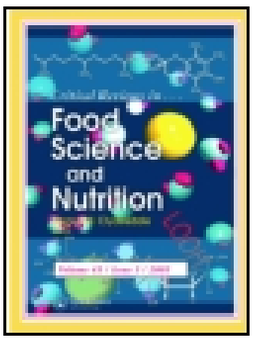

Critical Reviews in Food Science and Nutrition

ISSN: 1040-8398 (Print) 1549-7852 (Online) Journal homepage: http://www.tandfonline.com/loi/bfsn20

Nutritional chemistry of the peanut (Arachis hypogaea)

Ondulla T. Toomer

To cite this article: Ondulla T. Toomer (2017): Nutritional chemistry of the peanut (Arachis hypogaea), Critical Reviews in Food Science and Nutrition, DOI: 10.1080/10408398.2017.1339015

To link to this article: http://dx.doi.org/10.1080/10408398.2017.1339015

Accepted author version posted online: 29 Jun 2017.
Published online: 29 Jun 2017.

CRITICAL REVIEWS IN FOOD SCIENCE AND NUTRITION
https://doi.org/10.1080/10408398.2017.1339015

Nutritional chemistry of the peanut (*Arachis hypogaea*)

Ondulla T. Toomer

United States Department of Agriculture-Agricultural Research Service, Market Quality and Handling Research Unit, Raleigh, NC, USA

ABSTRACT

Peanuts, *Arachis hypogaea*, are one of the most widely consumed legumes globally due to its nutrition, taste, and affordability. Peanuts are protein and energy-rich and have been utilized worldwide to address the nutritional needs in developing countries. Currently, its role in a heart-healthy diet has warranted tremendous attention among consumer groups and within the scientific community. Additionally, current studies have identified the value in the phytonutrient composition of peanuts, such as resveratrol, isoflavonoids, phenolic acids, and phytosterols, which may enhance overall health and wellness. This article presents a comprehensive review of the nutritional chemistry of peanut components (macronutrients—proteins, lipids, carbohydrates; micronutrients—vitamins, minerals, phytonutrients) as related to health and use within the body. An improved comprehensive knowledge and better understanding of the nutritional chemistry of peanuts enables us to better harness the power of these nutrients in improved peanut products within the food and feed industry.

KEYWORDS

Peanut nutrition; peanut nutritional chemistry; phytonutrients

Introduction

Peanut seeds are approximately 22 to 30% crude protein (Patee and Young, 1982; Settaluri et al., 2012) and are a great vegetarian source of protein and healthy fats. However, in years past peanuts and tree nuts were perceived as an unhealthy food due to their high fat content of $\geq$ 50% w/w (Zhao et al., 2012). Current research studies have demonstrated that dietary inclusion of peanuts and tree nuts has been linked to reduced heart disease (Jones et al., 2014), certain types of cancers (Gonzalez and Salas-Salvadó, 2006), and improved weight management (Moreno et al., 2013). A study published in the *New England Journal of Medicine* reported that eating nuts daily can reduce death from heart disease by 29%, and even eating peanuts just twice a week can reduce risk by 24% (Boa et al., 2013). Other studies have demonstrated that regular peanut consumption helps to decrease blood pressure among hypertensive individuals with significant reduction in diastolic blood pressure (Jones et al., 2014).

In parallel, four large epidemiological studies (Adventist Health Study, Iowa Women's Health Study, Nurses' Health Study, and Physician's Health Study) examined the effect of frequent nut consumption (tree nuts and peanuts) on the risk of coronary and ischemic heart disease. The Adventist Health Study (Fraser et al., 1992) demonstrated that subjects who consumed nuts more than four times a weeks experienced significantly fewer coronary heart disease-related events and fewer definite fatal coronary heart disease-related events (Francisco and Resurreccion, 2008). The Nurses' Health Study showed that women who consumed more than one ounce of nuts per week had significantly lower risk of total coronary heart disease in comparison to women who consumed less than one ounce of nuts per month (Hu et al., 1998). Similarly, the Iowa Women's Health Study demonstrated that regular consumption of nuts significantly reduced the risk of coronary heart disease-related deaths in postmenopausal women (Ellsworth et al., 2001). In addition, the Physician's Health Study (Albert et al., 2002) reported that deaths related to cardiac events were significantly reduced in subjects who regularly consumed nuts (tree nuts and peanuts). Hence, today the overall image and consumer perception of peanuts has shifted from an energy dense food to a beneficial food item associated with improved health benefits.

The peanut also known as the groundnut and/or goober is a *legume* and taxonomically classified as *Arachis hypogaea*, is believed to have originated in Central and South America with cultivation spread to other parts of the world (Settaluri et al., 2012). Today, peanuts are cultivated in China, India, Africa, Japan, South America, and the United States, with more than 300 varieties grown worldwide (Settaluri et al., 2012). The four market types of peanuts grown commercially in the United States are runner, Virginia, Spanish, and Valencia.

The runner cultivar predominates 80% of the United States peanut market and is primarily used for peanut butter, and has an attractive uniform kernel size (American Peanut Council, 2014). Runners are predominately grown in Georgia, Alabama, Florida, Texas, and Oklahoma. The Virginia type peanuts have the largest kernel size and processed-in-shell are grown mainly in Virginia and North Carolina for gourmet snacks, and comprises approximately 15% of the United States peanut market (American Peanut Council, 2014). Spanish peanuts have a

CONTACT Ondulla T. Toomer Ondulla.Toomer@ars.usda.gov United States Department of Agriculture-Agricultural Research Service, Market Quality and Handling Research Unit, 400 Dan Allen Drive, Schaub Hall, Box 7624, Raleigh, NC 27695, USA.

Color versions of one or more of the figures in the article can be found online at www.tandfonline.com/bfsn.

smaller kernel size with a higher oil content than other peanut types are often used in peanut candies and snacks. Spanish type peanuts are grown in Oklahoma and Texas and compromise approximately 4% of the United States peanut market (American Peanut Council, 2014). Valencia type peanuts are produced primarily in New Mexico and comprise approximately 1% of the US peanut market (American Peanut Council, 2014). Valencia type peanuts have three or more small kernels to a pod and are very sweet in taste and are therefore are usually roasted and sold in-the-shell.

Peanuts can be eaten either raw, boiled, or roasted and are widely used to prepare a variety of packaged foods (peanut butter, candies, confections, and snack products) in the United States and are relied upon as a protein extender in developing countries.

This comprehensive review aims to focus on the nutritional chemistry of peanut components as related to health and use within the body. In contrast, previous peanut nutrition reviews have focused primarily on peanut allergens (Sáiz et al., 2013) and/or peanut phytonutrients exclusively (Francisco and Resurreccion, 2008; Sales and Resurreccion, 2014). All foods are composed of chemical compounds, which can be defined as macro- or micro-nutrients such as proteins, carbohydrates, fats, or vitamins, minerals and phytonutrients, respectively.

Macronutrients

Proteins

As peanuts are technically legumes they are more closely related to chickpeas and soybeans than to almonds, walnuts, or other tree nuts, and are more protein rich, and more nutritionally complete than tree nuts (Ros, 2010). Iqbal et al. (2016) reported that peanut proteins sampled from peanut seeds from different geographic locations around the world were the same, however with differing amounts. The peanut seed contains 32 different proteins (Pele, 2010) with only 18 of these proteins identified as allergenic (Yusnawan et al., 2012). To date, 17 peanut proteins (Ara h 1 through Ara h 17) have been identified as peanut allergens responsible for peanut allergy by the World Health Organization and International Union of Immunological Societies (WHO/IUIS, 2017) Allergen Nomenclature Sub-Committee, with Ara h 1, Ara h 2, and Ara h 6 being major peanut allergens (Porterfield et al., 2009). Nevertheless, only Ara h 1 through Ara h 10 have been well identified and biochemically characterized (Table 1). Other studies have defined Ara h 2 and Ara 6 as the major peanut allergens due to allergenic potency in allergenic-mediated assays (Zhuang and Dreskin, 2013). Interestingly, peanut allergens in particular have been shown to be extremely resistant to proteolytic digestion, and heat or chemical denaturation (Koppelman et al., 2010; Iqbal and Ateeq, 2013, Toomer et al., 2013). Other reports have demonstrated that allergenic food proteins are stable to digestion, and a common feature of many food allergens (Wickham et al., 2009).

These allergenic proteins found within peanut encompass seven different protein families, separated into two major protein fractions albumins and globulins (Patee and Young, 1982; Settaluri et al., 2012). The globulin fractions were first categorized as storage or reserve proteins of the peanut seed by Johns and Jones (1916) and by Jones and Horn (1930). The two-globulin fractions, arachin, and non-arachin compromise approximately 87% of the peanut seed proteins (Basha and Pancholy, 1981).

Globulin proteins

Ara ha 1 is a glycoprotein belonging to the vicilin (7S) legume globulin family. It compromises approximately 12–16% of peanut proteins (deJong et al., 1998) and affects 35–95% of the peanut-allergic population (Mari et al., 2006). Native Ara h 1 exists as a trimer formed by three identical monomers, with its basic structure and core region very similar to other 7S globulins. Mature Ara h 1 has 21 linear epitopes, with 14 within the core region of the protein. However, in the native trimer formation these epitopes are either slightly or significantly buried

Table 1. Proteins Found in Whole Conventional Peanuts.

Protein Superfamily	Protein Family	Allergen	Isoforms	MW (kDa)	Prevalence of IgE Binding	Biological Function
Cupins	Globulin-Vicilin-Type (7 S globulin)	Ara h 1	Ara h 1.0101	≈64 (trimer 180 kDa)	35-95%	
	Globulin-Glycinin-type (11 S globulin)	Ara h 3	Ara h 3.0101, Ara h 3.0201	60	≈50%	Trypsin Inhibitor
	Globulin-Glycinin-type (11 S globulin)	Ara h 4*	Ara h 3.0201	60	>50%	
Prolamin	Albumins-Conglutin (2 S Albumin)	Ara h 2	Ara h 2.0101, Ara h 2.0201	≈17	>95%	Trypsin Inhibitor
	Albumins-Conglutin (2 S Albumin)	Ara h 6	Ara h 6.0101	15	38%	
	Albumins-Conglutin (2 S Albumin)	Ara h 7	Ara h 7.0101, Ara h 7.0201	15	13%¥	
	Non-Specific Lipid Transfer Proteins	Ara h 9	Ara h 9.0101, Ara h 9.0201	9.8	45%	Transport between cell membranes
Profilin	Profilin	Ara h 5	Ara h 5.0101	15	13%¥	Regulate polymerization of actin
Bet v 1 Family	Pathogenesis-Related Protien	Ara h 8	Ara h 8.0101, Ara h 8.0201	17	70%	plant protection pathogen invasion
Glycosyl transferase GT-C	Oleosin	Ara h 10	Ara h 10.0101, Ara h 10.0201	16	21%	Structural stability in plant oil bodies
		Ara h 11	Ara h 11.0101	14	not known	
Scorpion toxin-like knottin	Defensins	Ara h 12	Arah 12.0101	5 to 12	not known	host defense peptides
		Ara h 13	Ara h 13.0101	5 to 12	not known	host defense peptides

*Ara h 4 found to be an isomer of Ara h 3 = renamed Ara h 3.02
¥ = 5 patients out of 40 were IgE reactive

explaining the relatively weak Immunoglobulin E (IgE) reactivity in the native form, and strong IgE reactivity in the denatured form (Zhou et al., 2013).

Ara h 3 is a seed storage protein belonging to the legumin (11S globulin) family (Koppelman et al., 2003) and is IgE immunoreactive in approximately 50% of peanut allergic patients and functions as a trypsin inhibitor (Dodo et al., 2004; Wen et al., 2007). Mature Ara h 3 is a hexamer (360–380 kDa) formed by two trimers (Jin et al., 2009), with each monomer having four linear epitopes (Rabjohn et al., 1999). Ara h 3 in the native form has the fourth epitope fully exposed, while the other three epitopes are completely or almost buried; suggesting that epitopes 1 and 2 may not be recognized by IgE antibodies in the native form, while epitopes 4 and part of epitope 3 may be reactive in the native form of Ara h 3 (Jin et al., 2009). Ara h 4 is an isoform of Ara h 3 (Table 1) and is no longer thought to be a distinct allergen and thus has been renamed to Ara h 3.02 (WHO/IUIS Allergen Nomenclature Sub-Committee, 2017).

Albumin proteins

Ara h 2 is a glycoprotein and accounts for approximately 6 to 9% of total peanut protein (Koppelman et al., 2001) with a molecular weight of approximately 17 kDa (Sáiz et al., 2013). Ara h 2 is a 2S albumin also known as conglutin and functions as a trypsin inhibitor (Table 1), proteins that reduce the biological activity of trypsin the digestive enzyme responsible for dietary protein digestion (Maleki et al., 2003). Over 95% of the peanut allergy population in the United States are Ara h 2 IgE reactive (Koppelman et al., 2004; Scurlock and Burks, 2004; Palmer et al., 2005) and therefore the most potent peanut allergen among peanut-sensitive subjects and therefore clinically important in peanut allergen sensitivity. Structurally, Ara h 2 has five α-helices arranged in right-handed super helix connected by several extended loops with four conserved disulfide bridges and 10 highly exposed epitope binding sites (Zhou et al., 2013).

Ara h 6, also a conglutin (2S albumin) protein, has a molecular weight of 15 kDa and is 59% homologous to Ara h 2 with similar allergenicity (Koppelman et al., 2005; Chen et al., 2013). Studies by Suhr et al. (2004) and Lehmann et al. (2006) demonstrated that Ara h 6 is a heat and digestive stable protein resistant to proteolytic treatment. Ara h 7 also belongs to the conglutin (2 S albumin) protein family and has a molecular weight of 15 kDa, with 35% sequence homology to Ara h 2 and Ara h 6 and recognized by 13% of the sera from 40 peanut-allergic patients (Maleki et al., 2003). Currently, the WHO/IUIS Allergen Nomenclature Subcommittee (2017) recognizes two Ara h 7 isoforms, Ara h 7.0101 and Ara h 7.0201 (Table 1).

Other proteins (profilin, pathogenesis-related proteins, nonspecific lipid transfer proteins, oleosin, defensins)

Ara h 5 with a molecular weight of 15 kDa belongs to the profilin family of proteins (Table 1) and functions to regulate the polymerization of actin (Breiteneder and Radauer, 2004; WHO/IUIS Allergen Nomenclature Sub-Committee, 2017). Ara h 5 is present in low levels in peanut extracts and is only IgE reactive with approximately 13% of 40 peanut-allergic patients (Zhou et al., 2013).

Ara h 8 with a molecular weight of 17 kDa has been identified as a pathogenesis-related protein (Table 1) produced in plants in the event of pathogen invasion and serve as host protection (Van Loon, 1985). Ara h 9 functions as a non-specific lipid-transfer protein to shuffle phospholipids and other fatty acid groups between cell membranes with a molecular weight of 9.8 kDa (Sáiz et al., 2013). However, few allergen studies have been conducted to biochemically characterize the allergenicity of Ara h 9.

Ara h 10 (16 kDa) and Ara h 11 (14 kDa) belong to the oleosin structural protein family (Table 1) and are found in the plant oil bodies and serve to stabilize the oil body structure during maturation (Sáiz et al., 2013). Due to their association with oil bodies, Ara h 10 and Ara h 11 extraction and isolation are complicated and proven unsatisfactory (Millichip et al., 1996). Ara h 12 and Ara h 13 proteins are peanut defensins (Table 1) with molecular weights ranging from 5 to 12 kDa (WHO/IUIS Allergen Nomenclature Subcommittee, 2017), which function as host defense peptides against bacteria and/or fungi (Bublin and Breiteneder, 2014).

Lipids

Most peanuts grown worldwide are primarily produced for edible oil, due to its desirous mild taste and high smoke point relative to other vegetable-based cooking oils. After oil extraction from the seed, the remaining peanut meal is approximately 50% protein (Zhao et al., 2012).

Highly processed peanut oil (acid extracted, heat distilled) has been shown not to contain peanut proteins and can be safely consumed by peanut allergic subjects. However, cold-pressed or cold-extruded peanut oils, processed at lower temperatures may contain traces of peanut protein and may elicit allergic responses in peanut-sensitive patients (du Plessis and Steinman, 2004). Aflatoxin, a carcinogenic compound produced by the two fungi, *Aspergillus flavus* and *Aspergillus parasiticus*, are generally not found in refined peanut oil. However, aflatoxin contaminants can be found in crude or lightly processed peanut oil (Sanders, 2002).

In many countries, peanuts seeds provide a significant nutritious contribution to the diet due to their rich protein, lipid, and fatty acid content. Traditional peanut seed ranges in oil content from 44 to 56% with an average of 50% (Cobb and Johnson, 1973; Grosso et al., 1997), with a light yellow color and slightly nutty flavor. Flavor and quality of peanuts and peanut products are largely a function of the seed lipid chemistry, while peanut lipid and fatty acid composition is greatly dependent upon cultivar, seed maturity, and environmental conditions and geographic location (Brown et al., 1975; Young, 1996).

The chemical and physical properties of fats and oils are mainly determined by the fatty acid profile of the oil and their position within the triacylglycerol molecule. Peanut oil has a high oleic content, which is associated with good oxidative and frying stabilities. Peanut oil is a non-drying oil, which does not harden when exposed to air and solidifies from 0 to 3°C (Young, 1996; O'Brien, 2004). The three major fatty acids present in peanut oils as acylglycerols, esters formed from glycerol and fatty acids are Palmitic (C16:0), Oleic (C18:1), and linoleic (C18:2) acids. Normally Steric (C18:0), Arachidic (C20:0), Eicosenoic (C20:1), Behenic (C22:0), and Lignoceric (C24:0) acids occur in minor proportions, while trace levels of linolenic (C18:3) can also be present (Carrin and Carelli, 2010).

Peanut cultivars from the United States, Argentina, Bolivia, and Poland have the following fatty acid distributions: C16:0 = 9.3 to 13.0%, C18:0 = 1.1 to 3.6%, C18:1 = 35.6 to 58.3%, C18:2 = 20.9 to 43.2%, C20:0 = 0.3 to 2.4%, C20:1 = 0.7 to 3.2%, C22:0 = 1.8 to 4.4%, and C24:0 = 0.4 to 1.9% (Branch et al., 1990; Grosso and Guzman, 1991; Grosso et al., 1994). In parallel, African peanut oils contain 44.5, 32.3, and 13.9% of C18:1 (Oleic), C18:2 (Linoleic), and C16:0 (Palmitic), respectively (Carrin and Carelli, 2010). Studies by Hinds (1995) demonstrated that peanut seeds increased oleic fatty acid content, while decreasing palmitic and linoleic fatty acids during maturation.

Within the last decade, genetic manipulations have been utilized to alter peanut chemistry and improve nutritional quality of peanuts and peanut products. The incorporation of high-oleic traits into peanut breeding lines has resulted in elevated high-oleic fatty acid content and extended shelf life. Normal-oleic conventional peanut cultivars have a lipid profile of 52% oleic fatty acid and 27% linoleic fatty acids, while new high-oleic peanut cultivars have a lipid profile of 80% oleic fatty acids and 2% linoleic fatty acids (Isleib et al., 2006). The primary difference found in high-oleic peanuts is the replacement of linoleic acid by oleic fatty acids in the peanut oil.

Most of the fatty acids present in peanut oil are present as triacylglycerols (TAG) at approximately 93.3 to 95.8% of weight (Sanders, 2002). Studies by Sanders (2002) demonstrated that TAG content is dependent upon seed maturation and increases incrementally until full maturation. Sanders (2002) also concluded that not only does environment and location affect fatty acid composition, but also affects the spatial arrangement of these fatty acids found within specific TAG molecules, with a higher percentage of oleic or linoleic fatty acids in the *sn*-2 position. Moreover, the spatial arrangement of these fatty acids within the glycerol skeleton is nutritionally important. During the digestive process, fatty acids found within the *sn*-2 position are conserved, while fatty acids at the *sn*-1 and *sn*-3 positions are released by pancreatic lipase (Carrin and Carelli, 2010). Therefore, long-chain saturated fatty acids present preferentially at these positions and with melting points higher than human body temperature (C18:0, C20:0, C22:0, C24:0) remain free and solid within the intestinal lumen with weak intestinal absorption and therefore have no effect on plasma lipids (Dubois et al., 2007).

Free fatty acids (FFA) and diacylglycerols (DAG) can also be found in unprocessed peanut oil. Crude peanut oil can have an FFA content as low as 0.3%, while commercial oil ranges from 0.5 to 1.5% (Padley et al., 1994). FFA and DAG levels within peanut oil vary and are dependent upon seed maturity. For example, the Florunner peanut variety has a reduction in FFA content from 4.5 to 0.7%, and DAG levels from 2.4 to 0.5%, between the white immature stage to full maturity (Ayres, 1983). Healthy mature peanut seeds have an FFA content of less than 0.5% (Carrin and Carelli, 2010). However, if the seeds are damaged (i.e., mold), FFA content up to 5% may be found (Ayres, 1983). Thus, high levels of FFA may indicate poor handling, immaturity, or mold growth (Sanders et al., 1992).

Phospholipid content in peanut oil is very low (0.3 to 0.7%) and is a major constituent of the cell membranes of the seed. Peanut phospholipids (PL) have a high degree of unsaturation and the major PL in conventional peanut oils are phosphatidylcholine, phosphatidic acid, phosphatidylethanolamine, phosphatidylinositol, and phosphatidylglycerol (Singleton and Stikeleather, 1995).

Carbohydrates

Oil or dry roasted peanuts contain approximately 21.51 g of carbohydrates per 100 g (USDA, Food Composition Database, 2017) with starch as the major carbohydrate. However, peanut research has demonstrated that peanut carbohydrate content is dependent upon cultivar, maturation, and geographic location (Pattee and Young, 1982) and may contain the following carbohydrates in varying quantities (major to minor): sucrose, fructose, glucose, inositol, raffinose, stachyose. Pattee and Young, (1982) reported that upon thermal processing (roasting), sucrose undergoes hydrolysis liberating fructose and glucose, which in turn react with free amino acids to form the characteristic flavor of roasted peanuts (Pattee and Young, 1982). Defatted peanut flour has been shown to contain approximately 38% total carbohydrates (Fig. 1) of which account for oligosaccharides 18%, starch, 12.5%, hemicellulose A 0.5%, hemicellulose B 3.5%, and cellulose (fiber) 4.5% (Tharanathan et al., 1975). Of the oligosaccharide fraction, approximately 13.90% sucrose, 0.89% raffinose, 1.56% stachyose, and 0.41% verbascose in unprocessed peanut flour (Tharanathan et al., 1975). Raffinose, stachyose, and verbascose, non-digestible short-chain oligosaccharides (Fig. 1), are indigestible in the human digestive tract and pass unchanged to the colon, and are subject to bacterial fermentation in the lower gut causing abdominal bloating (Li et al., 2013). The enzyme alpha-galactosidase responsible for their digestion and can be purchased as an over-the-counter supplement to prevent gas after eating legumes.

Starch is a homopolysaccharide made up of α-D glucose residues joined by glycosidic bonds. Upon digestion, salivary and pancreatic amylase catalyzes the hydrolysis of starch to maltose and maltotriose, isomaltose (Zeeman et al., 2010). Subsequently, these disaccharides are catalyzed by digestive enzyme sucrose-isomaltase (Fig. e 1) found within the apical brush border membrane of the small intestine to liberate two units of glucose (Gericke et al., 2017), the functional unit of energy needed to fuel growth, development, and maintenance.

Pattee et al. (2000) demonstrated that carbohydrate composition is also dependent upon market-type and maturation. However, in these studies while the carbohydrate values were different between market-types, they were not significantly different. Moreover, studies by Pattee et al. (1998) reported that increased sweetness directly related to carbohydrate content was associated with superior flavor profiles, with reduced bitterness and improved roasted peanut flavor.

Micronutrients

Vitamins

Peanuts provide a valuable source of water-soluble B vitamins and Vitamin E (tocopherol). Vitamin E (tocopherol) is a fat-

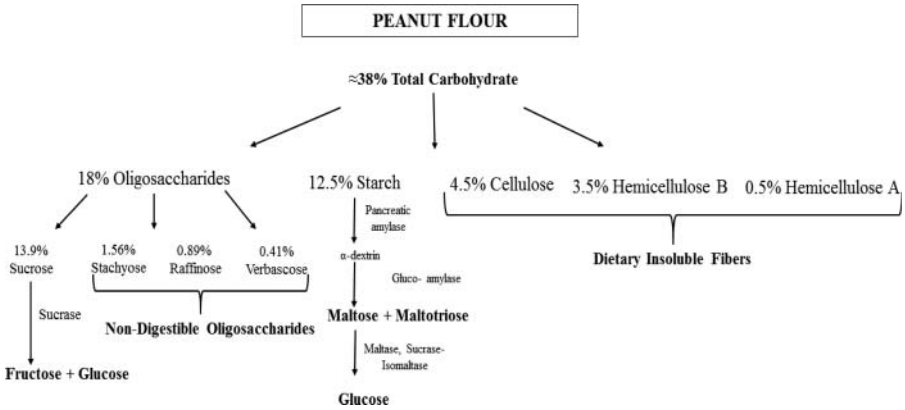

Figure 1. Carbohydrates in peanut flour from whole conventional peanuts. Defatted contains approximately 38% total carbohydrates comprising 18% oligosaccharides, 12.5% starch, 0.5% hemicellulose A, 3.5% hemicellulose B, and 4.5% cellulose (fiber) (Tharanathan et al., 1975). The oligosaccharide fraction is comprised of 13.90% sucrose, 0.89% raffinose, 1.56% stachyose, and 0.41% verbascose. Raffinose, stachyose, and verbascose, non-digestible oligosaccharides are indigestible in the human digestive tract and are subject to bacterial fermentation in the lower gut causing abdominal bloating. Starch is a homopolysaccharide made up of α-D glucose residues joined by glycosidic bonds. Salivary and pancreatic amylase catalyzes the hydrolysis of starch to maltose and maltotriose. Subsequently, these disaccharides are catalyzed by digestive enzyme maltase, sucrose, and/or sucrose-isomaltase to free glucose.

soluble vitamin that is an antioxidant. Tocols (fundamental unit of the tocopherol family) are naturally occurring antioxidants found in plant oils like peanuts and include four tocopherol and four tocotrienol (members of the Vitamin E family) isomers, designated as α, β, γ, and δ. These antioxidants inhibit lipid peroxidation in foods by stabilizing hydro-peroxides and other free radicals. Studies have demonstrated that these antioxidants decrease during processing of the peanut oil, with chemical refining removing as much as 10 to 20% of the tocopherols and tocotrienols, and 30 to 60% being lost with deodorization or steam distillation (O'Brien, 2004). Crude peanut oil has only 30 to 40% of the tocopherol content that soybean oil has, but has almost three times as much tocotrienols than soybean oil (Carrin and Carelli, 2010). Studies by Chun (2002) reported a tocopherol content of 8.2 mg/ 100 g in raw conventional peanuts and a tocopherol content of 4.1 mg/100 g in roasted peanuts (Table 2).

Hashim et al. (1993) found that tocopherol content in peanut oil was dependent upon stage of maturation and peanut cultivar, with there being significant differences between maturity stages of runner- and Virginia-type peanut cultivars. Upon comparisons of tocopherol content in peanut oil from peanuts grown in Argentina, China, and the United States, Sanders et al. (1992) found that peanut oil from peanuts grown within the United States had higher contents of tocopherols (210.1 to 243.8 ppm) and lowest in peanut oil from peanuts grown in China (102.9 to 183.9 ppm). Therefore, peanuts can provide a dietary source of vitamin E important to human nutrition.

Additionally, peanuts are a good source of water-soluble vitamin thiamine (B_1), which functions as a coenzyme in carbohydrate and amino acid metabolic pathways (Table 2). Studies by Dougherty and Cobb (1970a) reported thiamine content in the peanut seed to be about 1.0 mg/100 g, while thiamine content in peanut testa (skin) to be considerably higher at approximately 3.8 mg/100 g (Dougherty and Cobb, 1970b). Peanuts are also an efficient source of riboflavin (B_2), which functions as a coenzyme in carbohydrate, lipid, and protein metabolic pathways and is approximately 0.098 mg riboflavin/100 g of dry roasted peanuts (Settaluri et al., 2012). Peanuts also provide B vitamin, niacin (B_3) an essential coenzyme in metabolic respiratory pathways within the mitochondria with an approximate amount of 13.525 mg/100 g of dry roasted peanuts (Table 2, Settaluri et al., 2012).

Vitamin B_5, pantothenic acid is responsible for the formation of Coenzyme A, which is responsible for vital reactions in energy metabolism, synthesis of cholesterol, and synthesis of heme. Vitamin B_5 is present in peanuts at approximate amounts of 1.395 mg/100 g of dry roasted peanuts (Table 2, Settaluri et al., 2012). Additionally, in smaller amounts vitamin B_6 (pyridoxine) and vitamin B_9 (folic acid) can be found in peanuts with approximate amounts of 0.256 mg and 145 μg per 100 g of dry roasted peanuts, respectively (Table 2, Settaluri et al., 2012). Vitamin B_6 (pyridoxine) functions biologically as an essential coenzyme in amino acid, glucose, and lipid metabolism pathways. Moreover, vitamin B_6 (pyridoxine) is needed for neurotransmitter, histamine, and hemoglobin synthesis, while vitamin B_9 (folic acid) is an essential vitamin needed for RNA, DNA synthesis, amino acid metabolism, cell division, and fetal development.

Minerals

Peanuts are a good dietary source of the macro minerals (Derise et al., 1974; Settaluri et al., 2012), which are the minerals needed daily in a quantity greater than 100 mg/day. Studies by Derise et al. (1974) demonstrated only slight variations in the mineral content between various peanut cultivars (Virginia-70, NC-2, Florigiant) and between raw and roasted peanuts. Peanuts contain approximately 658 mg/ 100 g (Table 2, Settaluri et al., 2012) of the vital mineral potassium, which functions along with sodium to maintain the bodies' electrolyte balance, and muscle and neurological function. Settaluri et al. (2012) also demonstrated that peanuts provide the macro minerals magnesium (175 mg), calcium (54 mg), and phosphorus (358 mg) per 100 g of dry roasted peanuts (Table 2). Magnesium is needed for normal muscle and nerve function and maintenance of blood pressure, while calcium is required for normal bone and tooth development and muscle function. Phosphorus (358 mg) along

Table 2. Micronutrients content of whole conventional peanuts.

Vitamins	Class	Name	Peanut content	Main biological function	References
	Fat soluble	Tocopherol	8.2 mg/ 100 g raw, 4.1 mg/ 100 g roasted	Antioxidant	Chun, 2002
	Water soluble	Thiamine (B1),	1.0 mg/100 g peanut seed	coenzyme in carbohydrate and amino acid metabolic pathways	Dougherty and Cobb, 1970a
		Riboflavin (B2)	0.098 mg/100 g of dry roasted peanuts	coenzyme in carbohydrate, lipid, and protein metabolic pathways	Settaluri et al., 2012
		Niacin (B3)	13.525 mg/100 g of dry roasted peanuts	coenzyme in metabolic respiratory pathways	Settaluri et al., 2012
		Pantothenic acid (B5)	1.395 mg/100 g of dry roasted peanuts	formation of Coenzyme A	Settaluri et al., 2012
		Pyridoxine (B6)	0.256 mg/100 g of dry roasted peanuts	coenzyme in amino acid, glucose, and lipid metabolism pathways	Settaluri et al., 2012
		Folic acid (B9)	145 μg/100 g of dry roasted peanuts	nucleic acid synthesis, amino acid metabolism, early development	Settaluri et al., 2012
Minerals	Macro minerals	Potassium	658 mg/ 100 g of dry roasted peanuts	electrolyte balance, and muscle and neurological function	Settaluri et al., 2012
		Magnesium	175 mg/ 100 g of dry roasted peanuts	muscle and nerve function and maintenance of blood pressure	Settaluri et al., 2012
		Sodium	≈5.56 mg/ 100 g of roasted peanuts	electrolyte balance, hydration, function nerves and muscles	Derise et al., 1974
		Calcium	54 mg/ 100 g of dry roasted peanuts	normal bone and tooth development and muscle function	Settaluri et al., 2012
		Phosphorus	358 mg/ 100 g of dry roasted peanuts	bone and teeth formation, tissue growth and repair	Settaluri et al., 2012
	Trace minerals	Zinc	3.31 mg/100 g of dry roasted peanuts	immune system function, wound healing, cell division, and growth	Settaluri et al., 2012
		Iron	2.26 mg/ 100 g of dry roasted peanuts	essential element for blood production and oxygen transfer	Settaluri et al., 2012
		Manganese	≈2.06 mg/ 100 g of roasted peanuts	formation connective tissues, blood clotting factors, sex hormones, nerve function	Derise et al., 1974
		Copper	0.671 mg/ 100 g of dry roasted peanuts	red blood cells, healthy blood vessels, nerves, bones, immune support	Settaluri et al., 2012
		Selenium	7.5 μg/ 100 g of dry roasted peanuts	antioxidant, prevent cell damage	Settaluri et al., 2012

with calcium is required for bone and teeth formation, and protein synthesis in tissue growth and repair. Peanuts contain approximately 5.56 mg of sodium per 100 g of roasted peanuts (Table 2. Derise et al., 1974). Dietary sodium is important biologically for electrolyte balance, hydration, and proper functioning of nerves and muscles. In general, peanuts are a good dietary source of potassium, phosphorus, and magnesium.

Peanuts also provide a source of trace minerals, which are minerals needed daily in a quantity less than 100 mg/day. Settaluri et al. (2012) reported that peanuts provide the trace

Table 3. Phytonutrient content of whole conventional peanuts.

Class	Name	Peanut content	Biological function	References
Isoflavonoid	Daidzein	49.7 μg/ 100 g of dry roasted peanuts	precursor S-equol a non-steroidal selective agonist of the beta estrogen receptor	Mazur et al., 1998; Mazur, 1998
	Genistein	82.6 μg/ 100 g of dry roasted peanuts	antioxidant, anthelmintic, angiogenesis inhibitor, inhibits cancer cell growth	Mazur et al., 1998; Mazur, 1998
Phenolic acids	p-coumaric acid	6.9 mg/100 g of dry roasted peanuts		Talcott et al., 2005a, 2005b
Phytosterols	β-sitosterol	61 mg to 114 mg/100 g of roasted peanuts	may inhibit cancer growth, protect against heart disease	Awad et al., 2000
Stilbenes	Resveratrol	0.48 μg/g to 3.96 μg/g* peanut content with abiotic stress	anti-obesity, anti-diabetic, neuroprotective, cardioprotective, chemo-protective	Rudolf and Resurreccion, 2006

*peanut content with abiotic stress

minerals zinc (3.31 mg), iron (2.26 mg), copper (0.671 mg), and selenium (7.5 μg) per 100 g of dry roasted peanuts (Table 2). Derise et al. (1974) reported that peanuts provide approximately 2.06 mg of manganese per 100 g of roasted peanuts. Zinc is important biologically for proper function of the immune system, wound healing, and repair. Iron is an essential element needed for heme blood production and the transfer of oxygen within the body, while copper is needed for the formation of red blood cells and healthy blood vessels, nerves, and bones. Manganese is biologically important for the formation of connective tissue, bones, blood clotting factors, sex hormones, and nerve function, while the trace mineral selenium functions as an antioxidant. Based on these results, peanuts can provide over 70% of the daily need for copper and 14% of the daily need for selenium (Settaluri et al., 2012).

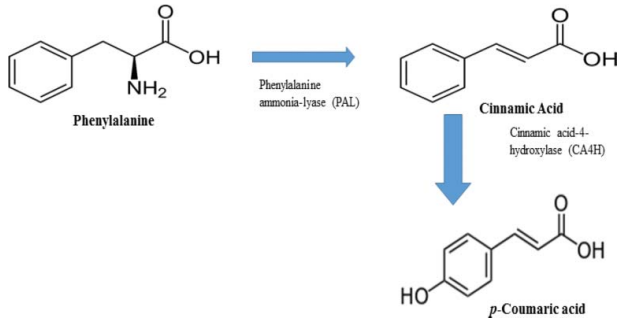

Figure 2. Plant phenylpropanoid pathway-biosynthesis of *p*-coumaric acid. L-Phenyalanine is an essential amino acids for mammals and dietary intake is necessary for protein synthesis. Fungi and plants produce phenylalanine via the shikimic acid pathway. Phenylalanine is directly utilized for protein synthesis in plants or metabolized through the phenylpropanoid pathway. Phenylalanine ammonia-lyase (PAL) is the enzyme responsible for the conversion of phenylalanine to intermediate cinnamic acid. Cinnamic acid-4-hydroxylase (CA4H) catalyzes the conversion of cinnamic acid to p-coumaric.

Phytonutrients

Isoflavonoids

Unlike macronutrients (proteins, fats, and carbohydrates), vitamins and minerals, phytonutrients are not essential to maintain life. However, when consumed they may help in disease prevention and promote health and wellness. Studies have demonstrated that peanuts provide valuable sources of these phytonutrients and therefore of nutritional importance as a functional food (Mazur et al., 1998, Mazur 1998). Studies by Mazur et al. (1998) and Mazur (1998) reported that peanut seeds had an isoflavonoid content of daidzein and genistein in the greatest amounts with a content of 49.7 μg/100 g and 82.6 μg/100 g, respectively (Table 3). Daidzein and genistein are isoflavone compounds that are commonly found in a number of plants, and are particularly abundant in soybeans and soy products (USDA Database, 2008).

Genistein and daidzein have similar structures to human estrogen and therefore are both identified as phytoestrogens. In plants, genistin is the precursor glucoside and in vivo is hyrdrolyzed by digestive enzymes to genistein and glucose. Various studies have demonstrated that genistein functions biologically also as an antioxidant (Han et al., 2009, Zhao et al., 2016), anthelmintic (Tandon et al., 2003), angiogenesis inhibitor (Farina et al., 2006), and inhibits cancer cell growth (Gossner et al., 2007; Raynal et al., 2008; Nakamura et al., 2009; Kim et al., 2009). In vivo daidzein is metabolized by human intestinal bacteria to produce end metabolite, S-equol, also known as (*S*)-(–)-4',7-isoflavandiol, an enantiomer of the naturally occurring isoflavondiol estrogen a non-steroidal selective agonist of the beta estrogen receptor (Muthyala et al., 2004). Research studies have reported the use of S-equol for the successful treatment of menopausal symptoms in women (Jackson et al., 2011).

Phenolic acids

Phenolic acids are abundant in plant-based foods (seeds, skins of fruits, leaves of vegetables) and therefore readily available in a balanced diet. Upon consumption phenolic acids are readily absorbed through the intestinal walls and may directly function as an antioxidant by preventing cellular damage due to oxidative free radicals (Gonçalves et al., 2017; Viapiana and Wesolowski, 2017). Moreover, studies have demonstrated that dietary phenolic acids promoted anti-inflammation in LPS challenged mice (Choi et al., 2017), in a murine model of induced-colitis (Monk et al., 2016), and a murine model of high dietary fat (Chang et al., 2015).

Normal oleic acid peanuts (raw and/or dry roasted) had the highest content of polyphenolic compounds in comparison to mid and high oleic peanut varieties, with free *p*-coumaric acid, three esterified derivatives of *p*-coumaric, and two esterified derivatives of hydrobenzoic acid identified as the predominate polyphenolic compounds found in peanuts (Talcott et al., 2005a, 2005b). Benzoic acid is a polyphenolic compound with gallic acid and cinnamic acid as derivatives (Natella et al., 1999), and *p*-coumaric as the hydroxyl derivative of cinnamic acid (Fig. 2). Whole raw peanuts had a range of 8 mg/kg to 66 mg/kg if *p*-coumaric acid (Table 3) among peanut cultivars, with the value increasing to an average of 69 mg/kg upon dry roasting (Table 3, Talcott et al., 2005a, 2005b). These studies demonstrate that differences in the content of polyphenolic compounds between peanut cultivars were highly dependent upon background genetics, growth conditions, disease resistance, post-harvest handling, and thermal treatment.

Phytosterols

Phytosterols, (i.e., plant sterols) are essential components of cell membranes and are similar to cholesterol. β-sitosterol has been shown to be the predominate source of phytosterol (PS) in peanuts with a content of 61 mg to 114 mg/100 g of roasted peanuts (Table 3; Awad et al., 2000). Studies have reported that β-sitosterol consumption may inhibit cancer growth (Awad et al., 1998; Roussi et al., 2005; Imanaka et al., 2008) and protect against heart disease (Peanut Institute, 2000; Bouchenak and Lamri-Senhadji, 2013). Among the peanut cultivars, Valencia peanuts (raw, dry, or oil roasted) contained the highest phytosterol content, with peanut butter (144–157 mg/100 g) and peanut flour (55–60 mg/100 g) containing significant amounts of phytosterols as well (Awad et al., 2000). Unrefined peanut oil has a phytosterol content of approximately 207 mg

phytosterols/100 g, which is 38% higher than the phytosterols content found in olive oil (Awad et al., 2000). Studies by Koehler and Song (2002) also determined that phytosterol content was not only influenced by cultivar, but also by maturation, with increasing amounts with maturity.

While phytosterols are similar to cholesterol, they are poorly absorbed across the intestinal mucosa and therefore circulating concentrations are low. After uptake by the intestinal enterocytes, phytosterols are actively excreted back into the intestinal lumen by the cassette (ABCG5/G8) transporter (Nissinen et al., 2002). It has been documented that phytosterols displace dietary cholesterol from uptake during the digestive process and therefore reduce the uptake and levels of circulating cholesterol (Nissinen et al., 2002). In a placebo-controlled study, moderate (0.46 g/day) and high (2.1 g/day) consumption of phytosterols reduced cholesterol absorption by about 10 and 25%, respectively, while significantly increasing excretion of endogenous and dietary cholesterol by 36 and 74%, respectively (Racette et al., 2010).

Stilbenes

Stilbenes chemically contain two phenyl compounds joined by a two-carbon methylene bridge. Stilbenes are a member of the vast group of polyphenols naturally occurring in plants. Stilbenes, like isoflavonoids are classified as phytoestrogens. Resveratrol (3,5,4′-trihydroxy-trans-stilbene), the most extensively studied stilbene, is a polyphenol phytoalexin mainly found in the skin of grapes. Recently, resveratrol has attracted tremendous scientific interest due to its potential health benefits related to cardiovascular (Diaz et al., 2016), chemo-protective (Zhang et al., 2015), anti-obesity (Zou et al., 2017), anti-diabetic (Tran et al., 2017), and neuroprotective (Pineda-Ramírez et al., 2017) properties.

Stilbenes found in peanut include resveratrol, 3-isopentadienyl resveratrol, and various arachidins (Ku et al., 2005). Resveratrol in peanuts serves to protect the plant from plant pathogens (Higgs, 2003). While peanuts are a source of resveratrol, the content is low in comparison to that of grapes. However, resveratrol content is much greater in the leaves, roots, and shells of peanuts (Chung et al., 2003). Seo et al. (2005) reported that resveratrol peanut content was dependent upon post-harvest processing, with a 45- to 65-fold increase after a treatment of 20-hour soaking/66-hours drying. Other studies by Rudolf and Resurreccion (2006) reported significant increases (0.48 μg/g to 3.96 μg/g) in resveratrol content with abiotic stresses (Table 3). Boiled peanuts having 10-fold greater resveratrol content in comparison to roasted peanuts or peanut butter (Rudolf, 2003).

Upon consumption, dietary resveratrol is well absorbed across the intestinal mucosa; however, its bioavailability is low due to its rapid metabolism and elimination. Resveratrol is rapidly metabolized by conjugation to glucuronic acid and/or sulfate, forming resveratrol glucuronides, sulfates, and/or sulfoglucuronides, with these sulfate conjugates being the major metabolites found in the plasma and urine of humans (Burkon and Somoza, 2008). Administration of single oral doses of 25 mg of *trans*-resveratrol to healthy volunteers resulted in peak blood concentrations of total resveratrol (i.e., *trans*-resveratrol plus its metabolites) around one hour later, at approximately 1.8–2 μmoles/l (μM), depending on whether resveratrol was administered in wine, vegetable juice, or grape juice (Goldberg et al., 2003; Walle et al., 2004).

Conclusion

Prior to the United States Civil War, peanuts were considered a regional Southern food. After the United States Civil War, technological advancements led to increased demand for peanut oil, peanut butter, and peanut products (roasted and salted and confections). Additionally, the scientific discoveries of George Washington Carver identified numerous non-food uses of the peanut and peanut plant, which encouraged the cultivation of peanuts as a profitable rotational crop for cotton (Agricultural Marketing Resource Center, 2017). Peanuts and peanut butter became an integral part of the American Armed Forces rations during World War I and II, with the United States Army popularizing the peanut butter and jelly sandwich for sustenance during military maneuvers in World War II (National Peanut Board, 2017).

Today, peanuts are the 12th most valuable cash crop grown in the United States with an estimated farm value over one billion U.S. dollars (American Peanut Council, 2017). Americans eat more than six pounds of peanut products annually, valued at more than $2 billion retail, with peanut butter accounting for approximately $850 million (American Peanut Council, 2017). Peanut butter has become an extremely popular, nutritious, and economical sandwich spread for children and adults across the United States.

As with many other food items, interest in nutritional composition and chemistry is resultant of their use in human food. Over the years, peanut feeding studies have demonstrated that regular peanut consumption has been linked to reduced heart disease (Jones et al., 2014), certain types of cancers (Gonzalez and Salas-Salvadó, 2006), and improved weight management (Moreno et al., 2013). Moreover, other studies have identified the value in the phytonutrient composition of peanuts, which may improve overall health and wellness (Bouchenak and Lamri-Senhadji, 2013; Sales and Resurreccion, 2014).

An improved knowledge or better understanding of the nutritional chemistry of peanuts enables us to better harness the power of these nutrients in improved peanut products within the food industry. Moreover, improved understanding of the nutritional chemistry of peanuts may help to identify the use of peanuts and/or peanut components more effectively in the agricultural feed industry to improve the health, growth, and performance of production animals.

While improved comprehensive understanding of the nutritional chemistry of peanuts better enables us to not only address issues related to nutrition and hunger worldwide, but also potentially improve health and wellness of consumers by knowledge of the functional components found within peanuts. Therefore, the spectrum of new and emerging peanut nutrition-related research continues to greatly expand. This review provides substantial evidence that peanuts are a nutritious food and food ingredient packed with health-promoting bioactive compounds and worthy of additional nutrition-related experimentation regarding this valuable agricultural commodity.

Acknowledgments

The author would like to thank Sabrina Whitley-Ferrell, in the Market Quality and Handling Research Unit for her assistance with the preparation of this manuscript. The author has no conflicts of interest to declare.

References

Agricultural Marketing Resource Center. (2017). Peanuts. http://www.agmrc.org/commodities-products/nuts/peanut-profile/. Accessed Date June 1, 2017.

Albert, C. M., Gaziano, J. M., Willett, W. C. and Manson, J. E. (2002). Nut consumption and decreased risk of sudden cardiac death in the physicians' health study. *Arch. Intern. Med.* **162**(12):1382–1387.

American Peanut Council. (2014). The Peanut Industry. https://www.peanutsusa.com/about-peanuts/the-peanut-industry3/9-peanut-types-and-production.html. Accessed Date June 1, 2017.

American Peanut Council. (2017). The Peanut Butter Portraits. https://peanutbutterportraits.wordpress.com/the-industry/. Accessed Date June 1, 2017.

Awad, A. B., Chan, K. C., Downie, A. C. and Fink, C. S. (2000). Peanuts as a source of beta- sitosterol, a sterol with anticancer properties. *Nutr. Cancer.* **36**(2):238–241.

Awad, A. B., von Holtz, R. L., Cone, J. P., Fink, C. S. and Chen, Y. C. (1998). beta-Sitosterol inhibits growth of HT-29 human colon cancer cells by activating the sphingomyelin cycle. *Anticancer Res.* **18** (1A):471–473.

Ayres, J. L. (1983). Peanut oil. *J. Am. Oil Chem. Soc.* **60**:357–359.

Bao, Y., Han, J., Hu, F. B., Giovannucci, E. L., Stampfer, M. J., Willett, W. C. and Fuchs, C. S. (2013). Association of nut consumption with total and cause-specific mortality. *N. Engl. J. Med.* **369**(21):2001–2011.

Basha, S. M. and Pancholy, S. K. (1981). Identification of methionine-rich polypeptides in peanut (Arachis hypogaea L.) seed. *J. Agric. Food Chem.* **29**(2):331–335.

Bouchenak, M. and Lamri-Senhadji, M. (2013). Nutritional quality of legumes and their role in cardiometabolic risk prevention: A review. *J. Med. Food.* **16**(3):185–198.

Branch, W. D., Nakayama, T. and Chinnan, M. S. (1990). Fatty acid variation among US Runner-type peanut cultivars. *J. Am. Oil Chem. Soc.* **67**:591–593.

Breiteneder, H. and Radauer, C. (2004). A classification of plant food allergens. *J. Allergy Clin. Immunol.* **113**(5):821–830.

Brown, D. F., Carter, C. M., Mattil, K. F. and Darroch, J. G. (1975). Effect of variety, growing location and their interaction on the fatty acid composition of peanuts. *J. Food Sci.* **40**:1055–1060.

Bublin, M. and Breiteneder, H. (2014). Cross-reactivity of peanut allergens. *Curr. Allergy Asthma Rep.* **14**(4):426–428.

Burkon, A. and Somoza, V. (2008). Quantification of free and protein-bound trans-resveratrol metabolites and identification of trans-resveratrol-C/O-conjugated diglucuronides - two novel resveratrol metabolites in human plasma. *Mol. Nutr. Food Res.* **52**(5):549–557.

Carrin, M. E. and Carelli, A. A. (2010). Peanut oil: Compositional data. *Eur. J. Lipid Sci. Technol.* **112**:697–707.

Chang, W. C., Wu, J. S., Chen, C. W., Kuo, P. L., Chien, H. M., Wang, Y. T. and Shen, S. C. (2015). Protective effect of vanillic acid against hyperinsulinemia, hyperglycemia and hyperlipidemia via alleviating hepatic insulin resistance and inflammation in high-fat diet (hfd)-fed rats. *Nutrients.* **7**(12):9946–9959.

Chen, X., Wang, Q., El-Mezayen, R., Zhuang, Y. and Dreskin, S. C. (2013). Ara h 2 and Ara h 6 have similar allergenic activity and are substantially redundant. *Int. Arch. Allergy Immunol.* **160**(3):251–258.

Choi, K. C., Son, Y. O., Hwang, J. M., Kim, B. T., Chae, M. and Lee, J. C. (2017). Antioxidant, anti-inflammatory and anti-septic potential of phenolic acids and flavonoid fractions isolated from *Lolium multiflorum*. *Pharm. Biol.* **55**(1):611–619.

Chun, J. Y. (2002). Vitamin E content and stability in peanuts and peanut products during processing and storage. Dissertation. The University of Georgia Athens, Athens, GA. http://purl.galileo.usg.edu/uga_etd/chun_ji-yeon_200212_phd. Accessed Date June 1, 2017.

Chung, I. M., Park, M. R., Chun, J. C. and Yun, S. J. (2003). Resveratrol accumulation and resveratrol synthase gene expression in response to abiotic stresses and hormones in peanut plants. *Plant Sci.* **164**:103–109.

Cobb, W. Y. and Johnson, B. R. (1973). Physiochemical properties of peanuts. In Peanuts: Culture and Uses. Chapter 6. pp. 209–256. American Peanut Research and Education. Stillwater OK, US., Stone Printing Company, Roanoke, VA.

deJong, E. C., Zijiverden, M. V., Spanhaak, S., Koppelman, S. J., Pellegrom, H. and Penninks, A. H. (1998). Identification and partial characterization of multiple major allergens in peanut proteins. *Clin. Exp. Allergy.* **28**(6):743–751.

Derise, N. L., Lau, H. A., Ritchey, S. J. and Murphy, E. W. (1974). Yield, proximate composition and mineral element content of three cultivars of raw and roasted peanuts. *J. Food Sci.* **39**:264–266.

Diaz, M., Degens, H., Vanhees, L., Austin, C. and Azzawi, M. (2016). The effects of resveratrol on aging vessels. *Exp. Gerontol.* **85**:41–47.

Dodo, H. W., Viquez, O. M., Maleki, S. J. and Konan, K. N. (2004). cDNA clone of a putative peanut (Arachis hypogaea L.) trypsin inhibitor has homology with peanut allergens Ara h 3 and Ara h 4. *J. Agric. Food Chem.* **52**(5):1404–1409.

Dougherty, R. H. and Cobb, W. Y. (1970a). Characterization of thiamine in the raw peanut. *J. Agric. Food Chem.* **18**:921–925.

Dougherty, R. H. and Cobb, W. Y. (1970b). Localization of thiamine within the cotyledon of dormant groundnut. *J. Sci. Food. Agric.* **21**:411–415.

Dubois, V., Breton, S., Linder, M., Fanni, J. and Parmentier, M. (2007). Fatty acid profiles of 80 vegetable oils with regard to their nutritional potential. *Eur. J. Lipid Sci. Tehnol.* **109**:710–732.

du Plessis, K. and Steinman, H. (2004). Practical aspects of adverse reactions to peanut. *Curr. Allergy. Clin. Immunol.* **17**:10–14.

Ellsworth, J. L., Kushi, L. H. and Folsom, A. R. (2001). Frequent nut intake and risk of death from coronary heart disease and all causes in postmenopausal women: the Iowa women's health study. *Nutr. Metab. Cardiovasc Dis.* **11**(6):372–377.

Farina, H. G., Pomies, M., Alonso, D. F. and Gomez, D. E. (2006). Antitumor and antiangiogenic activity of soy isoflavone genistein in mouse models of melanoma and breast cancer. *Oncol. Rep.* **16**(4):885–891.

Francisco, M. L. and Resurreccion, A. V. (2008). Functional components in peanuts. *Crit. Rev. Food Sci. Nutr.* **48**(8):715–746.

Fraser, G. E., Sabaté, J., Beeson, W. L. and Strahan, T. M. (1992). A possible protective effect of nut consumption on risk of coronary heart disease. The adventist health study. *Arch. Intern. Med.* **152**(7):1416–1424.

Gericke, B., Amiri, M., Scott, C. R. and Naim, H. Y. (2017). Molecular pathogenicity of novel sucrase-isomaltase mutations found in congenital sucrase-isomaltase deficiency patients. *Biochim. Biophys. Acta.* 1863 (3):817–826.

Goldberg, D. M., Yan, J. and Soleas, G. J. (2003). Absorption of three wine-related polyphenols in three different matrices by healthy subjects. *Clin. Biochem.* **36**(1):79–87.

Gonçalves, S., Moreira, E., Grosso, C., Andrade, P. B., Valentão, P. and Romano, A. (2017). Phenolic profile, antioxidant activity and enzyme inhibitory activities of extracts from aromatic plants used in Mediterranean diet. *J. Food Sci. Technol.* **54**(1):219–227.

Gonzalez, C. A. and Salas-Salvadó, J. (2006). The potential of nuts in the prevention of cancer. *Br. J. Nutr.* **96**:S87–S94.

Gossner, G., Choi, M., Tan, L., Fogoros, S., Griffith, K. A., Kuenker, M. and Liu, J. R. (2007). Genistein-induced apoptosis and autophagocytosis in ovarian cancer cells. *Gynecol. Oncol.* **105**(1):23–30.

Gross, N. R. and Guzman, C. A. (1991). Protein, oil content and fatty acid composition of Bolivian groundnut cultivars. *IAN.* **9**:20–21.

Grosso, N. R., Lamarque, A., Maestri, D. M., Zygadlo, J. A. and Guzman, C. A. (1994). Fatty acid variation of runner peanut among geographic localities from Cordoba (Argentina). *J. Am. Oil Chem. Soc.* **71**:541–542.

Grosso, N. R., Zygadlo, R., Lamarque, J. A., Maestri, A. L., Guzman, D. M. and Proximate, C. A. (1997). Fatty acid and sterol compositions of aboriginal peanut seeds from Bolivia. *J. Sci. Food Agric.* **73**:349–356.

Han, R. M., Tian, Y. X., Liu, Y., Chen, C. H., Ai, X. C., Zhang, J. P. and Skibsted, L. H. (2009). Comparison of flavonoids and isoflavonoids as antioxidants. *J. Agric. Food Chem.* **57**(9):3780–3785.

Hashim, I. B., Koehler, P. B., and Eitenmiller, R. R. (1993). Tocopheroles in runner and Virginia cultivars at various maturity stages. *J. Am. Oils. Chem. Soc.* **70**:633–635.

Higgs, J. (2003). The beneficial role of peanuts in the diet-Part 2. *Nutr. Food Sci.* **33**:56–64.

Hinds, M. J. (1995). Fatty acid composition of Caribbean-grown peanuts at three maturity stages. *Food Chem.* **53**:7–14.

Hu, F. B., Stampfer, M. J., Manson, J. E., Rimm, E. B., Colditz, G. A., Rosner, B. A., Speizer, F. E., Hennekens, C. H. and Willett, W. C. (1998). Frequent nut consumption and risk of coronary heart disease in women: Prospective cohort study. *BMJ.* **317**(7169):1341–1345.

Imanaka, H., Koide, H., Shimizu, K., Asai, T., Kinouchi, Shimizu, N., Ishikado, A., Makino, T. and Oku, N. (2008). Chemoprevention of tumor metastasis by liposomal beta-sitosterol intake. *Biol. Pharm Bull.* **31** (3):400–404.

Iqbal, A. and Ateeq, N. (2013). Effect of processing on the detectability of peanut protein by ELISA. *Food Chem.* **141**(3):1651–1654.

Iqbal, A., Shah, F., Hamayun, M., Ahmad, A., Hussain, A., Waqas, M., Kang, S. M. and Lee, I. J. (2016). Allergens of Arachis hypogaea and the effect of processing on their detection by ELISA. *Food Nutr. Res.* **60**:28945–28951.

Isleib, T. G., Pattee, H. E., Sanders, T. H., Hendrix, K. W. and Dean, L. O. (2006). Compositional and sensory comparisons between normal- and high-oleic peanuts. *J. Agric. Food Chem.* **54**(5):1759–1763.

Jackson, R. L., Greiwe, J. S., Desai, P. B. and Schwen, R. J. (2011). Single-dose and steady-state pharmacokinetic studies of S-equol, a potent nonhormonal, estrogen receptor β-agonist being developed for the treatment of menopausal symptoms. *Menopause.* **18**(2):185–193.

Jin, T., Guo, F., Chen, Y. W., Howard, A. and Zhang, Y. Z. (2009). Crystal structure of Ara h 3, a major allergen in peanut. *Mol. Immunol.* **46**(8–9):1796–1804.

Johns, C. O. and Jones, D. B. (1916). The globulins *Arachin* and Conarachin. *J. Biol. Chem.* **28**:77–87.

Jones, D. B. and Horn, M. J. (1930). The properties of *Arachin* and Conarachin and the proportional occurrences in the peanuts. *J. Agric. Res.* **40**:672–682.

Jones, J. B., Provost, M., Keaver, L., Breen, C., Ludy, M. J. and Mattes, R. D. (2014). A randomized trial on the effects of flavorings on the health benefits of daily peanut consumption. *Am. J. Clin. Nutr.* **99**(3):490–496.

Kim, S. H., Kim, S. H., Kim, Y. B., Jeon, Y. T., Lee, S. C. and Song, Y. S. (2009). Genistein inhibits cell growth by modulating various mitogen-activated protein kinases and AKT in cervical cancer cells. *Ann. N Y Acad. Sci.* **1171**:495–500.

Koehler, P. E. and Song, L. H. (2002). Changes in phytosterols of peanuts during maturation. Abstract#61D-23. Annual Meeting and Food Exposition. Anaheim California.

Koppelman, S. J., de Jong, G. A., Laaper-Ertmann, M., Peeters, K. A., Knulst, A. C., Hefle, S. L. and Knol, E. F. (2005). Purification and immunoglobulin E-binding properties of peanut allergen Ara h 6: Evidence for cross-reactivity with Ara h 2. *Clin. Exp. Allergy.* **35**(4):490–497.

Koppelman, S. J., Hefle, S. L., Taylor, S. L. and de Jong, G. A. (2010). Digestion of peanut allergens Ara h 1, Ara h 2, Ara h 3, and Ara h 6: A comparative in vitro study and partial characterization of digestion-resistant peptides. *Mol. Nutr. Food Res.* **54**(12):1711–1721.

Koppelman, S. J., Knol, E. F., Vlooswijk, R. A., Wensing, M., Knulst, A. C., Hefle, S. L., Gruppen, H. and Piersma, S. (2003). Peanut allergen Ara h 3: Isolation from peanuts and biochemical characterization. *Allergy.* **58** (11):1144–1151.

Koppelman, S. J., Vlooswijk, R. A., Knippels, L. M., Hessing, M., Knol, E. F., van Reijsen, F. C. and Bruijnzeel-Koomen, C. A. (2001). Quantification of major peanut allergens Ara h 1 and Ara h 2 in the peanut varieties Runner, Spanish, Virginia, and Valencia, bred in different parts of the world. *Allergy.* **56**(2):132–137.

Koppelman, S. J., Wensing, M., Ertmann, M., Knulst, A. C. and Knol, E. F. (2004). Relevance of Ara h1, Ara h2 and Ara h3 in peanut-allergic patients, as determined by immunoglobulin E Western blotting, basophil-histamine release and intracutaneous testing: Ara h2 is the most important peanut allergen. *Clin. Exp. Allergy.* **34**(4):583–590.

Ku, K. L., Chang, P. S., Cheng, Y. C. and Lien, C. Y. (2005). Production of stilbenoids from the callus of Arachis hypogaea: A novel source of the anticancer compound piceatannol. *J. Agric. Food Chem.* **53**(10):3877–3881.

Lehmann, K., Schweimer, K., Reese, G., Randow, S., Suhr, M., Becker, W. M., Vieths, S. and Rösch, P. (2006). Structure and stability of 2S albumin-type peanut allergens: implications for the severity of peanut allergic reactions. *Biochem. J.* **395**(3):463–472.

Li, T., Lu, X., and Yang, X. (2013). Stachyose-enriched α-galacto-oligosaccharides regulate gut microbiota and relieve constipation in mice. *J. Agric. Food Chem.* **61**(48):11825–11831.

Maleki, S. J., Viquez, O., Jacks, T., Dodo, H., Champagne, E. T., Chung, S. Y. and Landry, S. J. (2003). The major peanut allergen, Ara h 2, functions as a trypsin inhibitor, and roasting enhances this function. *J. Allergy Clin. Immunol.* **112**(1):190–195.

Mari, A., Scala, E., Palazzo, P., Ridolfi, S., Zennaro, D. and Carabella, G., (2006). Bioinformatics applied to allergy: allergen databases from collecting sequence information to data integration. The allergome platform as a model. *Cell. Immunol.* **244**(2):97–100.

Mazur, W. (1998). Phytoestrogen content in foods. *Baillieres Clin. Endocrinol. Metab.* **12**(4):729–742.

Mazur, W. M., Dukem, J. A., Wahala, K., Rasku, S. and Adlercreutz, H. (1998). Isoflavanoids and lignans in legumes: Nutritional and health aspects in humans. *Nutr. Biochem.* **9**:193–200.

Millichip, M., Tatham, A. S., Jackson, F., Griffiths, G., Shewry, P. R. and Stobart, A. K. (1996). Purification and characterization of oil-bodies (oleosomes) and oil-body boundary proteins (oleosins) from the developing cotyledons of sunflower (*Helianthus annuus L.*). *Biochem. J.* **314** (1):333–337.

Monk, J. M., Lepp, D., Zhang, C. P., Wu, W., Zarepoor, L., Lu, J. T., Pauls, K. P., Tsao, R., Wood, G. A., Robinson, L. E. and Power, K. A., (2016). Diets enriched with cranberry beans alter the microbiota and mitigate colitis severity and associated inflammation. *J. Nutr. Biochem.* **28**:129–139.

Moreno, J. P., Johnston, C. A., El-Mubasher, A. A., Papaioannou, M. A., Tyler, C., Gee, M. and Foreyt, J. P. (2013). Peanut consumption in adolescents is associated with improved weight status. *Nutr. Res.* **33** (7):552–526.

Muthyala, R. S., Ju, Y. H., Sheng, S., Williams, L. D., Doerge, D. R., Katzenellenbogen, B. S., Helferich, W. G. and Katzenellenbogen, J. A. (2004). Equol, a natural estrogenic metabolite from soy isoflavones: Convenient preparation and resolution of R- and S-equols and their differing binding and biological activity through estrogen receptors alpha and beta. *Bioorg. Med. Chem.* **12**(6):1559–1567.

Nakamura, Y., Yogosawa, S., Izutani, Y., Watanabe, H., Otsuji, E. and Sakai, T. (2009). A combination of indol-3-carbinol and genistein synergistically induces apoptosis in human colon cancer HT-29 cells by inhibiting Akt phosphorylation and progression of autophagy. *Mol. Cancer.* **8**:100–115.

Natella, F., Nardini, M., Di Felice, M. and Scaccini, C. (1999). Benzoic and cinnamic acid derivatives as antioxidants: Structure-activity relation. *J. Agric. Food Chem.* **47**(4):1453–1459.

National Peanut Board. (2017). History of Peanuts & Peanut Butter. http://nationalpeanutboard.org/peanut-info/history-peanuts-peanut-butter.htm. Accessed Date June 1, 2017.

Nissinen, M., Gylling, H., Vuoristo, M. and Miettinen, T. A. (2002). Micellar distribution of cholesterol and phytosterols after duodenal plant stanol ester infusion. *Am. J. Physiol. Gastrointest. Liver Physiol.* **282**(6): G1009–G1015.

O'Brien, R. D. (2004). Fats and Oils. Formulating and Processing for Applications. CRC Press, Boca Raton, USA.

Padley, F. B., Gunstone, F. D. and Harwood, J. L. (1994). Occurrence and characteristics of oils and fats. **In**: The Lipid Handbook, pp. 377–392. Gunstone, F. D., Harwood, J. L., and Padley, F. B., Eds., Chapman & Hall, London UK.

Palmer, G. W., Dibbern, D. A. Jr, Burks, A. W., Bannon, G. A., Bock, S. A., Porterfield, H. S., McDermott, R. A. and Dreskin S. C. (2005). Comparative potency of Ara h 1 and Ara h 2 in immunochemical and functional assays of allergenicity. *Clin. Immunol.* **115**(3):302–312.

Pattee, H. E., Isleib, T. G., Giesbrecht, F. G. and McFeeters, R. F. (2000). Relationships of sweet, bitter, and roasted peanut sensory attributes with carbohydrate components in peanuts. *J. Agric. Food Chem.* **48** (3):757–763.

Pattee, H. E., Isleib, T. G., Gorbet, D. W. and Giesbrecht, F. G. (1998). Selection of alternative genetic sources of large-seed size in Virginia-type peanut: Evaluation of sensory, composition, and agronomic characteristics. *J. Agric. Food Chem.* **50**(17):4885–4889.

Pattee, H. E. and Young, C. T. (1982). Peanut Science and Technology, pp. 655–668. American Peanut Research and Education Society, Inc., Yoakum, TX, USA.

Peanut Institute. (2000). Peanuts contain a phytosterol thought to inhibit cancer and help the heart. Available from http://www.peanut-institute.org/news-and- information/downloads/20000629_phytosterol_inhibits_cancer.pdf. Accessed March 24, 2017.

Pele, M. (2010). Peanut allergens. *Roman. Biotechnol. Lett.* **15**(2):5205.

Pineda-Ramírez, N., Gutiérrez Aguilar, G. F., Espinoza-Rojo, M. and Aguilera, P. (2017). Current evidence for AMPK activation involvement on resveratrol-induced neuroprotection in cerebral ischemia. *Nutr. Neurosci.* **14**:1–19.

Porterfield, H. S., Murray, K. S., Sclinchting, D. G., Chen, X., Hansen, K. C., Duncan, M. W. and Dreskin, S. C. (2009). Effector activity of peanut allergens a critical role for Ara h 2, Ara h 6 and their variants. *Clin. Exp. Allergy.* **39**(7):1099–1108.

Rabjohn, P., Helm, E. M., Stanley, J. S., West, C. M., Sampson, H. A., Burks, A. W. and Bannon, G. A. (1999). Molecular cloning and epitope analysis of the peanut allergen Ara h 3. *J. Clin. Invest.* **103**(4):535–542.

Racette, S. B., Lin, X., Lefevre, M., Spearie, C. A., Most, M. M., Ma, L. and Ostlund, R. E. Jr. (2010). Dose effects of dietary phytosterols on cholesterol metabolism: A controlled feeding study. *Am. J. Clin. Nutr.* **91** (1):32–38.

Raynal, N. J., Momparler, L., Charbonneau, M. and Momparler, R. L. (2008). Antileukemic activity of genistein, a major isoflavone present in soy products. *J. Nat. Prod.* **71**(1):3–7.

Ros, E. (2010). Health benefits of nut consumption. *Nutrients.* **2**(7):652–682.

Roussi, S., Winter, A., Gosse, F., Werner, D., Zhang, X., Marchioni, E., Geoffroy, P., Miesch, M. and Raul, F. (2005). Different apoptotic mechanisms are involved in the antiproliferative effects of 7beta-hydroxysitosterol and 7beta-hydroxycholesterol in human colon cancer cells. *Cell. Death Differ.* **12**(2):128–135.

Rudolf, J. L. (2003). Development of An HPLC Method for Resveratrol and Optimization of Post-Harvest Stress to Induce Production in Peanuts. Master's thesis, University of Georgia, Athens, GA.

Rudolf, J. L. and Resurreccion, A. V. A. (2006). Elicitation of resveratrol in peanut kernels by application of abiotic stresses. *J. Agric. Food Chem.* **53**:10186–10192.

Sáiz, J., Montealegre, C., Marina, M. L. and García-Ruiz, C. (2013). Peanut allergens: An overview. *Crit. Rev. Food Sci. Nutr.* **53**(7):722–737.

Sales, J. M. and Resurreccion, A. V. (2014). Resveratrol in peanuts. *Crit. Rev. Food Sci. Nutr.* **54**(6):734–770.

Sanders, T. H. (2002). Groundnut (peanut) oil. **In**: Vegetable Oils in Food Technology. Composition Properties, and Uses, pp. 231–243. Gunstone, F. D., Ed. Blackwell Publishing Ltd., Oxford, UK.

Sanders, T. H., Vercelloti, J. R., Crippen, K. L. and Hinsch, R. T. (1992). Quality factors in exported peantus from Argentina, China and the United States. *J. Am. Oil Chem. Soc.* **69**:1032–1035.

Scurlock, A. M. and Burks, A. W. (2004). Peanut allergenicity. *Ann. Allergy Asthma Immunol.* **93**(5 Suppl 3):S12–S18.

Seo, S. J., Lee, S. S., Chun, J., Lee, H. B. and Lee, J. (2005). Optimization for the post-harvest induction of trans-resveratrol in raw peanuts. Abstract#99B-31. 2005. IFT Annual Meeting, 15–20, New Orleans, LA US

Settaluri, V. S., Kandala, C. V. K., Puppala, N. and Sundaram, J. (2012). Peanuts and their nutritional aspects-a review. *Food Nutr. Sci.* **3**:1644–1650.

Singleton, J. A. and Stikeleather, L. F. (1995). High-performance liquid chromatography analysis of peanut phospholipids. I. Injection system for simultaneous concentration and separation of phospholipids. *J. Am. Oil. Chem. Soc.* **72**:481–483.

Suhr, M., Wicklein, D., Lepp, U. and Becker, W. M. (2004). Isolation and characterization of natural Ara h 6: Evidence for a further peanut allergen with putative clinical relevance based on resistance to pepsin digestion and heat. *Mol. Nutr. Food Res.* **48**(5):390–399.

Talcott, S. T., Duncan, C. E., Pozo-Insfran, D. D. and Gorbet, D. W. (2005a). Polyphenolic and antioxidant changes during storage of normal, mid, and high oleic acid peanuts. *Food Chem.* **89**:77–84.

Talcott, S. T., Passeretti, S., Duncan, C. E. and Gorbet, D. W. (2005b). Polyphenolic content and sensory properties of normal and high oleic acid peanuts. *Food Chem.* **90**:379–388.

Tandon, V., Da, S. B. and Saha, N. (2003). Anthelmintic efficacy of Flemingia vestita (Fabaceae): Effect of genistein on glycogen metabolism in the cestode, Raillietina echinobothrida. *Parasitol Int.* **52**(2):179–183.

Tharanathan, R. N., Wankhede, D. B. and Rao, M. R. R. (1975). Carbohydrate composition of groundnuts (Arachis hypogaea). *J. Sci. Food Agric.* **26**:749–754.

Toomer, O. T., Do, A., Pereira, M. and Williams, K. (2013). Effect of simulated gastric and intestinal digestion on temporal stability and immunoreactivity of peanut, almond, and pine nut protein allergens. *J. Agric. Food Chem.* **61**(24):5903–5913.

Tran, H. T., Liong, S., Lim, R., Barker, G. and Lappas, M. (2017). Resveratrol ameliorates the chemical and microbial induction of inflammation and insulin resistance in human placenta, adipose tissue and skeletal muscle. *PLoS One.* **12**(3):e0173373.

USDA Database for the Isoflavone Content of Selected Foods (Release 2.0). (2008). https://www.ars.usda.gov/ARSUserFiles/80400525/Data/isoflav/Isoflav_R2.pdf. p. 15, 22–28. Accessed Date June 1, 2017.

USDA, Food Composition Database (2017). Basic Report:16096, Peanuts, virginia, oil-roasted, with salt. https://ndb.nal.usda.gov/ndb/foods/show/4834. Accessed Date June 1, 2017.

Van Loon, L. C. (1985). Pathogenesis-related proteins. *Plant Mol. Biol.* **4** (2–3):111–116.

Viapiana, A. and Wesolowski, M. (2017). The phenolic contents and antioxidant activities of infusions of *Sambucus nigra L. Plant Foods Hum. Nutr.* **72**(1):82–87.

Walle, T., Hsieh, F., Delegge, M. H., Oatis, J. E, Jr. and Walle, U. K. (2004). High absorption but very low bioavailability of oral resveratrol in humans. *Drug Metab. Dispos.* **32**(12):1377–1382.

Wen, W. H., Borejsza-Wysocki, W., DeCory, T. R. and Durst, R. A. (2007). Peanut allergy, peanut allergens and methods for the detection of peanut contamination in food products. *Compr. Rev. Food Sci. F.* **6**(2):47–58.

WHO/IUIS Allergen Nomenclature Sub-Committee. Allergen Nomenclature Search Database. (2017). Arachis hypogaea (Peanut, groundnut) http://www.allergen.org/search.php?allergensource=Arachis+hypogaea. Accessed Date June 1, 2017.

Wickham, M., Faulks, R. and Mills, C. (2009). *In vitro* digestion methods for assessing the effect of food structure on allergen breakdown. *Mol. Nutr. Food Res.* **53**(8):952–958.

Young, T. (1996). Peanut oil. **In**: Bailey's Industrial Oil and Fat Products, Vol. 3, 5th Ed., pp. 377–392. Hui, Y. H., Ed. John & Wiley Sons, New York, USA.

Yusnawan, E., Marquis, C. P. and Lee, N. A. (2012). Purification and characterization of Ara h1 and Ara h3 from four peanut market types revealed higher order oligomeric structures. *J. Agric. Food Chem.* **60** (41):10352–10358.

Zeeman, S. C., Kossmann, J. and Smith, A. M. (2010). Starch: Its metabolism, evolution, and biotechnological modification in plants. *Annu. Rev. Plant Biol.* **61**:209–234.

zhang, l., chen, d. f., xia, x. y., yip, c., rieder, m. j. and bend, j. r. (2015). Potential complementary therapy for adverse drug reactions to sulfonamides: Chemoprotection against oxidative and nitrosative stress by TCM constituents and defined mixtures. *J. Pharm. Pharm. Sci.* **18** (4):528–546.

Zhao, L., Wang, Y., Liu, J., Wang, K., Guo, X., Ji, B., Wu, W. and Zhou, F. (2016). Protective effects of genistein and puerarin against chronic alcohol-induced liver injury in mice via antioxidant, anti-inflammatory, and anti-apoptotic mechanisms. *J. Agric. Food Chem.* **64** (38):7291–7297.

Zhao, X., Chen, J., and Du, F. (2012). Potential use of peanut by-products in food processing: A review. *J. Food Sci. Technol.* **49**(5):521–529.

Zhou, Y., Wang, J. S., Yang, X. J., Lin, D. H., Gao, Y. F., Su, Y. J., Yang, S., Zhang, Y. J. and Zheng, J. J. (2013). Peanut allergy, allergen composition, and methods of reducing allergenicity: A review. *Int. J. Food Sci.* **2013**:909140–909148.

Zhuang, Y. and Dreskin, S. C. (2013). Redefining the major peanut allergens. *Immunol. Res.* **55**(1–3):125–134.

Zou, T., Chen, D., Yang, Q., Wang, B., Zhu, M. J., Nathanielsz, P. W. and Du, M. (2017). Resveratrol supplementation of high-fat diet-fed pregnant mice promotes brown and beige adipocyte development and prevents obesity in male offspring. *J. Physiol.* **595**(5):1547–1562.