" used in France (Hill, 2004). Phenolic acids found in marjoram include trans-2-hydroxycinnaminic acid and rosmarinic acid (Neveu et al., 2010) and contains the biflavone amentoflavone as is its most prevalent flavonoid (Baatour et al., 2013).

Experiments on marjoram extract in vitro indicate that components of this plant have anti-diabetic potential and these results are corroborated in vivo. Diabetic rats given marjoram extract orally had lower fasting glucose, lower glycated hemoglobin, improved oral glucose tolerance test results and greater hepatic glycogen compared to the diabetic control (Pimple et al., 2011). In vitro, marjoram extract prevented hemoglobin, albumin and LDL glycation (Dearlove et al., 2008) (Perez Gutierrez, 2012) as well as inhibited α-glucosidase activity and antagonized PPAR-γ (Koga et al., 2006; Mueller et al., 2008; Pimple et al., 2011) indicating it may have wide-spread mechanisms of action. Marjoram extract may also protect against diabetic nephropathy; rats with streptozotocin induced diabetes were provided marjoram extract orally and had lower renal mitochondrial lipid peroxidation, decreased renal weight and reduced renal AGEs compared to the control (Perez Gutierrez, 2012).

Marjoram extract may also have anti-inflammatory activity. When treated with marjoram extract, LPS stimulated macrophages expressed less iNOS and prevented NO production (Mueller et al., 2010; Tsai et al., 2007). Marjoram extract also exhibited NO radical scavenging activity (Tsai et al., 2007). However, in contrast to the results seen from experiments on other herbs, marjoram extract did not significantly affect production of IL-6, TNF-α or IL-10 and did not affect protein expression of COX-2 in LPS-stimulated macrophages (Mueller et al., 2010).

Oregano (Origanum vulgare)

Origanum vulgare is native to Europe and has a large number of varieties and subspecies. Culinary oregano is often dried

and used in dishes that also contain onions, tomatoes or garlic. Leaves and flowering tops of oregano are also used in tea (Bown, 2001). Oregano is an ingredient in the French collection of herbs referred to as "*bouquet garni*" (Hill, 2004). Other culinary origanum species include *Origanum onites*, *Origanum heracleoticum* and *Origanum majorana* (Hill, 2004). *Oreganum vulgare* is particularly high in polyphenols, particularly rosmarinic acid (Neveu et al., 2010).

Oregano extract exhibits a wide range of anti-diabetic properties. Oregano extract inhibits α-glucosidase (Kwon et al., 2006) aldose reductase (Koukoulitsa et al., 2006) and prevents albumin glycation (Dearlove et al., 2008) in vitro. Additionally, extracts from cultured shoot tissue of various clonal lines of oregano inhibited pancreatic amylase activity by between 9 and 57%. This inhibition by oregano extract was positively correlated with the bioactive components rosmarinic acid, quercetin, protochatechuic acid and p-couramic acid (McCue et al., 2004). Oregano extract has been shown to antagonize PPAR-γ and increase insulin-stimulated glucose uptake by adipocytes without stimulating adipocyte differentiation (Mueller et al., 2008; Christensen et al., 2009). This suggests a potential role for oregano extract to help manage diabetes. (Mueller et al., 2008) tested the antagonistic potential of oregano varieties from various regions and found that *Origanum onites*, Turkish oregano, also exhibited this property.

The in vitro anti-diabetic activities of oregano may also occur in vivo. When rats were administered streptozotocin to induce diabetes and subsequently provided with extract of oregano by gavage for two weeks, they experienced reduced serum glucose just 4 h after the first treatment. The maximal reduction in glucose was observed after four days and lower blood glucose was maintained for the entire two weeks. Interestingly, during the study period, there was no variation in plasma insulin levels (Lemhadri et al., 2004) which suggests that the anti-diabetic activity of oregano extract is independent of the action of the pancreas.

Research has also been completed regarding the potential hypolipidemic effects of consuming oregano extract. Forty-five non-smoking men were provided with a juice beverage containing 300 mg GAE of oregano extract or 600 mg GAE oregano extract for 4 weeks. Subjects provided with the 300 mg GAE had 2% higher urinary phenolic acid concentration after 24 h whereas the subjects provided with 600 mg GAE had 85% higher urinary phenolic acid concentration. However, there was no change in serum lipid profile or plasma homocysteine after 4 wks. However, after 90 min there was a significant reduction in LDL oxidation in the 300 mg GAE group which was not present after 4 weeks (Nurmi et al., 2006).

In vitro, oregano extract has been shown to alleviate nitrative stress. Extract from *Origanum vulgare* dose dependently increased endothelial nitric oxide synthase (eNOS) activity in umbilical vein endothelial cells (Mueller et al., 2008) and inhibited iNOS protein expression and NO production in LPS-activated macrophages (Tsai et al., 2007) suggesting that oregano consumption may still provide vascular benefits despite the results of the human study summarized above. Experiments on *Origanum onites* conveyed similar results (Mueller et al., 2010).

Oregano has also been shown to decrease markers of inflammation in vitro. LPS-stimulated macrophages produced less IL-6 and TNF-α when incubated with extract from *Origanum onites* compared to the LPS-stimulated control. *Origanum vulgare* extract decreased basal NFκB activity in human monocytes and strongly inhibited NFκB activity to just 9% of the LPS-stimulated monocytes and TNF-α -stimulated monocytes (Paur et al., 2008). *Origanum vulgare* has also been shown to inhibit soybean lipoxygenase activity (Koukoulitsa et al., 2006).

Parsley (Petroselinum crispum)

There are two common varieties of *Petroselinum crispum* which are native to southeastern Europe. There is curly leaf parsley (syn. *P. crispum* var. *crispum*) and flat leaf parsley (*P. crispum* var. *neapolitanum)* and flat leaf parsley is said to have a stronger flavor (Bown, 2001). Parsley leaves are often used as a garnish or to flavor sauces and savory dishes. It is also sautéed with butter and other herbs to make traditional Italian *gremolata* or French *persillade* (Bown, 2001) and is a component of the herb blends "*fines herbes*" and "*bouquet garni*." Extract from parsley is high in the flavonoid apigenin (Agricultural Research Service, 2011).

Parsley extract has been shown to reverse hyperglycemia and protein glycation associated with diabetes in several organ systems. Parsley extract orally administered to streptozotocin-induced diabetic rats lowered circulating glucose after 28 days (Ozcelik et al., 2001; Sener et al., 2003; Bolkent et al., 2004). Rats in this model system also had less histological evidence of hepatocyte damage (Bolkent et al., 2004), greater hepatic glutathione (GSH) and lower hepatic AGEs but extract treatment did not affect hepatic lipid peroxidation (Ozsoy-Sacan et al., 2006). In other organs, diabetic rats had improved aorta and heart lipid peroxidation in addition to increased aorta and heart GSH (Sener et al., 2003). The effect of parsley was also studied in the eyes of diabetic rats, however oral administration of parsley extract did not affect lens protein glycation or lens GSH (Ozcelik et al., 2001). The mechanism for the anti-diabetic action of parsley extract may be inhibiting absorption of glucose by inhibiting α-glucosidase (Taguchi et al., 2010). In contrast to many other herbs, parsley extract did not inhibit PPAR-γ (Mueller and Jungbauer, 2009). This is interesting because herbs like thyme, sage, bay leaf, marjoram and rosemary are also high polyphenols yet have this antagonistic ability (Mueller and Jungbauer, 2009). Future investigation into the phenolic profile of these herbs may shed light upon which phenolic components of these herbs is responsible for PPAR-γ inhibition.

Parsley also has anti-inflammatory potential. In carrageenan-induced paw edema, rats given parsley extract orally one hour prior to carrageenan injection had less edema than the no-extract control (Al-Howiriny et al., 2003). Mice

provided parsley extract orally and concomitantly challenged with a cotton pellet inserted subcutaneously for four days had smaller granulomas compared to the animals not receiving the extract (Al-Howiriny et al., 2003).

Greater parsley consumption may also be beneficial in the treatment for hypertension. Healthy rats administered parsley extract orally had greater urinary volume and greater urinary excretion of sodium, potassium and chloride after 5 h compared to the no-extract control. Interestingly, the positive control furosemide had no effect on urinary potassium (Devi et al., 2010).

Rosemary (Rosmarinus officinalis)

Rosmarinus officinalis has a large number of varieties and is native to coastal Mediterranean areas. It is used to flavor meat dishes, soups and stews and is a component of the French collection of herbs referred to as "*bouquet garni*" (Bown, 2001; Hill, 2004). Leaves are also steeped in vinegar or oil to flavor sauces or steeped in water to make tea (Bown, 2001). Rosemary extract is high especially high in rosmarinic acid (Neveu et al., 2010).

Rosemary extract shows both subacute and acute anti-diabetic potential. Seven days after three consecutive days of intraperitoneal injection of rosemary extract, alloxan-induced diabetic mice had lower blood glucose compared to the diabetic control (Abu-Al-Basal, 2010). Similarly, eight days of oral rosemary extract administration to alloxan-induced diabetic rabbits produced lower fasting glucose and greater serum insulin (Bakirel et al., 2008). These effects were also observed just six hours after a single oral dose of rosemary extract (Bakirel et al., 2008). Similar effects on blood glucose were observed at the same dosage in healthy animal rabbits (Bakirel et al., 2008).

One mechanism for the hypoglycemic effect of rosemary extract may be its ability to inhibit enzymes involved in absorption and metabolism. Several studies report that rosemary extract inhibits α-glucosidase activity in vitro (Koga et al., 2006; Kwon et al., 2006; Cazzola et al., 2011) while there are conflicting results regarding the effect of rosemary on α-amylase (Kwon et al., 2006; Cazzola et al., 2011) which may be a result of differing methods of extraction. It is interesting to note that consuming rosemary extract in water for four days did not affect intestinal activities of maltase or sucrase in streptozotocin-induced diabetic rats (Koga et al., 2006). This may be a consequence of the difference in concentration achieved *in vivo* compared to the concentration used in vitro. Furthermore, extract from rosemary has been shown to be a PPAR-γ agonist and may be antidiabetic through that mechanism (Ibarra et al., 2011).

Rosemary extract may also be anti-inflammatory in addition to being anti-diabetic. In LPS-stimulated macrophages, rosemary extract reduced macrophage viability (Peng et al., 2007), inhibited iNOS protein expression (Mueller et al., 2010) and decreased NO production (Peng et al., 2007; Tsai et al., 2007) compared to the LPS control. Rosemary also exhibited NO scavenging activity (Tsai et al., 2007). Rosemary extract has also been shown to inhibit 15-lipoxygenase (Gawlik-Dziki, 2012) and COX-2 (Yi and Wetzstein, 2010) enzymes. Interestingly, rosemary extract did not affect NFκB binding activity in LPS induced monocytes (Paur et al., 2008) suggesting that rosemary extract exhibits its anti-inflammatory effect independent of potential NFκB -mediated upregulation of genes for COX-2 and iNOS.

Rosemary may also protect against hypertension. Healthy rats orally administered rosemary extract daily for seven days had increased urine volume and sodium, potassium and chloride excretion over the last 3 days of treatment with no change in plasma electrolytes or plasma urea (Haloui et al., 2000). Rosemary extract may be exhibiting this effect by inhibiting angiotensin converting enzyme, as (Kwon et al., 2006) showed that rosemary extract almost completely inhibited the activity of this enzyme in vitro.

Further contributing to the multitude of beneficial effects of rosemary, this herb may also be hypolipidemic. Lean or genetically obese Zucker rats were fed a diet containing ground rosemary and after 64 days, both the lean and obese rats had lower body weight compared to their respective control. Rosemary extract had no effect on serum triglycerides or cholesterol in the obese group; however, it did reduce these factors in lean animals. Obese rats consuming the diet containing rosemary extract had reduced lipase activity in stomach, duodenum, and ileum/jejunum compared to obese control, while lean rats only reduced lipase activity in the stomach (Romo Vaquero et al., 2012). In vitro, rosemary extract was shown to be a more potent inhibitor of hormone sensitive lipase than five of its purified constituents and inhibited pancreatic lipase (Bustanji et al., 2010; Ibarra et al., 2011). Thus, rosemary extract may exert a hypolipidemic effect by inhibiting lipase activity and preventing lipid absorption.

Sage (Salvia officinalis)

Salvia officinalis is native to the Mediterranean and is used to flavor pork, stuffings, stews and sausages and is a component of "*bouquet garni*". Dried and fresh sage leaves are steeped in water to make herbal tea (Bown, 2001). Sage is high in polyphenols, particularly the phenolic acid rosmarinic acid (Neveu et al., 2010).

Sage extract may reduce hyperglycemia associated with diabetes after acute or sub-acute treatment. Wistar rats with streptozotocin-induced diabetes were intraperitoneally administered sage extract and after three hours had experienced reduced serum glucose but no changes in serum insulin (Eidi et al., 2005). In a longer study of 14 days, diabetic rats orally administered sage extract also had reduced blood glucose in addition to increased serum insulin and reduced serum triglycerides and total cholesterol (Eidi and Eidi, 2009). The highest

dose of extract administered in this study was equally as effective as the anti-diabetic drug glibenclamide at reversing diabetic symptoms. Interestingly, when diabetic rats were given sage extract intraperitoneally for 6 days, animals did not experience a significant change in serum glucose (Hajzadeh et al., 2011). In both the six day and 14 day studies, sage leaves were extracted in polar solvents and the oral doses were both 400 mg/kg however in the six day trial the extract was administered intraperitoneally. One hypothesis for this may be that sage extract treats hyperglycemia on the level of the gastrointestinal tract and is supported by the observation that sage extract inhibits α-amylase and α-glucosidase in vitro (Kwon et al., 2006; Cazzola et al., 2011).

Sage may also convey its anti-diabetic effect by antagonizing PPAR-γ (Mueller and Jungbauer, 2009). Sage extract was also found to moderately transactivate PPAR-γ by the same group that identified it as an antagonizer. However work by (Christensen et al., 2010) indicates that sage extract does not stimulate adipocyte differentiation yet still increases insulin stimulated glucose uptake by adipocytes. Beyond enzyme inhibition, sage has been shown to prevent albumin glycation in vitro (Cazzola et al., 2011) as well as reduce serum urea, uric acid and creatinine in diabetic rats (Eidi and Eidi, 2009).

Sage extract also improves the management of glucose in healthy animals. After five hours, healthy rats injected with sage did not have any variation in blood glucose (Eidi et al., 2005). When they were provided sage orally for 14 days, healthy rats consuming sage had lower serum glucose than the animals that did not consume sage. When given an intraperitoneal glucose tolerance test, there was no difference in glucose clearance between control and sage groups (Lima et al., 2007). This further supports the hypothesis that sage-extract acts on the gastrointestinal level and that in healthy animals, long term consumption of sage provides anti-diabetic benefits as well.

Research has also been completed regarding the potential anti-inflammatory activity of sage. Rats administered sage extract one hour prior to formalin or carrageenan injection had decreased inflammation at the injection site compared to the no-sage treatment control (Qnais et al., 2010). This may be a result of the effect of sage on inflammatory cytokine production. In LPS-stimulated macrophages treatment with sage extract decreased IL-6 and TNF-α protein production and inhibited iNOS protein expression (Mueller et al., 2010) compared to the LPS treated control. Additionally, sage extract inhibits COX-1 and COX-2 activity in vitro (Yi and Wetzstein, 2010).

Studies in humans indicate that sage extract also has beneficial effects on blood lipid profile. In a randomized double-blind placebo controlled clinical study, male and female subjects between 20 and 60 years old with newly diagnosed primary hyperlipidemia consumed a capsule containing 500 mg sage extract every eight hours for two months. At the end of the study period subjects had lower serum total cholesterol, triglycerides, LDL and VLDL compared to both baseline and the placebo group. Subjects also had increased HDL compared to the placebo group and baseline (Kianbakht et al., 2011). When healthy female subjects between 40 and 50 years old consumed sage aqueous infusion twice daily they had lower plasma LDL, total cholesterol and greater plasma HDL following two weeks of treatment. They also had increased erythrocyte superoxide dismutase activity and catalase activity. Plasma fasting glucose, post-prandial glucose and blood pressure were not affected (Sa et al., 2009).

Tarragon (Artemesia dracunculus)

Tarragon originated in northern Europe but is used frequently in the cuisine of southern France. It is primarily in béarnaise and béchamel sauces but also in flavoring chicken and egg dishes (Bown, 2001). It is a component of the French "bouquet garnis" and can also steeped in vinegars and oils to make dressings (Hill, 2004). Tarragon contains chalcones such as davidigenin and 2′,4′-dihydroxy-4-methoxydihydrochalcone as well as flavanones like sakuranetin (Eisenman et al., 2011).

Several in vivo studies have been performed in animals examining the effect of tarragon on diabetes. Extract from tarragon administered orally decreased fasting plasma glucose in genetically diabetic KK-Aγ mice, streptozotocin induced diabetic mice and high-fat diet induced diabetic mice (Ribnicky et al., 2006; Ribnicky et al., 2009; Watcho et al., 2010) but did not change plasma glucose levels in healthy mice (Ribnicky et al., 2006). Additionally, tarragon extract improved peripheral neuropathy in high fat diet induced obese mice and in streptozotocin induced diabetic mice (Watcho et al., 2010; Watcho et al., 2011). This may be in part due to the inhibitory effect of tarragon extract on aldose reductase (Logendra et al., 2006). However, in the streptozotocin induced diabetic mice there was no change in sorbitol pathway intermediates found in the sciatic nerve in the animals given tarragon extract (Watcho et al., 2011).

Research in vitro identifies potential mechanisms for this anti-diabetic effect. In human skeletal muscle cells isolated from individuals with type II diabetes, glucose uptake was significantly increased with tarragon treatment. However, tarragon extract did not significantly affect protein levels of insulin receptor substrate (IRS)-1, IRS-2, phosphatidylinositol-3 kinase, Akt, insulin receptor or glucose transporter type-4 but it did decreased levels of protein tyrosine phosphatase1B (PTP1B) (Wang et al., 2008). PTP1B has been implicated as a modulator of IRS-1 dephosphorylation (Calera et al., 2000) and thus, the action of tarragon may be a result of its effects on protein levels of this enzyme. Another mechanism of the action of tarragon may be by inhibition of phosphoenolpyruvate carboxykinase (PEPCK). Treatment of H4IIE hepatoma cells with tarragon extract inhibited PEPCK mRNA expression (Govorko et al., 2007) and these results were corroborated in vivo in streptozotocin-induced diabetic while the treatment did not effect PEPCK in healthy rats (Ribnicky et al., 2006). Lastly, tarragon extract may also prevent or mitigate diabetes

by increasing the binding of glucagon-like peptide-1 to its receptor in vitro (Ribnicky et al., 2006).

Thyme (Thymus vulgaris)

The thymus genus has a great deal of species, yet thymus vulgaris is the most widely used culinary and medicinal thyme species. *Thymus vulgaris* is native to Italy and the western Mediterranean and is used in a wide variety of savory dishes (Bown, 2001). It is a component of the French collections of herbs referred to as "*herbes de Provence*" and "*bouquet garni*" (Hill, 2004). Fresh thyme is high in rosmarinic acid and luteolin (Neveu et al., 2010).

To determine the effect of thyme on diabetes, rats were injected with streptozotocin to induce diabetes were orally administered thyme extract. After 28 days there was no change in fasting blood glucose however there was an improvement in serum lipid profile compared to the diabetic control (Ozkol et al., 2012). In these diabetic rats, thyme also protected against ocular and systemic oxidative stress. Thyme extract increased retinal and lens GSH. In the erythrocytes, thyme extract increased GSH, decreased MDA and increased catalase activity. There was no change in plasma MDA or GSH indicating that the antioxidant effect of thyme extract likely occurs in the erythrocyte portion of blood (Ozkol et al., 2012). More research needs to be performed regarding the effect of thyme on glucose management since in vitro thyme extract as well as other herbs high in rosmarinic acid antagonize PPAR-γ (Mueller and Jungbauer, 2009).

Regarding inflammation, thyme extract significantly inhibited metabolic activity, neutrophil adhesion and superoxide production in ovine neutrophils in a dose-dependent manner (Farinacci et al., 2008). Thyme extract also inhibited IL-6 release (Mueller et al., 2010), iNOS protein expression (Tsai et al., 2007), NO release (Tsai et al., 2007), COX-1 activity and COX-2 activity (Yi and Wetzstein, 2010) in LPS-stimulated macrophages. The effect of thyme on IL-6, iNOS and COX-2 may be a result of the inhibition of NFκB activity. In LPS-stimulated monocytes, thyme extract strongly inhibited NFκB activity to just 11% of the LPS-stimulated control and in TNF-α stimulated monocytes, thyme extract inhibited NFκB binding activity to 13% of the TNF-α -stimulated control (Paur et al., 2008).

Thyme extract may also be hypolipidemic, but may only be so after long term treatment. In hyperlipidemic rats, oral administration thyme extract did not affect plasma total cholesterol, HDL or LDL and increased plasma triglycerides after 24 h (Ramchoun et al., 2012). However, in diabetic rats thyme extract reduced serum LDL and triglycerides and increased serum HDL after 28 days (Ozkol et al., 2012). This indicates that lower dosing over an extending period of time may be required to see the hypolipidemic effect of thyme extract and that further research must be done in hyperlipidemic models to define the effect of thyme on blood lipid profile.

CONCLUSION

Herbs and spices have been used for culinary and medicinal purposes for hundreds of years yet research on them has only been performed in the past decades. This review has summarized the multiple health benefits of herbs and spices found in Mediterranean cuisine and has provided mechanistic explanations wherever possible. It is hoped that the information in this review will guide future research on these culinary species and justify future research on their effects on diabetes, inflammation, hyperlipidemia and hypertension in humans as well as support future research on potential therapeutic benefit of bioactive compounds isolated from these plants.

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