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Type 2 diabetes diminishes the benefits of dietary antioxidants: Evidence from the different free radical scavenging potential

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ABSTRACT

The development of food fortified with polyphenols and polyphenol-rich foods represents a novel approach for preventing or managing type 2 diabetes. Herein, taking advantage of several radical scavenging, the impact of plasma proteins in diabetes on the benefits of dietary polyphenols was investigated. It illustrated that plasma proteins masked the dietary polyphenols, thus reducing their radical scavenging potential. The plasma proteins from type 2 diabetics bind and protect (i.e., mask) the polyphenol antioxidants less effectively than the non-glycosylated ones in healthy blood do. In the blood of diabetics the less-protected (non-masked) antioxidants react with free radicals before being delivered to the tissues that need them. We should pay more attention to *in vivo* benefits of dietary polyphenols for type 2 diabetics.

1. Introduction

Diabetes mellitus is one of the most significant public health problems in the world. The number of diabetics is expected to reach 438 million by 2030 (approximately 7.5% of the adult population) (Wild, Roglic, Green, Sicree, & King, 2004). Recently, the WHO (2013) reported that there are about 347 million people worldwide have diabetes. Moreover, the majority of this increase was reported in developing countries (South America, China, and India), which are undergoing westernisation. For example, more than 100 million Chinese developed to type 2 diabetes in 2012. Diabetes is characterised as hyperglycemia with a high level of glucose (>6.10 mM), which can react with plasma proteins through a non-enzymatic process to form glycated hemoglobin and glycated albumin (Godzien, García-Martínez, Martinez-Alcazar, Ruperez, & Barbas, 2013).

These non-enzymatic modifications via the Maillard reaction have far-reaching effects on the metabolism and regulation, and may be responsible for increased infection and cancer rates within diabetics (Bierhaus, Hofmann, Ziegler, & Nawroth, 1998). Recently, Zwang, Gormally, Johal, and Sazinsky (2012) hypothesised that the glycation of serum proteins may improve the available free iron pool for bacteria in blood serum and weaken our innate immunity. Using fluorescence spectroscopy we have showed that the increasing glycation of plasma proteins in type 2 diabetics' blood can reduce their binding capability for dietary polyphenols (Xie, Xiao, Kai, & Chen, 2012).

Natural polyphenols especially flavonoids, phenolic acids and stilbenes are the most important antioxidants in human diets and have attracted great interests since the 1990s due to growing evidences of their beneficial effect on human health (Deng et al., 2013; Shen & Chang, 2013; Xiao & Högger, 2014; Xiao, Kai, Yamamoto, & Chen, 2013; Xiao, Muzashvili, & Georgiev, 2014; Xiao, Ni, Kai, & Chen, 2013, in press).

Evidence from epidemiological studies suggests that there is powerful evidence linking dietary polyphenols consumption with the risk factors defining type 2 diabetes, even if some opposite results occurred (Liu et al., 2014; van Dam, Naidoo, & Landberg, 2013). However, how dietary polyphenols impact major endpoints of type 2 diabetes are still inconclusive due to the complex relationships among the risk factors of type 2 diabetes and the high number of dietary polyphenols (including their metabolites) present in the diet.

Almost all polyphenols show anti-oxidant potential which most likely mediate their beneficial health effects associated with cancer, diabetes, hypertension, cardiovascular and neurodegenerative diseases, aging, and so on (Thomas & Pfeiffer, 2012; Xiao, 2013). In this context, dietary polyphenols are proved to be highly active and are considered as viable alternatives to conventional drugs for various free radicals mediated diseases. However, the influence

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of high levels of glucose and glycated plasma proteins in diabetics on the free radical scavenging potential of natural antioxidants in blood is not clear. Herein, taking advantage of several anti-oxidative assays involving 2,2-diphenyl-1-picrylhydrazyl (DPPH) free radical, 2,2′-azinobis-(3-ethylbenzothiazoline-6-sulphonic acid) (ABTS) free radical, and superoxide anion radical scavenging and ferric reducing antioxidant potential (FRAP) assay, the difference between healthy human plasma proteins (HPP) and type 2 diabetes plasma proteins (TPP) on the antioxidant potential of dietary polyphenols was compared. Fifty dietary polyphenols (Table 1) were studied.

2. Materials and methods

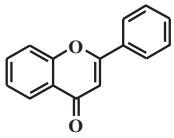
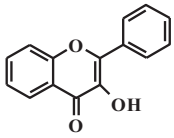
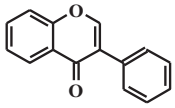
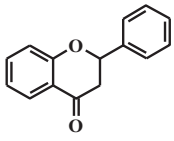
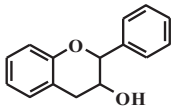
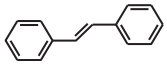
2.1. Chemicals

Biochanin A, genistein, apigenin, puerarin, catechin (C), epicatechin (EC), luteolin, GCG, DPPH, ABTS, EGCG, EGC, ECG, nitrotetrazolium blue chloride (NBT), potassium persulfate, and phenazine methosulfate (PMS) were purchased from Aladin Co. Ltd. (Shanghai, China). Nicotinamide adenine dinucleotide (NADH) was obtained from Sangon Biotech Co. Ltd (Shanghai, China). Flavone, chrysin, and baicalein (99.5%) were obtained commercially from Wako Pure Chemical Industries (Osaka, Japan). Flavanone, 7-hydroxyflavanone, 6-hydroxyflavanone, 6-methoxyflavanone, 6-hydroxyflavone, 6-methoxyflavone, naringenin, quercetin, and fisetin naringenin were purchased from TCI Chemical Industries (Tokyo, Japan). The other polyphenols were obtained commercially from Shanghai Tauto Biotech CO., Ltd (Shanghai, China). The working solutions of polyphenols (1.0×10^{-3} M) were prepared by dissolving each polyphenol in methanol. All other reagents and solvents were of analytical grade and all aqueous solutions were prepared using freshly double-distilled water.

2.2. Apparatus

UV Absorbance was read on a TU-1810 UV-spectrophotometer (Beijing, China). The fluorescence spectra were recorded on a

Table 1
The chemical structure of natural polyphenols tested in this study.

Subclass	Name	Substitutions		
		OH	OCH_3	Others
Flavones	Flavone			
	7-Hydroxyflavone	7		
	6-Hydroxyflavone	6		
	6-Methoxyflavone		6	
	7,8-Dihydroxyflavone	7,8		
	Baicalein	5,6,7		
	Chrysin	5,7		
	Baicalin	5,6		7-β-D-glucuronide
	Luteolin	5,7,3′, 4′		
	Wogonoside	5	8	7-O-glucuronide
Flavonols	Galangin	3,5,7		
	Kaempferide	3,5,7	4′	
	Kaempferol	3,5,7, 4′		
	Quercetin	3,5,7,3′, 4′		
	Quercitrin	5,7,3′, 4′		3-O-β-D-glucoside
	Myricetin	3,5,7,3′, 4′, 5′		
	Myricetrin	5,7,3′, 4′, 5′		3-O-rhamnoside
	Fisetin	3,7,3′, 4′		
	Rutin	5,7,3′, 4′		3-α-L-Rham-1,6-D-Glc
Isoflavones	Formononetin	7	4′	
	Genistein	5,7,4′		
	Daidzein	7,4′		
	Daidzin	4′		7-Glucoside
	Genistin	5,4′		7-Glucoside
	Biochanin A	5,7	4′	
	Puerarin	7,4′		8-C-glucoside
	Sophoricoside	5,7		4′-O-glucoside
Flavanone	Naringenin	5,7, 4′		
	Naringin	5,4′		7-Neohesperidose
	Hesperidin	5,7, 3′	4′	
	Hesperitin	5,3′	4′	7-α-L-Rham-1,6-D-glc
	Dihydromyricetin	3,5,7,3′, 4′, 5′		
	Flavanone			
	7-Hydroxyflavanone	7		
	6-Hydroxyflavanone	6		
	Silibinin			
	4′-Hydroxyfavanone	4′		
	Neohesperidin	5′	4′	7-O-neohesperidoside
Flavanonol	GCG (2,3-trans)	5,7,3′,4′,5′		3-Gallate
	EGCG (2,3-cis)	5,7,3′,4′,5′		3-Gallate
	ECG (2,3-cis)	5,7,4′,5′		3-Gallate
	EGC (2,3-cis)	3,5,7,3′,4′,5′		
	EC (2,3-cis)	3,5,7,4′,5′		
Stilbenes	Resveratrol	3,5,4′		
	Polydatin	5,4′		3-Glucoside

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