VITAMIN E CONTENT AND STABILITY IN PEANUTS AND PEANUT PRODUCTS DURING PROCESSING AND STORAGE

by

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(Under the Direction of Ronald R Eitenmiller)

ABSTRACT

Vitamin E content and changes in moisture, color, microstructure and oxidative stability of raw and roasted peanuts were investigated during processing and storage. The mean α-tocopherol levels (mg/100g) were 8.2 for raw peanuts, 4.1 for roasted peanuts, 9.4 for peanut butter, 5.5 for reduced-fat peanut butter and 12.3 for peanut oils. Contribution of the oil and stabilizer added to the roasted peanuts during peanut butter processing was 4% of α-T and 5% of γ-T in peanut butter. Tocopherol loss by peanut butter manufacture was below 6%. Oven roasting at 140 to 160 ºC rapidly decreased moisture content. About 5, 12, 20, and 10% of initial levels of α-, β-, γ- and δ-T in peanuts were lost during roasting at 160 ºC, respectively. Tocopherol levels of peanuts slightly increased at 140 ºC and were remained constant at 150 ºC. Tocopherol levels of peanut oils prepared by pressing the roasted peanuts significantly decreased at all roasting temperatures ($p<0.05$). After roasting at 160 ºC for 20 min, a 16% loss of α-T occurred in the peanut oils. Swollen epidermal cells and rupture of parenchyma tissue of peanuts were observed in roasted peanuts by SEM. During storage at 21 ºC, lipid oxidation rapidly progressed in roasted peanuts. Under vacuum, lipid oxidation was significantly retarded for both raw and roasted peanuts ($p<0.05$). Tocopherol levels of raw and roasted peanuts exponentially decreased with increasing peroxide value during storage ($\alpha\text{-T}=8.8956e^{-0.0468PV}$, $R^2 = 0.9246$). Under air, α- and γ-T of roasted peanuts dramatically decreased during the initial four weeks of storage, resulting in about 84 and 68% losses for α- and γ-T, respectively. α-T showed the least stability during storage and the most stability to roasting. After 12 weeks of storage, more than 50% of α-T remained for roasted peanuts stored under vacuum compared to about 10% stored under air.

INDEX WORDS: Vitamin E, HPLC, Direct extraction, Peanuts, Peanut butter, Peanut oils, Roasting, Peanut butter manufacture, Storage, Oxidative stability, Microstructure, Color, Scanning electron microscopy

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A Dissertation Submitted to the Graduate Faculty of The University of Georgia in Partial Fullfillment of the Requirements of the Degree

DOCTOR OF PHILOSOPHY

ATHENS, GEORGIA

2002

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DEDICATION

This dissertation is dedicated to

My parents in law

and

My parents

For constant support, encouragement, and

unconditional love.

To

My beloved husband

Sung-Gil Choi

For his patience, deepest love and encouragement.

AND

To

My beloved sons

Seung-Beom Choi and Seung-Zoo Choi,

who give me strength, joy, patience and love.

Without all of you, nothing would be possible. I always love you.

ACKNOWLEDGMENTS

I would like to express my deepest appreciation and gratitude to my major professor, Dr. Ronald R. Eitenmmiller for his inspiration, invaluable guidance, lovely patience, constant encouragement, and financial support throughout my Ph.D. program. His belief and care of me helped me finish my research. I also would like to thank Philip E. Koehler, Dr. Robert D. Phillips, Dr. Robert L. Shewfelt, Dr. William L. Kerr for serving on my advisory committee. I am very grateful to the faculty and staff at the Food Science and Technology Department for their guidance and kind help toward completing my study.

Thanks to all graduate students and postdoctoral fellows past and present with whom I have worked in the laboratory and the department: Lin Ye, Junsoo Lee, Nantipa Pansawat (Tang), Jin-Kyung Kim, Yoo-Kyung Kim, In-Sook Son, Sunisa Siripongvutikorn, Walairut Chantarapanon (Jane), Poom, Panada Banjongsinsiri, Yusof Maskat, and others for their encouragement, assistance, and friendships.

Appreciation is also extended to Ms. Wonda Rogers who always listened to my voice and is a strong helper to work in the Lab. I would like to give a special thanks to Ms. Rosemarie Goodrum for encouragement and guidance for my defense seminar. Thanks are extended to Dr. Glene Ware and Jae-Choul Lee for assistance in statistics for experiments. I also like to thank to Mr. Robert Karn and TaraFoods, Albany, Georgia for providing all peanut and peanut butter samples and detail information for this research. I like to express special thanks to my wonderful friends in a church group and Alumni Family of Korea University for their love and friendships.

I have no words to express my sincere gratitude and indebtedness to my beloved parents, parents in law, husband and two sons, relatives and my wonderful neighbors for their unconditional love, unwavering encouragement, and faith during my entire life span.

Last, I would like to thank and worship my heavenly Father from whom all the blessing of my entire life and without whom nothing would be possible.

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CHAPTER I

INTRODUCTION

INTRODUCTION

Tree nuts and peanuts are becoming established as significant dietary components contributing to lowered incidence of coronary heart disease (CHD). Five large epidemiological studies including the Adventists Health Study (Fraser et al., 1992, 1995, 1997a, 1997b), the Iowa Women's Health Study (Prineas et al., 1993; Kushi et al., 1996), the Nurses' Health Study (Hu et al., 1998), the Cholesterol and Recurrent Events (CARE) Study (Brown et al., 1999), and the Physicians' Health Study (Albert and Willett, 2002) reported that overall, consuming nuts more than 5 times/week resulted in an 18-51% reduction in CHD risk. It was strongly supported by clinical findings that diets increasing the quantities of MUFA and PUFA supplied from nut slower total and LDL cholesterol levels which are risk factors for CHD (Spiller et al., 1992, 1998; Sabaté et al., 1993; Morgan and Clayshulte, 2000; Zambón et al., 2000; Curb et al., 2000; Rajaram et al., 2001; Almario et al., 2001; Hyson et al., 2002). This protective effect of nuts may be through multiple mechanisms due to the variety of nutrients and bioactive substances present in nuts. Folate, vitamin B-6, vitamin E, selenium, sterols, fiber and other phytochemicals as well as MUFA and PUFA in peanuts are considered as possible components to have protective effects on health (Sabate and Hook, 1996; Kris-Etherton et al., 1999).

Peanuts contain approximately 50% oil composed of about 81% unsaturated fatty acids of which about 39% are polyunsaturated (USDA, Nutrient Data Bank, 2002). Therefore, peanut lipids meet the criteria of high MUFA content for a cholesterol-lowering diet. Conversely, it makes peanuts susceptible to oxidative rancidity during processing and storage that is the most common cause of quality loss. The quality loss of lipids due to oxidation is of economic and nutritional importance to the peanut industry because of off-flavor development and the loss of nutrients such as essential fatty acids, amino acids, and lipid-soluble vitamins such as vitamin E.

Vitamin E delays the oxidation process by eliminating the free-fatty acid radicals, although it can neither completely prevent the autoxidation of lipids nor reverse the formation of peroxides. Due to its role as a scavenger of free radicals, vitamin E is also believed to protect against onset of chronic diseases, mainly cancer and cardiovascular

diseases (Burton an Ingold, 1989; Burton and Traber, 1990; Rader et al., 1997). Thus, it is heavily in demand as a supplementary nutrient. Natural food sources are of increasing interest to the consumer and industry.

Peanuts and peanut products are good sources of Vitamin E. The levels of vitamin E in peanuts are influenced by cultivar, genotype, growing conditions, origins, year, regions, and maturity stages (Baurnfeind, 1980; Sanders et al., 1992; Hashim et al., 1993a, 1993b). After harvesting, the vitamin E level of peanuts and peanut products can be influenced by processing, storage, and marketing, primarily due to the antioxidant role of vitamin E.

Peanuts are processed into peanut butter, snacks, confections and peanut oils. Roasting is a critical step which imparts flavor and inactivates lipoxygenase in peanuts but can decrease shelf life (St, Angelo et al., 1977, 1979). Nutrient compositional changes of peanuts associated with oil, protein, carbohydrate, mineral and water-soluble vitamin contents during roasting were reported (Derise et al., 1974; St. Angelo et al., 1977; Oupadissakoon et al., 1984; Damame et al., 1990).

Storage studies on several tree nuts including almond, macadamia, pecan, almond, walnut and cashew nut indicated that vitamin E content decreased as storage time increased. Rate of vitamin E loss is highly affected by storage temperature, the packaging material and availability of oxygen (Fourie and Basson, 1989; Yao et al., 1992; Erickson et al., 1994; Senesi et al., 1991, 1996; Lavedrine et al., 1997; Lima et al., 1998). Usually, roasted peanuts are more susceptible to oxidation compared to raw peanuts, suggesting that vitamin E might be less stable in roasted peanuts compared to raw peanuts during storage. A relationship between vitamin E stability and lipid oxidation has not been clearly established in peanuts.

The objectives of this study were:

1. To provide more complete data on the vitamin E content of commercial peanuts and peanut products including roasted peanuts, peanut butter and peanut oils.
2. To investigate the effect of commercial peanut butter manufacture on vitamin E content of peanuts

3. To study vitamin E stability of peanuts during roasting and delineate relationships to physical changes including color, moisture and microstructure of peanuts during roasting
4. To examine vitamin E content of peanut oils prepared from roasted peanuts as affected by roasting temperature and time
5. To examine vitamin E stability of raw and roasted peanuts during storage and to study the effect of vacuum packaging on vitamin E stability related to oxidative stability of peanuts.

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CHAPTER II

LITERATURE REVIEW

1. Peanut Production

The United States is a major producer of peanuts contributing approximately 10% to the world production. Seven states account for approximately 98% of all peanuts grown in the U.S.: Georgia (37.7%) grows the major proportion of all peanuts followed by Texas (23.2%), Alabama (10.5%), North Carolina (9.3%), Florida (6.4%), Virginia (5.4%), and Oklahoma (5.2%) (Percentages based on 1997 production of *quota and non-quota peanuts*) (USDA, NASS, 1998). The U.S. produces runner (73%), Virginia (22%), Spanish (4%), and Valencia (1%) peanuts (American Peanut Council, 2002). Each type is distinctive in size and flavor. Traditionally, in the U.S., runner peanuts are predominantly utilized for peanut products.

Growing

Peanuts are the seeds of an annual legume which grows close to the ground and produces its fruit below the soil surface. U.S. peanuts are planted after the last frost in April or May when soil temperatures reach 65-70°F (20°C). Peanuts may be cultivated up to three times, depending on the region, to control weeds and grasses. A climate with approximately 200 frost free days is ideal for a good crop. Warm weather adequate moisture and rich, sandy soil are required for good yield.

Harvesting

Harvesting occurs in two stages. Digging, the first stage, begins when about 70% of the pods are mature. The digger loosens the plant and cuts the tap root. A shaker lifts the plant from the soil, gently shakes the soil from the peanuts and inverts the plant, exposing the pods in a windrow for sun drying. The second stage of the harvest is curing. After curing in the field for 2 or 3 days, a combine separates the pods from the vines, placing the peanut pods into a hopper on the top of the machine. Freshly dug peanuts are then placed into wagons for further curing with forced hot air slowly circulating through the wagons. In this final stage of curing, moisture content is reduced to 8-10% for safe storage.

Grading

After proper curing, farmers' stock peanuts (harvested peanuts which have not been shelled and cleaned) are inspected and graded to establish the quality and value of the product. The inspection process determines the overall quality and on-farm value of the shelled product for commercial sales. Farmers' stock peanuts are separated into three classifications: Segregation I, Segregation II, or Segregation III. These classifications are based on the amount and type of damage apparent on the peanut kernels. Peanut shellers can buy only Segregation I for use in edible products. Those peanuts which are not classified as Segregation I are crushed for oil.

Shelling

Segregation I peanuts are first cleaned to remove stones, soil, bits of vines and other foreign material. The cleaned peanuts move by conveyer belt through shelling machines in which the peanuts are forced through perforated grates which separate the peanut kernels from the hulls. Shakers separate the kernels and the pods. The kernels are then sorted by size on various screens into market grades.

Inspection

The edible nuts are individually inspected with electronic eyes. In this process, discolored or defective nuts as well as any remaining foreign material are eliminated, which decreases the possibility of aflatoxin contamination.

2. Peanut Products

The prime market for U.S. peanuts is edible consumption unlike manufacturing in other countries where the end products are peanut oil, cake and meal. U.S. per capita consumption is approximately 6.5 pounds per person excluding crushing (American Peanut Council (APC), 2000). The major use of raw shelled peanuts is in peanut butter (40%), snack peanuts (10%) and confectionary (25%) (APC, 2000). Only 15% of U.S. production is normally crushed for oil.

Peanut butter

A physician in ST. Louis, Missouri, who believed peanut butter would be a good, nutritious food for patients, reportedly was the first to commercially manufacture peanut butter in the U.S. (Woodroof, 1983). Peanut butter is one of the America's favorite foods. Found in about 75% of American homes, peanut butter is considered by many to be a staple food like bread and milk. Peanut butter is microbiologically stable and semiperishable at ambient temperature due to low moisture content (<2%) and low water activity (a_w<0.4) at which microbial growth should not occur (Jay, 1996). The manufacture of peanut butter is divided into six steps, beginning with shelled peanuts;

(1) Roasting: Batch roasting of shelled peanuts requires heating at approximately 320°F with a holding time for 40-60 min to obtain a satisfactory roast. Currently used multi-stage roasters apply two or more separate heating zones with different temperatures to produce more even roast by raising the temperature of the nuts slowly to the roasting temperature. Young found that the optimum process requires approximately five separate heating zones to raise the temperature of the nuts slowly to the roasting temperature (Moss and Otten, 1989). He also found that peanuts roasted at low temperature for longer time have the best flavor and a longer shelf life due to more even roast. A Proctor-Schwartz roaster commercially used in Tara Foods (Albany, GA) uses two heating zones.
(2) Cooling: Roasting should be stopped at a definite point to produce a uniform product.
(3) Dry blanching: The peanuts are passed through the blancher and subjected to abrasion between brushes to loosen, crack and remove the skins.
(4) Picking and inspecting: The blanched nuts are screened and inspected to remove poor quality nuts, rocks or other undesirable matter.
(5) Grinding and cooling: Peanut butter is usually made by two grinding operations. The first reduces the peanuts to a medium grind and the second to a smooth even-texture. During grinding, the peanut butter is heated to about 170°F. After the stabilizer is added, the peanut butter is rapidly cooled. Cooling crystallizes the stabilizer, causing trapping of the peanut oil that was released by the grinding. For crunchy peanut butter, small piece of peanut granules can be added after the second grind.

(6) Packaging: Vacuum packaging is commercially used to minimize residual air in the headspace and in the butter to prevent oxidation.

By Federal regulation, in the U.S., any product labeled "peanut butter" must be at least 90% peanuts. The remaining 10% may include salt, sweetener and stabilizer (hydrogenated vegetable oils) which prevents the peanut oil from separating and rising to the top.

Snack peanuts

Americans consume more than 300 million pounds of snack peanuts each year (APC, 2000). Most snack peanuts are shelled and blanched prior to roasting. Peanuts can be oil-roasted in continuous cookers that take a steady stream of peanuts through hot oil for about 5 min or dry-roasted in a oven with dry, hot forced air. The peanuts are then roasted and packaged for sale. Often, in-shell peanuts are roasted with salt and occasionally with spicy seasonings. The largest pods are sold as in-shell peanuts.

Confectionery

The confectionery industry uses about 25% of the U.S. crop to make candy (APC, 2000). Five of the eight top selling chocolate candies contain peanuts or peanut butter.

3. Composition and Nutritional Value

Peanuts are an excellent source of nutrients with high lipid and protein content along with reasonable amounts of carbohydrates, vitamins, and minerals. The composition of peanuts was first reviewed by Hokffpauir and Guthrie (1945). The ranges of constituents in peanut kernels are 3.9-13.2% moisture content, 21.0-36.4% protein, 35.8-54.2% lipid, 1.2-4.3% crude fiber, 6.0-24.9% nitrogen-free extract, 1.8-3.1% ash, 0.1-0.3% reducing sugars, 1.9-5.2% disaccharide sugars, 1.0-5.3% starch, and 2.2-2.7% pentosans (Hokffpauir and Guthrie, 1945). The chemical composition of peanuts is affected by cultivar, maturity, curing, kernel size, treatments with herbicides and fungicides, year, location, season, variety or genotype, harvest time, processing and storage, but not always by a statistically significant amount (Worthington and Hammons,

1971; Worthington et al., 1972; Young et al., 1972, 1974a, 1974b; Pattee et al., 1974; Basha et al., 1976; Sanders, 1980; Ahmed and Young, 1982; Knauft et al., 1986; Branch et al., 1990; Chiou et al, 1992a, 1992b, 1995; Sanders et al., 1992; Basha, 1992; Basha and Young, 1992; Hashim et al., 1993a; Dutta et al., 1994; Grosso and Guzman, 1995a, 1995b; Bland and Lax, 2000; Pattee et al., 2000). The compositions of peanuts and peanut butter recently provided by USDA (Nutrient Database, Release 15, 2002) are shown in Table 1.

Peanuts contain about 25% protein. Over 87% of the peanut proteins are globulins of which the two major types are arachin (63%) and conarachin (33%) (Basha and Cherry, 1976). The arachin fraction, localized in the aleurone bodies, is rich in threonine and proline but has a chemical score of only 31-38 (Lusas, 1979). Conarachin found in the cytoplasm has a chemical score of 68-82 (Cobb and Johnson, 1973). Lysine is a limiting amino acid for school age children. Lysine, methionine and threonine levels in peanuts are deficient for the infant. However, peanut protein is recommended as a food supplement (Natarajan, 1980), provided other sources of essential amino acids are available in the diet.

Peanuts contain about 50% oil composed of 81% of unsaturated fatty acids of which about 39% is polyunsaturated (USDA, Nutrient Database, 2002) (Table 1-1). The quantities of fatty acids in peanuts (% of total fatty acids) are 8.2-15.1% palmitic (16:0), 1.1-7.2% stearic (18:0), 31.5-60.2% oleic (18:1), 19.9-45.4% linoleic (18:2), 0.8-3.2% arachidic (20:0), 0.6-2.6% eicosenoic (20:1) 1.8-5.4% behenic (22:0), 0.5-2.5% lignoceric (24:0) acids (Hammond et al., 1997). The principal sites of lipid storage in peanuts are the spherosomes, particles of about 1.0 to 2.0 μm in diameter bound by a membrane. Isolated peanut spherosomes contain 99.55% neutral lipid, 0.09% phospholipids, and 0.22% protein (Pattee et al., 1982a).

Peanuts contain about 20% carbohydrate. Sucrose is the most abundant carbohydrate present in peanuts (2.9-6.4%) and other sugars include stachyose, raffinose,glucose, fructose, arabinose and galactose (Ahmed and Young, 1982).

Table 1-1. Compositions of Raw and Dry-Roasted Peanuts and Peanut Butter (Value / 100 g of Edible Portion)

Nutrient	Units	Raw peanuts (all types)	Dry-roasted peanuts (all types, with salt)	Peanut butter (smooth style, with salt)
Proximates				
Water	g	6.50	1.55	1.22
Energy	kcal	567	585	593
Protein	g	25.80	23.68	25.21
Total lipid (fat)	g	49.24	49.66	51.03
Ash	g	2.33	3.60	3.25
Carbohydrate, by difference	g	16.14	21.51	19.28
Fiber, total dietary	g	8.5	8.0	5.9
Minerals				
Calcium, Ca	mg	92	54	38
Iron, Fe	mg	4.58	2.26	1.84
Magnesium, Mg	mg	168	176	159
Phosphorus, P	mg	376	358	369
Potassium, K	mg	705	658	669
Sodium, Na	mg	18	813	467
Zinc, Zn	mg	3.27	3.31	2.92
Copper, Cu	mg	1.144	0.671	0.138
Manganese, Mn	mg	1.934	2.083	0.447
Selenium, Se	mcg	7.2	7.5	7.5

USDA Nutrient Database for Standard Reference, Release 15 (August 2002)

Table 1-1. Compositions of Raw and Dry-Roasted Peanuts and Peanut Butter (Value / 100 g of Edible Portion)

Nutrient	Units	Raw peanuts (all types)	Dry-roasted peanuts (all types, with salt)	Peanut butter (smooth style, with salt)
Vitamins				
Vitamin C, total ascorbic acid	mg	0.0	0.0	0.0
Thiamin	mg	0.640	0.438	0.083
Riboflavin	mg	0.135	0.098	0.105
Niacin	mg	12.066	13.525	13.403
Pantothenic acid	mg	1.767	1.395	0.806
Vitamin B-6	mg	0.348	0.256	0.454
Folate, total	mcg	240	145	74
Folic acid	mcg	0	0	0
Folate, food	mcg	240	145	74
Folate, DFE	Mcg_DFE	240	145	74
Vitamin B-12	mcg	0.00	0.00	0.00
Vitamin A, IU	IU	0	0	0
Vitamin E	Mg_ATE	9.130	7.410	10.000
Lipids				
Fatty acids, total saturated	g	6.834	6.893	10.344
Fatty acids, total monounsaturated	g	24.429	24.640	24.276
Fatty acids, total polyunsaturated	g	15.559	15.694	13.788
Cholesterol	mg	0	0	0
Phytosterols	mg	220	NA	102

USDA Nutrient Database for Standard Reference, Release 15 (August 2000)

Oligosaccharides found in legumes are not broken down by digestive enzymes and pass to the ileum and colon where they are acted upon by bacteria to produce flatulence. Due to low levels of gas-producing oligosaccharides, like stachyose and raffinose, peanuts have limited flatulence capacity (Worthington and Beuchat, 1974).

Peanuts contain about 3% ash. Potassium, magnesium, phosphorus and sulfur are high and unaffected by heating (Woodroof, 1983). The broad spectrums of elements present at low to trace levels also contribute to the nutritional importance of peanuts. Peanuts are an excellent source of several important vitamins such as folate, thiamin, nicotinic acid and vitamin E. However, thiamin is destroyed to a large degree by roasting and blanching (Woodroof, 1983).

Peanuts and peanut products are whole foods with excellent nutrientional value (one ounce). Percentages of daily value (%DV) provided by one ounce (or 30 whole) of dry roasted peanuts are shown in Table 1-2. Peanuts contain relatively large quantities of protein compared to other legumes or nuts. A small handful of peanuts provide 14% of the protein DV. This is especially important for children, vegetarians and people eating more meatless meals. Peanuts provide large amounts of unsaturated fat, “good fat”, with no cholesterol. One handful of peanuts (approximately one ounce) is also a good source of fiber, providing 8% of the fiber required each day per serving. High amounts of minerals and vitamins, especially vitamin E (25% DV) can be provided by just one ounce of peanuts.

4. Beneficial Effects of Peanut Product Consumption on Health

Tree nuts and peanuts are becoming established as significant dietary components contributing to lowered incidence of coronary heart disease (CHD) (Fraser et al., 1992, 1995, 1997b; Fraser and Shavlik, 1997; Sabatè and Fraser 1993, 1994; Dreher and Maher 1996; Turnstall-Pedoe 1998; Rainey and Nyquist 1997; Hu et al., 1998, 1999; Brown et al., 1999; Feldman, 1999; Fraser, 2000; Hu et al., 2001; Kris-Etherton, 2001; Ellsworth et al., 2001; Albert and Willet, 2002). Fraser et al (1992) first indicated the possible protective effect of nut consumption against CHD from The Adventists Health Study.

Table 1-2. Nutrients of Dry Roasted Peanuts

Nutrients	% DV (serving size: 1 oz)	Nutrients	% DV (serving size: 1 oz)
Protein	14	Folate	10
Total fat	21	Copper	10
Saturated fat	10	Phosphorous	10
Carbohydrate	2	Potassium	10
Fiber	8	Thiamin	8
Cholesterol	0	Zinc	6
Vitamin E	25	Iron	4
Niacin	19	Calcium	2
Magnesium	12		

USDA, Agricultural Research Service, 1998 (Nutrient Database for Standard Reference, Release 12)

Dietary information obtained from 31,208 non-Hispanic white California Seventh-Day Adventists showed that consumption of nuts at more than four times per week decreased fatal CHD events when compared to subjects who consumed nuts less than once per week (Fraser et al., 1992). The relationship persisted on covariate adjustment in almost all of 16 different subgroups of the population. The Iowa Women's Study of 34,000 women also found that nut consumption was associated with a 40% decrease in risk of CHD (Kushi et al., 1996; Ellsworth et al., 2001). Attention to the dietary benefits of nut consumption dramatically increased with the report based on the Nurses Health Study (Hu et al., 1998) of 86,016 women that indicated that consumption of five ounces of nuts per week significantly lowered the risk of CHD compared to women who never ate nuts or ate less than one ounce per month. Especially, frequent consumption of peanuts was associated with a low risk of CHD (Hu et al., 1998). Recently, Albert and Willett (2002) studied data from 21,454 male physicians (ages 40-84 years) who were enrolled in the U.S. Physicians' Health Study. Participants were followed up for an average of 17 years. The physicians had no history of heart disease at the beginning of the study in 1982 and were examined for dietary habits including frequency of nut consumption. Over the 17 years of this study, 201 sudden cardiac deaths and 566 heart disease deaths were observed. Compared with men who rarely or never consumed nuts, those who consumed nuts two or more times per week showed a 47% lower risk of sudden cardiac death and a 30% lower risk of total coronary heart disease death. However, the researchers reported that nut consumption was not associated with significantly reduced risk of nonsudden coronary heart disease death or nonfatal heart attack. Due to a large number of participants who varied in age, sex, race, smoking habits, alcohol use, exercise habits, diet and lifestyle, the extended statistical strength for these studies led to the authors conclude that frequent nut consumption protects against CHD.

Clinical studies support epidemiological findings for consumption of peanuts and various tree nuts. The predominant types of fat in nuts are mono- (MUFA) and poly unsaturated fat (PUFA). Diets increasing the quantities of MUFA and PUFA supplied from almond, walnuts, hazelnuts, macadamia and pecan lower total and LDL cholesterol levels which are risk factors for CHD. (Spiller et al., 1992, 1998; Sabaté et al., 1993; O'Byrne et al., 1997; Morgan and Clayshulte, 2000; Zambón et al., 2000; Curb et al.,

2000; Erario et al., 2001; Rajaram et al., 2001; Almario et al., 2001; Iwamoto et al., 2000, 2002; Hyson et al., 2002). The blood cholesterol lowering effects of peanuts were examined in two studies (O'Byrne et al. 1997; Kris-Etherton et al., 1999b). O'Byrne et al. (1997) reported that a low fat diet supplemented with peanuts improved serum lipoprotein profiles when compared to a regular low fat diet for postmenopausal women with high serum cholesterol levels. Kris-Etherton et al. (1999b) compared the CHD risk profile of an average American diet with those of four different cholesterol-lowering diets including high MUFA containing diets with olive oil, peanut oil and peanuts, and peanut butter. The high-MUFA diets lowered total and LDL cholesterols by 10 and 14%, respectively. The effects were similar to the American Heart Association/ National Cholesterol Education Program Step II diet. The high-MUFA diets did not lower HDL cholesterol compared to a 4% decrease in HDL-cholesterol with the Step II diet. The study indicated that high-MUFA diets may be preferable to a low fat diet for lowering serum cholesterol and that high consumption of peanuts and nuts might be a preferable dietary approach to low-fat diets to reduce CHD risk. The effect of nuts on CHD risk may be through multiple mechanisms due to the variety of nutrients and bioactive substances present in nuts (Kris-Etherton et al., 1999a, 2001).

Favorable fatty acid profile (high MUFA and PUFA), folate, vitamin B-6, vitamin E, selenium, sterols, fiber and other phytochemicals in peanuts are considered as possible components to have protective effects on health. Although diets high in both MUFA and PUFA lower blood cholesterol levels when substituted for saturated fatty acids, LDL from subjects fed diets enriched in MUFA are less susceptible to oxidation compared with those from subjects consuming diets high in PUFA (Abbey et al., 1993; Reaven et al., 1991; Hargrove et al., 2001). Peanut lipids meet the criteria of high MUFA content for cholesterol lowering diet. Peanuts are rich in vitamin E which protects against LDL oxidation in vitro (Jialal et al., 1995; Leenen et al., 2002). It is possible that the fatty acid composition and antioxidant content of peanuts could increase resistance of LDL to oxidation. In addition, vitamin E is known to delay the pathogenesis of a variety of degenerative diseases, such as cardiovascular disease, coronary heart disease, cancer, atherosclerosis, aging, Alzheimer disease, inflammatory diseases, neurological disorders, infectious diseases in the aged, cataract and age-related macular degeneration, and to

maintain the immune system (Han and Meydani, 1999; Bramley et al., 2000; Engelhart et al., 2002; Adams and Best, 2002). On the other hand, Wolk et al. (1999) found that a 10 g/day increase in total fiber intake was related with a 19% decrease in CHD risk. One ounce of peanuts provides about 2.4 -2.6 g dietary fiber. Therefore, peanut consumption at 2-7 servings/week will result in 1.3-4.8% decrease in CHD risk. However, more clinical study is needed to clearly explain the observations. In the nutrient data base of the USDA (2002), the sterol content for peanuts is 2.20 g/kg. Especially, beta-sitosterol, found in peanuts and peanut products, is known to inhibit cancer growth and protect against heart disease (Awad et al., 1996, 2000a, 2000b; Normén et al. 2001). Moreover, peanuts are one of the few foods that contain the plant chemical resveratrol (Sanders et al., 2000; Burns et al., 2002) which is associated with reduced cardiovascular disease and reduced cancer risk (Goldberg, 1995; Jang et al., 1997; Bhat et al., 2001). The average amount of resveratrol in one ounce of commonly eaten peanuts is 73μg (Sanders et al., 2000).

A recent comprehensive review by Hu et al. (2001) indicated that replacing saturated fat with unsaturated fat is more effective in lowering risk of CHD than simply reducing total fat consumption. This finding challenges the public's mind that reduced fat or fat-free diet is good for health, especially lowering risk of CHD and inhibiting obesity. Due to high fat content of peanuts, peanuts and peanut products tend to be excluded at mealtime, especially for those who is of interest in obesity. Fortunately, a recent study shows that snacking on peanuts and peanut butter is an effective way to control hunger without leading to weight gain (Kirkmeyer and Mattes, 2000). Following a snack of peanuts or peanut butter, the participant hunger was reduced for two and a half hours. When they were fed typical portions of other snacks, hunger returned within 30 min. Alper and Mattes (2002) observed 1.0 kg of body weight gain in participants who were provided about 500 kcal/day (90 g/day) as peanuts for 8 weeks with no other dietary modification, which is significantly low compared to the value of predicted weight gain (3.6 kg). Other studies also show that regular peanut consumption does not promote weight gain (Alper, 2001; Mattes and Lokko, 2002). A modest quantity (320 cal) of almonds in the diet each day for six months increased body weight only 0.40 kg, which is not statistically or biologically significant ($p \sim 0.09$) (Fraser et al., 2002). These findings

are important because it challenges the belief that eating high-fat foods like peanuts and peanut butter will necessarily lead to weight gain. Moreover, nearly 80% of the fat in peanuts is unsaturated fat (good fat) which may help lower LDL-cholesterol levels in blood.

The number of older adults around the world is increasing at a rapid rate. In 1960, less than one in ten people in the U.S. was 65 years or older. By 2030, it is estimated that one in four Americans will be over 65 years old (Wood et al., 1995). The statistics are similar in other countries as well. By 2025, over 20% of the populations in the United Kingdom, France, Italy, Switzerland, Japan and Sweden will be age 65 or older (Ribbe et al., 1997). With the increase in age, an increase in the incidence of many chronic diseases including diabetes, coronary heart disease, hypertension, high cholesterol and obesity occurs. Lifestyle, especially dietary habits like frequent peanut consumption, can help to combat the effects of aging on health.

5. Roasting

In the United States, about 90% of the peanut crop is processed into peanut butter, salted peanuts, confectionary products and peanut oils (APC, 2000). Peanuts are subjected to dry- or oil-roasting before use. The roasted peanuts are either eaten directly or used in candies, salads, desserts, etc. Roasting peanuts promotes flavor changes that ultimately enhance the overall palatability of the products. Although the roasting reduces moisture content and inactivates enzymes present in raw peanuts which cause lipid oxidation, it can destroy nutrient components and decrease shelf-life (St. Angelo et al., 1979; Oupadissakoon and Young, 1984a; Basha and Young, 1985).

There are a number of ways of roasting peanuts. The most common commercial method used for peanut butter manufacture is dry roasting but oil roasting is also used. A through-circulation type roaster which blows hot air through peanuts passing continuously through a chamber is the most widely used method for dry roasting. Some conventional roasters are heated by natural gas. Abundant carbon dioxide is produced simultaneously in the atmospheric environment during roasting (Woodroof, 1983). Some roasters are designed such that burning fuel does not come in direct contact with the peanuts. Others use electricity as the heat source, which does not create a significant

alteration in the roasting atmosphere. Depending on the type of roaster, unique peanut flavor and other sensory characteristics eventually result. On the other hand, Megahed (2001) stated that microwave heating of peanuts is not recommended, even for short periods. This is due to the formation of oxygenated compounds (epoxy and hydroperoxides) as well as conjugated fatty acids during microwave roasting of peanuts, which decreases in oil stability and accelerates oxidative rancidity in the roasted peanuts.

Color

The characteristic color results from the Maillard reaction (sugar-amino acid reaction) that produces melanoidin compounds (Hodge, 1953). The golden brown color of the melanoidins intensifies with increasing temperature and time of roasting. A secondary source of color formation in roasted peanuts is caramelization of sugars (Mason et al., 1966). Color is often used for the quality control of the roasting operation. However, the selection of a roast on the basis of color alone can cause flavor defects (Young and Trigano, 1986) because color is affected by other factors including peanut size, maturity, moisture content, etc. Smaller sized peanuts, which tend to be more immature, generally form darker roast colors. One possible reason for this is that immature nuts are high in sucrose levels and the darker color may be due to increased caramelization reactions (Pattee et al., 1982a). In addition, peanuts stored at higher moisture content levels (9% vs. 6%) produce darker color peanut butter with reduced flavor quality (Pattee et al., 1982b). This research also showed that the time required to obtain a specific roast color is dependent on peanut cultivars.

Colors of peanuts are mainly expressed in Hunter Lab and CIE L*a*b*. Young and Trigano (1986) studied the effect of time and temperature of roast on color development in peanuts by using the L*a*b* uniform color space scale (L*=lightness; a*=red-green; b*=yellow-blue). For temperature in the 160°C range, a constant L* value was maintained for the first few minutes of roasting, followed by a sharp decrease. The a* values initially decreased but then rapidly increased, producing a general reddening of the product. The b* values slightly increased, suggesting that the peanuts yellowed as roasting progressed. Roasting at higher temperatures (180 and 200°C) generally showed the same trends, but the rates of color change were more rapid. Moss and Otten (1989)

found a correlation between the yellow-blue (b*) color scale and the moisture content ratio. Pattee et al. (1991) reported that "Hunter L = CIELAB L* - 7" can be used to convert L* values to L values in the L* range from 52 to 65. Peanuts should be roasted to L* values of 58-59 or L values of 51-52 when an optimum roasted peanut attribute is of primary interest. Peanut-roasting studies usually give a time-temperature protocol or refer to degree of roast as "light", "medium", or "dark" with color measurement (Buckholz et al., 1980; Oupadissakoon and Young, 1984).

Flavor

Raw peanuts have a characteristic odor and flavor described as 'beany' or 'green'. Hexanal, acetaldehyde, methanol, pentane and ethanol are major contributors to the roasted peanut flavor (Pattee et al., 1970). Major volatiles from raw peanuts are produced upon rupture of the tissue by enzymatic reaction (lipoxygenase and alcohol dehydrogenase) (Pattee et al., 1974; Singleton et al., 1976). The profile of volatiles of raw peanuts can be changed during maturation and the level of enzymes (Pattee et al., 1970). Alcohol dehydrogenase is present at maximal amounts when levels of ethanol and acetaldehyde were high. Lipoxygenase is linked with the production of hexanal and pentane. Ethanol, pentane, acetone, dimethyl sulphide, methyl acetate, 2-methyl propanal, 2-butanone, 2- and 3-methyl butanal, pentanal, n-methyl pyrrole, dimethyl sulphide, hexanal, methyl formate, 2-propanal, and propanal have been isolated and identified as the flavor of raw peanuts (Lovegren et al., 1982; Pattee et al., 1965, 1969, 1970; Singleton et al., 1971; St. Angelo et al., 1984).

During roasting, the enzymes in raw peanuts are destroyed with formation of the unique nutty flavor. A desirable roasted flavor results from the Maillard reaction, including the Strecker degradation. Free amino acids and reducing sugars, released during roasting, are converted to pyrazines and carbonyls which contribute to the roasted peanut flavors (Newell et al., 1967; Mason et al., 1966, 1967, 1969; Koehler et al., 1969; Johnson et al., 1971a, 1971b; Shu and Waller, 1971). More than 300 components have been isolated from roasted peanut (Ahmed and Young, 1982, Mason et al., 1966, 1967; Ho et al., 1982; Rodriguez et al., 1989; Chiou et al., 1991a, 1991b; El-Kayati et al., 1998). Monosaccharides are involved in the formation of browning reaction products with free

amino acids. Aspartic acid, glutamine, asparagine, histidine and phenylalanine are associated with typical roasted flavor, while threonine, tyrosine, and lysine are considered the precursors of atypical roasted flavor (Newell et al., 1967).

Roasting time and temperature are the most significant factors influencing the formation of flavors (Buckholz et al., 1980; Magaletta and Ho, 1996). Peanuts roasted at lower temperatures for longer periods of time are known to have the best flavor and a longer shelf life. It may be due to the more even temperature distribution resulting in a higher probability of total enzyme inactivation. In addition, maturity, harvest season, atmospheric conditions during roasting, initial moisture content, peanut composition, and the type of roaster can affect strength and variety of flavors of roasted peanuts (Newell et al., 1967; Mason et al., 1969; Bucholtz et al., 1980; Oupadissakoon and Young, 1984; Sanders et al., 1989; Rodriguez et al., 1989; Chiou et al., 1991a, 1993; Ku et al., 1998).

Composition

In general, the longer the roasting time, the more extensive are browning reactions, resulting in decreases of free amino acids and soluble carbohydrates (Chiou et al., 1991a). McOsker (1962) observed that roasting decreased the availability of lysine, threonine, and methionine by 15, 11 and 10%, respectively. Basha and Young (1985) also showed a decrease in the methionine-rich proteins and aggregation of arachin proteins by roasting. Damame et al. (1990) reported that roasting significantly decreased methionine, tryptophan and *in vitro* protein digestibility and increased the soluble proteins. Acid soluble proteins are more susceptible to thermal breakdown than acid insoluble proteins during roasting (Basha and Ying, 1998). Although the proteins are denaturated, apparently their nutritive value is unchanged by moderate heating. Total sugars decrease when peanuts are subjected to a heavy roast, while the starch content is not significantly altered. Such constants of the oil as iodine number, saponification number, acetyl number, and free fatty acids do not change appreciably during roasting. The thiamin present in peanuts is destroyed up to 90%, while niacin, choline, and riboflavin are little affected by roasting (Fournire et al., 1949; Willich et al., 1952). Iron content in oil prepared from roasted peanuts decreased compared to oil from non-roasted peanuts (Chen and Chiou,

1995). On the other hand, Özdemir et al. (2001) showed that roasting significantly decreased thiamin, riboflavin and total amino acid levels in hazelnuts.

Structure

The raw peanut kernel consists of two cotyledons and the germ. The cotyledon consists of epidermis, vascular and parenchymal tissue. The epidermal tissue is a layer of cells compressing the cotyledon surface which is underlayered by the vascular tissue. The parenchyma tissue constitutes the greatest part of the kernel and contains large, isodiametric cells. Each parenchyma cell contains lipid bodies (spherosomes), aleurone protein bodies and starch granules. Cytoplasmic network surrounds these cellular orgarnells. Young and Schadel (1990, 1991, 1993) documented the microstructural changes in peanut cotyledons during roasting (oven roasting and oil cooking) with microscopy. In the raw peanut cotyledon, the cytoplasmic network surrounds the major storage reserve bodies of protein, lipid, and starch. Lipid bodies (spherosomes) are characterized as particles with a size of 1.0-2.0 μm in diameter which is bounded by a limiting membrane (Jack et al., 1967; Neucere and Hensarling, 1973). After roasting at 160°C for 16 min, a loss of this cellular organization of cytoplasmic network was observed (Young and Schadel, 1990). During roasting, cytoplasmic compartmentalization of the oil is lost in the majority of the cells as oil sacs rupture. Protein bodies distend as heat turns the small amount of water present in the cells into steam. However, starch granules remain relatively unaltered by this steam which is insufficient to gelatinize the individual granules. The cells of the rounded outer surface become slightly swollen as heat destroys the structure of the cytoplasmic network. The conduction of heat from the outer surface of the cotyledon to the inner surface destroys some of the middle lamellae of cell-to-cell junctions (Young and Schadel, 1990). At 160°C, thermal modification in peanut cotyledon microstructure is more severe after oil cooking than oven roasting (Young and Schadel, 1993). After oven roasting at 160°C for 19 min, greater than 50% disruption of the cytoplasmic network, severe protein body distension, and the presence of cell wall separation were observed along the middle lamellae (Young and Schadel, 1993). These physical changes of peanuts during roasting contribute much to their

susceptibility to oxidation (St. Angelo and Ory, 1975b; Yuki et al., 1978; Damame at al., 1990; Young and Schadel, 1991).

Oxidative stability

Roasted peanuts have shorter shelf life than raw peanuts (St. Angelo and Ory, 1975b; Yuki et al., 1978). This is due to chemical and physical changes of peanuts during roasting - destruction of natural antioxidants, breakdown of fatty acids, disruption of lipid bodies, membranes and cellular compartmentalization (Woodroof and Leahy, 1940; Damame et al., 1990; Young and Schadel, 1991). According to Woodroof and Leahy's study (1940) on microstructure of peanuts, oil droplets were fused into larger and fewer droplets within the cells and a small amount of oil escaped into the cell as free oil by dry roasting. The work suggested that the free oil in peanut kernels is more readily in contact with oxygen and, then, more susceptible to oxidation. Later, Young and Schadel's extensive work (1990, 1991, 1993) on the microstructure of oven-roasted peanuts confirmed the earlier research that oil in peanuts can be exposed to air and become more susceptible to oxidation, resulting in a shorter shelf-life for roasted peanuts compared to raw peanuts. Oil roasting is more detrimental to storage stability of peanuts as compared to dry roasting (Damame et al., 1990). More severe thermal modification in peanut cotyledon microstructure by oil roasting than oven roasting may explain shorter shelf life of oil roasted peanuts (Young and Schadel, 1993).

On the other hand, Cheng et al. (1987) and Huang et al. (1988) reported that the longer the roasting time of peanut kernels, the higher the oxidative stability of oils extracted from the peanuts. Chiou (1992) also showed that the oil prepared after 90 min of roasting peanuts was slightly more stable to oxidative changes than oils prepared from peanuts roasted for less time.

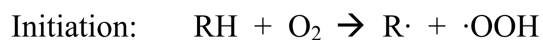
6. Lipid oxidation

Peanuts are considered semiperishable. Storage up to 5 years and longer under optimum storage conditions without significant deterioration is possible. Under undesirable conditions they may rapidly become spoiled due to mold, insects, discoloration, absorption of foreign flavors, staleness, or rancidity (Woodroof, 1983). The

relatively high unsaturated oil content of the kernel increases potential for oxidative deterioration. Rapid lipid oxidation is due to a number of causes: light, air, heat, trace metals, enzymes, microorganisms, and even the presence of free fatty acids that can act as free radical catalysts (Lea, 1962). The oxidation of fatty acids in peanuts results in off-flavors and quality loss in peanut products.

Mechanism of Autoxidation

The mechanism of autoxidation is characterized by three main phases: initiation, propagation, and termination. The initiation occurs as hydrogen is abstracted from unsaturated fatty acid of phospholipids in the presence of catalyst such as heat, light, and metal ions, resulting in forming a lipid free radical (R·). The lipid free radical in turn reacts with molecular oxygen to form a lipid peroxy radical (ROO·) which reacts with more unsaturated lipids to form lipid hydroperoxide (ROOH) in the propagation step. This step also provides new free radicals (R·), making it a self-propagating chain reaction. This process continues and destroys lipids and neighboring molecules with which radical react including proteins and nucleic acids (Halliwell and Gutteridge, 1989). The hydroperoxides ultimately break down to aldehydes, ketones and alcohols which are responsible for the characteristic flavors often described by 'rancid'. In the termination step, the formed radicals react with each other, resulting in formation of polymers such as dimmers and trimers. The rate of autoxidation increases with the degree of unsaturation (linolenate > linoleate > oleate) (Gardner, 1989). Chain breaking antioxidants such as vitamin E interact with the lipid free radicals, thus, increasing the lag phase of the reaction.

Initiation: 
$RH + O_2 \rightarrow R\cdot + \cdot OOH$

$RH \rightarrow R\cdot + \cdot H$

Propagation:
$R\cdot + O_2 \rightarrow ROO\cdot$

$ROO\cdot + RH \rightarrow ROOH + R\cdot$

Termination:
$R\cdot + R\cdot \rightarrow R\text{-}R$

$ROO\cdot + R\cdot \rightarrow ROOR$

Through *in vitro* studies, free fatty acids are shown to oxidize faster than triacylglycerol (Labuza et al., 1969), while membrane lipids are oxidized faster than emulsified triacylglycerols (Slabyj and Hultin, 1984), apparently because propagation is facilitated by the arrangement of phospholipids in the membrane. Proximity of the phospholipids to catalytic sites of oxidation (enzymic lipid peroxidation, heme-containing compounds), in addition to the high degree of polyunsaturation in phospholipids, may contribute to the importance of membrane lipids in tissue oxidation (Erickson, 1998). While phospholipids are the major contributors to the development of warm-over flavor in meats (Ang, 1988; Wu and Sheldon, 1988), a stronger negative correlation was found in pecans between head space hexanal and its precursor fatty acid from phospholipids fraction than from the triacylglycerol fraction or free fatty acid fraction (Erickson, 1993).

Enzymatic Oxidation and Lypoxygenase (LOX)

LOX prefers free fatty acids as substrates, although there are LOX isoenzymes which can react with triglycerides. LOXs contain one atom of nonheme iron. The native inactive LOX can be activated by oxidation of the iron atom from Fe^{2+} to Fe^{3+}. The LOX-catalyzed reaction is initiated by the stereospecific removal of hydrogen from the C-11 methylene group when 18:2 is used as a substrate under both aerobic and unaerobic conditions (Gardner, 1991). LOX, in turn, catalyzes the addition of molecular oxygen to a *cis, cis*-1,4-pentadiene unsaturated fatty acid releasing a fatty acyl hydroperoxides.

Under aerobic condition, LOX transforms fatty acids containing a *cis, cis*-1,4-pentadiene system into hydroperoxy-octadecadienoic acids. Under oxygen deficient conditions, the cycle catalyzes the hemolytic cleavage of the conjugated hydroperoxides to alkoxyl radicals, which are stabilized by further reaction such as oxidation, hydrogen abstraction, β-cleavage and rearrangement to various products (Chow, 1991). Most lipoxygenases in plants form 13(S)-hydroperoxy-*cis*-9,*trans*-11-octadecadienoic acid (13S-HPOD) and 9(S)-hydroperoxy-*trans*-10,*cis*-12-octadecadienoic acid (9S-HPOD) (Figure 1-1). The hydroperoxides can be broken down by enzyme or non-enzyme catalyzed scission reactions to yield specific chain length volatile compounds. An overview of the lipoxygenase or octadecanoid pathway of plants is shown in Figure1-2 (Gardner, 1995).

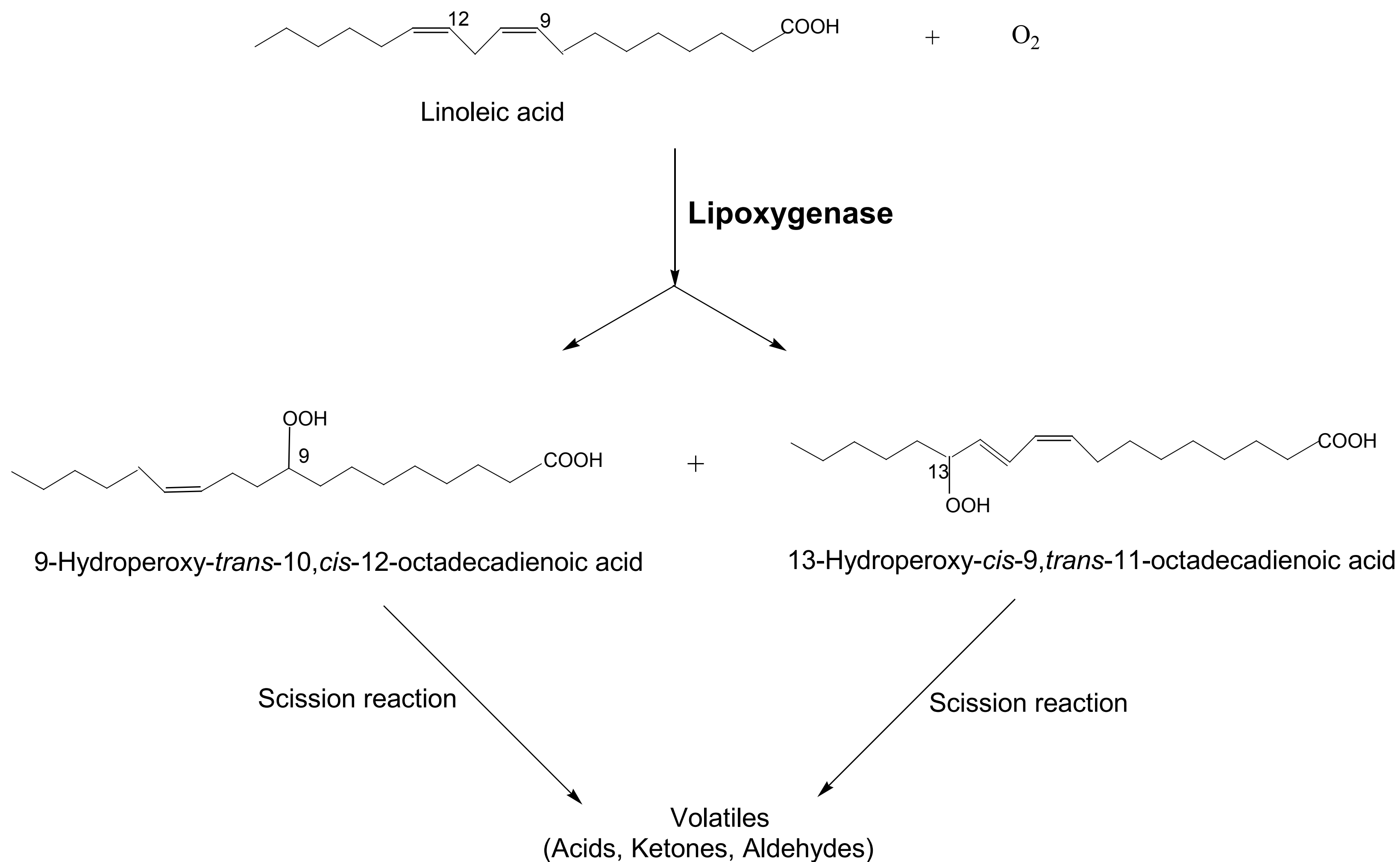


Figure 1-1. Preparation of Hydroperoxides from Linoleic Acid by Soybean Lipoxygenase (Siedow, 1992).

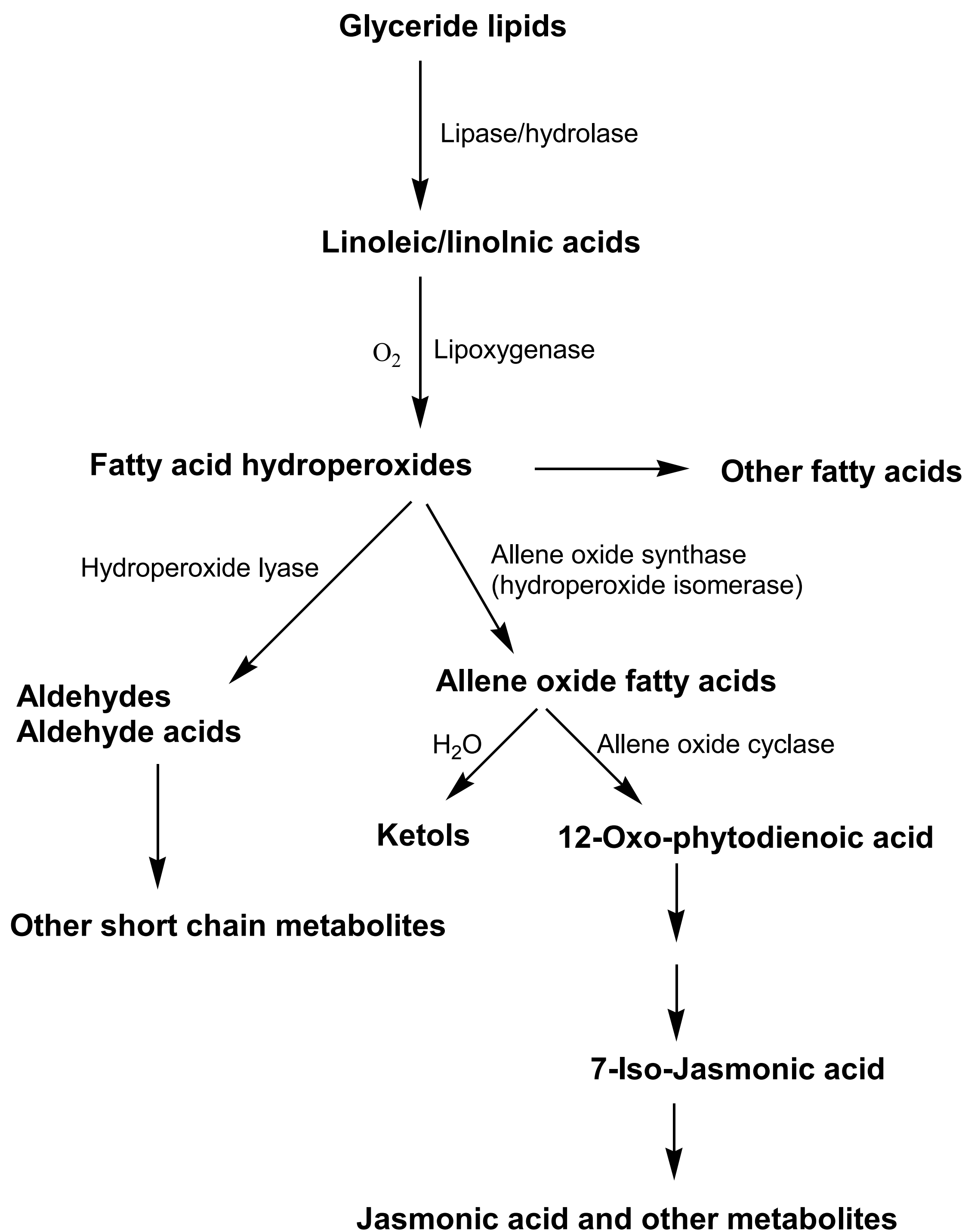


Figure 1-2. An Overview of the Lipoxygenase Pathway of Plants (Gardner, 1995)

LOX products including hydroperoxides and their free radical derivatives are very reactive compounds, causing disruption of membrane integrity (Gardner, 1983), inactivation of proteins and amino acids (Gardner, 1979), and degeneration of DNA by hydroperoxide attack on nucleotides (Inouye, 1984). Theses properties have led to speculation that lipoxygenase participates in the senescence process in plants.

Assay methods for lipid oxidation

The extent of lipid oxidation can be followed by several methods. The three most widely used methods for determining lipid oxidation in oil containing foods are measurements of the peroxide value (PV), the conjugated diene hydroperoxides (CDHP) and the thiobarbiturate (TBA) determination of malonaldehyde formation. Analysis of primary oxidation products has been based on PV and CDHP while the TBA test is used to determine secondary oxidation products. St. Angelo et al. (1975) compared two methods for PV and CDHP to determine lipid oxidation in peanut butter. There was a highly correlative linear relationship (PV = -18.46 + 4.67(CDHP), r^2=0.98) between the two methods. CDHP is quicker, more accurate, and simpler than the PV method. The TBA method colorimetrically measures a reddish color formed by the reaction of oxidation products with thiobarbituric acid. This method is valid for analysis of oils, but some difficulties are encountered by reaction of the oxidation products with protein or carbohydrates rather than with the reagent (Braddock and Dugan, 1973). A rapid static headspace gas chromatographic (GC) method developed by Young and Hovis (1990) can be used for measuring hexanal, a major volatile compound released during oxidation of peanuts. The total oxidation (TOTOX) value is often considered by many investigators as a useful indicator of lipid oxidation in oils. The TOTOX combines evidence about the past history with the present state of the samples which are represented by anisidine value (AnV) and PV, respectively (Rossell, 1983). AnV determines the amount of aldehydes, secondary oxidation products and the TOTOX value is calculated by the equation of TOTOX = 2PV + AnV.

Lipid oxidation measurements for oil seeds are conducted on Soxhlet extracts, pressed oil, and solvent extracts using hexane, isopropanol, or mixture of chloroform and methanol. Erickson (1994) stated that in case of lipid oxidation measurements conducted

on Soxhlet extracts or pressed oil fractions, lipid oxidation of membrane lipids, phospholipids, which have been implicated in early stages of oxidation (Erickson, 1993), would not have been accounted for. In general, phospholipids of oil seeds represent a very small percentage (< 1%) of the total lipid while triacylglycerols are high (> 96%) (Sanders, 1980; Erickson, 1993). Oxidation of phospholipids may be minimal in comparison to oxidation of triacylgycerols. Even though total lipids are extracted, the phopholipid fatty acid composition is overshadowed by the large quantities of storage lipid. Therefore, the contribution of the phospholipids is often ignored in storage studies. On the other hand, Erickson et al. (1994) reported that triacylglycerols may be the primary site of attack in raw pecans, and phospholipids, the primary site in roasted pecans.

Raw peanuts

Peanuts require special handling during cultivation, harvesting, and processing (digging, drying, combining, transporting, blanching). In each of these steps, they are susceptible to damage that can activate enzymes. Oxidation of unsaturated fatty acids in raw peanuts is caused mainly by lipoxygenase (St. Angelo and Ory, 1975b; St. Angelo et al., 1977a). The presence and activity of lipoxygenases in raw peanuts has been well documented (Siddiqi and Tappel, 1957; St. Angelo and Ory, 1972; Nelson et al., 1977; St. Angelo et al., 1979). Peanut lipoxygenase catalyzes only *cis*, *cis*-1,4-pentadiene bonds which are found in linoleic, linolenic and arachidonic acids, forming active *cis-trans* conjugated hydroperoxides (St. Angelo et al., 1977a). The hydroperoxides formed by the action of lipoxygenase on polyunsaturated fatty acids or triglycerides can be decomposed into acids, ketones, aldehydes, or other substances during processing or storage. These degradative products can then react with amino acids and proteins present in peanuts, resulting in formation of off-flavor and decreased nutritive values (Ory and St. Angelo, 1982). Linoleic acid can be oxidized to form monohydroxyperoxides that are precursors for volatile decomposition products such as nonanal, octanal, decanal and hexanal (Min et al., 1989). Oxidized linoleic acid can react with lysine and threonine. Changes in the saturation level of fatty acids by lipid oxidation might influence membrane integrity leading to increased permeability and leakage of cellular metabolites observed in deteriorated peanut seeds during storage (Nautigal and Zala, 1991).

Roasted peanuts

Lipoxygenase in raw peanuts is mostly inactivated during roasting (Chiou et al., 1991a; Chen and Chiou, 1995). Mitchell and Malphrus (1977) showed that the rate of hydroperoxide formation by enzymes in stored whole shelled peanuts first increased and then decreased with the time of steaming prior to storage. Lipoxygenase was not found in whole shelled Spanish peanuts after steaming for 2 min at 100°C. A 50% decrease of lipoxygenase activity was observed in Virginia-type peanuts after immersion in water at 79°C for 1.5 min (Branch et al., 1987). After roasting, however, lipid oxidation is catalyzed primarily by nonenzymic catalysts (St. Angelo and Ory, 1972; St. Angelo et al., 1977a, 1979). Storage studies on raw and roasted peanuts usually indicate that roasted whole nuts are less stable to oxidation than raw nuts. This is due to both chemical changes, such as destruction of natural antioxidants and breakdown of fatty acids (Damame at al., 1990), and physical changes during roasting, such as disruption of lipid bodies and cellular compartmentalization (Young and Schadel, 1991). The peroxide value is often used to follow shelf-life of roasted peanuts. The limiting peroxide values critical for acceptibility for roasted peanuts or peanut oil is reported to 20-30 meq/kg (St. Angelo et al., 1977b; Balasubramanyam et al., 1983; Narasimhan et al., 1986; Evranuz, 1993). Evranuz (1993) calculated 28 days of shelf life for roasted peanuts stored at 15°C with a peroxide value of 25 meq/kg oil. The short shelf life of shelled and roasted peanuts can be extended by adequate packaging.

7. Factors Affecting Storage Stability of Peanuts

Woodroof (1983) listed the following rules as requirements for successful storage of raw peanuts:

1. The peanuts should be of high initial quality, free of mold, insects, discoloration, rancidity, or off-odors.
2. The temperature in the storage room should be low.
3. The relative humidity should be 65-70%.
4. The storage atmosphere should be odorless and well circulated.
5. Moisture condensation on the peanuts should be prevented upon removal from the storage area.

Generic, cultural, handling, processing, and composition variations in raw peanuts used to prepare peanut products and environmental conditions under which they are exposed during distribution influence the stability of peanut products. Other factors influencing storage stability of peanuts and peanut products include variety (Young et al., 1974a; Shewfelt and Young, 1977; Wallerstein et al., 1989), maturity at harvest (Young et al., 1972; McNeill and Sanders, 1998), quality of the raw stock (St. Angelo et al., 1979), market grade and seed size (Pattee et al., 1982b; Pearson and Slay, 1983; Mozingo et al., 1988), packaging materials (Fonseca et al., 1995), and production conditions.

Fatty acid composition

Most importantly, the shelf life of peanuts highly depends on oil stability. The rates of oxidation of C_{18} fatty acids are approximately 1:10:100:200 for stearic (18:0), oleic (18:1), linoleic (18:2) and linolenic (18:3) acids, respectively (Lin, 1991). There is a negative correlation between the percentages of oleic and linoleic acids (Worthington and Hammons, 1971; Anderson et al., 1998), since linoleic acid is produced from oleic acid. Consequently, the amount of linoleic acid present or the oleic/linoleic acid (O/L) ratio in oil affects oil stability. Peanuts consist mainly of unsaturated fatty acids (80%), of which 30-50% is oleic and 20-45% is linoleic acids in percent of total fatty acids (Hammond et al., 1997). Several investigators reported that the O/L ratio in peanut oil is highly related with oil stability (Fore et al., 1953; Worthington et al., 1972; Mercer et al., 1990; Chiou et al., 1995). Liu and White (1992) found a significant positive correlation between linolenic acid content and peroxide value (PV) and high negative correlations between linolenic acid and flavor quality of oils stored at 60°C. A highly negative correlation of 0.924 was found between linoleic acid content and oil shelf-life for 66 cultivars of peanuts grown during 1 season (Holly and Hammons, 1968). However, the correlation coefficients were quite variable from year to year. The O/L ratio is regarded as the major factor that influences stability of peanut oil, which, in turn, affects the overall quality and stability of peanuts and peanut products during storage (Mugendi et al., 1998). Overall, to obtain peanuts and peanut products with a longer shelf-life, the O/L ratio should be high with less than 25% linoleic acid (Oupadissakoon, 1980).

Fatty acid composition of peanuts is influenced by cultivar, stage of maturity, geographic location, season, and environmental conditions (Pickett and Holley, 1951; Worthington and Holly, 1967; Worthington and Hammons, 1971; Worthington et al., 1972, Young et al., 1974a, 1974b; Holaday and Pearson, 1974; Brown et al., 1975; Branch et al., 1990; Chiou et al., 1995; Grosso and Guzman, 1995a, 1995b; Isleib et al., 1996). Worthington and Hammons (1971) compared the fatty acids in the three primary peanut types and reported a higher O/L ratio for the Virginia variety than for the Spanish with the runner being intermediate. Ahmed and Young (1982) suggested the selection or development of raw peanuts with low levels of linoleic acid to extend shelf-life of peanut products. O'Keefe et al (1993) showed that high-oleic peanut oil has much greater oxidation stability as compared to normal peanut oil. Shelf-life of high oleic peanuts (80% oleic acid) is greater than normal peanuts (53-55% oleic acid) (Braddock et al., 1995; Mugendi et al., 1998).

P*rocessing*

Blanching, the process of removing the skins, significantly affects the shelf life of raw and roasted peanuts (Woodroof, 1983). Shelf-life of unblanched roasted and unblanched salted and roasted peanuts is shorter than peanuts roasted after blanching (Shewfelt and Young, 1977; Branch et al., 1988). Water blanching improves the oxidative stability of roasted peanuts; whereas, spin blanching decreases it (St. Angelo et al., 1977b). The advantage of water blanching is that peanuts are not heated to destroy antioxidants. The spray of hot water used for water blanching dissolves some of the surface protein, and subsequent drying forms a glaze on the surface which is a protection against oxidation and mechanical injury (Woodroof, 1983). For spin blanching, steam is used to loosen the skins, which are removed by rapidly revolving spindles. St. Angelo et al. (1977b) reported that unblanched raw peanuts had longer shelf-life than blanched raw peanuts while unblanched roasted peanuts showed shorter shelf-life than blanched roasted peanuts. They suggested that if the peanuts are stored without vacuum-packaging after roasting, they should be water-blanched. If raw peanuts are stored, then they should be either unblanched or spin-blanched. Different shelf-lives of raw and roasted peanuts treated by blanching are shown in Table 1-3. Branch et al. (1987) reported that peanuts

Table 1-3. Shelf-Life of Peanuts in Literature

Samples	Shelf-life	References
Unblanched, roasted	59 days[a]	St.Angelo et al. (1977)
Spin-blanched, roasted	100 days[a]	St.Angelo et al. (1977)
Water-blanched, raw	110 days[a]	St.Angelo et al. (1977)
Water-blanched, roasted	153 days[a]	St.Angelo et al. (1977)
Spin-blanched, raw	>200 days[a]	St.Angelo et al. (1977)
Unblanched, raw	>200 days[a]	St.Angelo et al. (1977)
Unblanched, roasted, 4C storage	56 days[a]	St.Angelo and Ory (1975)
Unblanched, raw, 4C storage	>>200 days[a]	St.Angelo and Ory (1975)
Roasted, salted, shelled peanuts	24-42 days	Hoover and Nathan (1980)

[a] Time to reach a CDHP value of 8

immersed in hot water at 79°C for 90 s decreased lipoxygenase activity and appeared to be more stable during eight months of storage at non-refrigerated conditions compared to untreated peanuts. Among the blanching methods, dipping peanuts in H_2O_2 (hydrogen peroxide) solution prior to salting and roasting improves the oxidative stability of unblanched roasted and salted peanuts (Evranuz, 2000). Its effectiveness on increasing the stability of peanuts is dependent on the concentration of the H_2O_2 solution, moisture content of products and properties of packaging materials. Hydrogen peroxide when used to facilitate the removal of the peanut skins normally acts as a radical initiator in the autoxidation of unsaturated oils. According to the theory by Paulsen et al. (1976) and Clavero et al. (1993), hydrogen peroxide would react with catalase in the testa to form water and oxygen. The decreased rate of oxidation by pretreatment with hydrogen peroxide solutions in Evranuz's study (2000) may be explained by a lack of a prooxidant effect of hydrogen peroxide and reduction of peroxidase and catalase activities. The decreased prooxidative effects of denaturated heme-containing enzymes (peroxidase and catalase) by heat or chemical were observed (St. Angelo and Ory, 1975; Lingnert et al., 1989). Coxon et al. (1987) assumed to be due to the oxidation of Fe^{+2} to Fe^{+3} in the presence of hydrogen peroxide which made the potential catalysts less effective, resulting in reduced oxidation rates.

Environmental conditions during processing and storage can affect shelf-life of peanuts. Chiou et al (1991a) reported that peanuts roasted under O_2 for 25 min were observed to be the most stable, followed in order by peanuts roasted without aeration, under nitrogen, under air, and under carbon dioxide during initial stages of an oven test at 62°C. Pominski et al (1975) showed that after roasting, partially defatted peanuts cooled in air for 3.5 min and held under nitrogen until vacuum packaging have longer shelf life than those subjected to air through out the process.

Partially defatted peanuts are less stable to oxidative changes than full-fat peanuts although their oil content is lower compared to normal peanuts (Adnan et al., 1981; Brannan et al., 1999). To produce partially defatted peanuts, blanched peanuts are first pressed to remove 50-60% of the oil in peanuts. The pressed peanuts are immersed in hot water for a few min to facilitate expansion back to their original size, which is called reconstitution. After draining, the peanuts are roasted with salt or other favorable

ingredients. During mechanical pressing of peanuts, the subcellular organelles and membranes of the peanuts are ruptured, possibly promoting an interaction between lipoxygenase and oil (Neucere and Hensarling, 1973). The reconstitution operation effectively inactivates the enzyme, preventing enzyme-catalyzed oxidation (Adnan et al., 1981). However, high moisture content in defatted peanuts after reconstitution may contribute to decreased storage stability (Brannan et al., 1999).

Gamma irradiation is used to inhibit germination of peanuts. Levels of 2.5 and 5.0 KGy are effective in reducing the population of natural mold contaminants and retarding the outgrowth of *Aspergillus parasiticus* which produces aflatoxin (Chiou et al., 1990). Chiou et al. (1991c) indicated that oil in peanuts subjected to a gamma irradiation dose, e.g., 2.5 or 5.0 KGy, was less susceptible to oxidation measured by peroxide and conjugated hydroperoxide formation.

Packaging and environmental factors

Storage time between harvesting and consumption of peanuts may last from several months to more than one year. Optimum storage conditions and packaging materials can minimize enzymatic and non-enzymatic oxidation in nuts and insure high quality. Moisture content of peanuts is probably the most critical factor affecting stability during storing and marketing of peanuts and peanut products. At digging, peanuts have a moisture content of 35-55%. The peanuts are dried in the field until moisture is lowered to 18-25% (Woodroof, 1983). Prior to storage, raw peanuts must be dried to a safe level for maintenance of quality. Mechanical drying is commonly used to reduce the moisture to about 10%. Peanuts delivered from the farm are 10 to 10.5% moisture. During storage, peanuts reach an equilibrium moisture content for a given temperature and relative humidity (RH). Optimum storage conditions for farmer's stock peanuts are considered to be 7 to 12°C and 50 to 70% RH which should result in 6.5 to 7.5% of peanut moisture content (Smith and Davidson, 1982). Higher moisture levels induce mold development and lower levels cause brittleness and splits (Woodroof, 1983).

Moisture content that ensures the water monolayer coverage on food has a protective action against lipid oxidation, resulting in minimum lipid oxidation rate (Labuza, 1971). Below the monolayer moisture content, oxygen is more accessible to

lipids; and above it, chemical reactions occur more quickly. Evranuz (1993) found that the moisture content necessary to provide a monolayer for roasted peanuts is 2.1% that corresponded to a water activity of about 0.3 (or 30% equilibrium relative humidity (ERH)). During storage of unblanched salted roasted peanuts, the rate of peroxide formation decreased near or above the monolayer moisture level (2.1%) and then increased with further increase in moisture content. Maté et al. (1996a) also showed that lowering the water content below the monolayer value (from 30 to 21% ERH) affects lipid oxidation much more than increasing the water content above the monolayer value (from 30 to 53% ERH). On the other hand, Baker et al. (2002) reported that the oxidation values were the highest in the high oleic peanuts held under water activity of 0.67, followed by 0.12, 0.52, 0.44 and 0.33, respectively.

It is possible to store in-shell peanuts in silos if humidity and moisture levels are satisfactory, but controlled temperature and humidity storage is more reliable. Raw shelled peanuts require more careful and controlled storage than the in-shell peanuts to retain peanut quality. They are more vulnerable to insect damage. Low storage temperature is beneficial in lowering the undesirable nutritional and quality changes in the peanut kernels (Davison et al., 1982; Damame et al., 1990). Refrigerated storage with controlled humidity is now a standard practice for the commercial wholesale storage of peanuts. Almost all of the peanuts in refrigerated storage are in shelled form. However, temperatures around 0°C for peanut storage environments may not be appropriate in many areas due to the high cost.

Storage of unshelled nuts is impractical due to bulkiness of the shells which require extra space. Shelled peanuts show shorter shelf-life than unshelled peanuts. Unshelled peanuts stored at 21°C should retain edible quality for 6 months while shelled peanuts could be stored only for 4 months at 21°C (Woodroof, 1983). Woodroof et al. (1947) reported that both shelled and unshelled peanuts stored at -18°C for 2 years retained high edible quality. Stansbury and Guthrie (1947) also showed that at 1°C, unshelled peanuts could be stored for more than 2 years. Raw shelled peanuts could be safely stored at 4°C for at least 6 months before enzyme-catalyzed oxidation begins (St. Angelo et al., 1977a). Once this oxidation starts, storage at 4°C does not stop the degradation. At temperatures above 9°C, insect infestation is a problem in stored peanuts,

especially shelled peanuts (Thomson et al., 1951). To minimize insect damage, moisture levels must be maintained below 9% (Redlinger and Davis, 1982**).**

For roasted peanuts, even refrigeration does not retard lipid oxidation in the presence of air. In St. Angelo and Ory's study (1975b), roasted peanuts stored at 4°C showed that rate of oxidation increased greatly during 12 months, from a initial low value of 8 to 40 CDPH units in 50 weeks. There was no sign of lipid oxidation over the first 28 weeks in raw peanuts. They indicated that when oxidation was initiated in raw peanuts, the rate was still much slower (1/8) than that for roasted peanuts. Labuza (1982) reported that a 20°C drop in temperature increased the shelf life of fried nuts by two to three times. Evranuz (1993) reported the Q_{10} value of 1.6 for oxidation in unblanched salted roasted peanuts. He also calculated 28 days at 15°C, 10 days at 25°C, and 11 days at 35°C for shelf-lives of roasted peanuts under the assumption that the products remain acceptable until the PV reaches 25 meq/kg oil. Low temperature storage can reduce oxidative degradation of lipid in peanuts; but, lowering temperature alone is not sufficient to protect against oxidation.

Oxygen concentration is one of the most important environmental factors affecting lipid oxidation (Labuza, 1971; Vercelloti et al., 1992). The rate of oxidation is independent of oxygen concentration at very high oxygen partial pressure, while it is proportional to oxygen concentration at low oxygen partial pressure (Labuza, 1971). Controlled atmosphere storage and modified atmosphere packaging involve keeping products in atmospheres that usually have lower O_2 and higher CO_2 concentrations than ambient air. During roasting, some of the water in peanut tissue is removed, creating channels through which oxygen can rapidly migrate (Fritsch, 1994). Thus, especially, roasted peanuts must be protected against O_2. Nitrogen-flushing or vacuum packaging with proper containers such as metal cans, glass jars, and metalized films are all means used in industry to prevent oxygen contact to peanuts, resulting in increased shelf-life. Whey protein isolate (WPI)-based edible films have low O_2 permeability at low to intermediate relative humidity (McHugh and Krochta, 1994). Maté and Korchta (1996) coated peanuts by dipping them into an increased-viscosity WPI solution, followed by air-drying. They showed that WPI-based edible coatings are also good oxygen barriers on the peanut surface and could delay considerably oxygen uptake of dry roasted peanuts.

Other studies support the effect of WPI coating on stability of roasted peanuts (Maté and Korchta, 1998; Maté et al., 1996a, 1996b; Lee and Krochta, 2002; Lee et al., 2002). The use of coating on the peanuts acting as an oxygen barrier, combined with a simpler plastic film such as low density polyethylene acting as a moisture barrier, represents an alternative packaging system. On the other hand, chocolate coated peanuts have higher oxidation rates compared to uncoated peanuts at the same water activity (Reed, 2000).

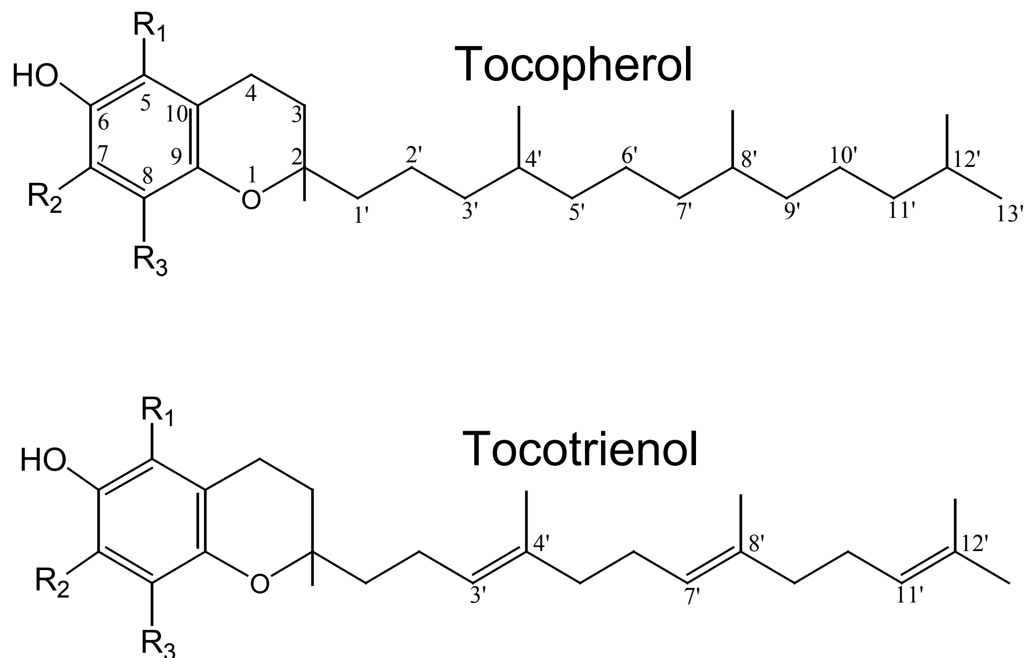
Shelf-life of nuts is extended by application of a coating containing antioxidants (Alikonis, 1961; Anon, 1965; Shea, 1965; Hoover and Nathan, 1980, 1981). Alikonis (1961) applied a coating which consists of zein, accetylated glycerides and an antioxidant in an alcohol solution by means of a spray and reported extended shelf-life of coated roasted peanuts at least 3 to 4 times the length of the uncoated peanuts when stored at room temperature. One manufacturer also reported that by coating granulated nuts for cake mixes with a 3-5% distilled acetylated monoglyceride and antioxidant combination, shelf-life was improved (Anon., 1965). Hoover and Nathan (1980) doubled the shelf-life of roasted peanuts by coating with 0.04% and 0.06% tertiary butylhydroquinone solutions.

Salt added to roasted peanuts affects stability of peanut products. Shelf-life of peanuts salted-in-the-shell for roasting is four to six weeks or approximately half that of unsalted nuts (Cecil and Woodroof, 1959). The use of salt containing low levels of calcium, copper and iron and storage under vacuum or nitrogen environment at cold temperature are recommended to extend the shelf-life of roasted peanuts (Ahmed and Pattee, 1987). Oxidative stability of the raw peanuts has a general tendency to decrease with seed size and storage time (Pattee et al., 1982b).

8. Vitamin E

Structure

Vitamin E is the generic term for tocopherols and tocotrienols that exhibit vitamin E activity. At present, eight naturally occurring homologs are included in the vitamin E family. These include four tocopherols (α-, β-, γ- and δ-tocopherol) and four tocotrienols (α-, β-, γ- and δ-tocotrienol). The structures for tocopherols and tocotrienols are given in Figure 1-3. Tocopherols have a saturated side chain while tocotrienols have a unsaturated side chain with double bonds at the 3′, 7′, and 11′ positions of the side chain. The eight



Tocopherol or Tocotrienol	R1	R2	R3
α-5,7,8-Trimethyl	CH3	CH3	CH3
β-5,8-Dimethyl	CH3	H	CH3
γ-7,8-Dimethyl	H	CH3	CH3
δ-8-Methyl	H	H	CH3

Figure 1-3. Structures of Tocopherols and Tocotrienols

vitamin E homologs differ in the number and location of the methyl groups on the chroman ring (Kijima, 1993). Tocopherols exist in eight diastereomeric forms due to the presence of three asymmetric carbon atoms at position 2 of the chroman ring and at 4′ and 8′ of the side chain. Although tocotrienols with double bonds in the side chain can form both *cis* and *trans* isomers, only *trans* forms exist in nature. Synthetic vitamin E is present in fortified foods and in vitamin supplements as esters of either the natural RRR- or the synthetic mixture (*all rac*-) forms.

Biological activity

The epimeric configuration at the 2 position is important in determining biological activity (Tappel, 1992; Stone and Papas, 1997). The Biological activity of tocopherols and tocotrienols was determined by the rat fetal resorption assay and was expressed in RRR-α-tocopherol equivalent (α-TE) units where one α-TE is the activity of 1 mg of RRR-α-T (Table 1-4) (NRC, 1989). Based on the individual biological activities of vitamin E homologs (Table 1-4), vitamin E content in foods has been reported as α-TE units by using the following conversion factors: α-T, 1; β-T, 0.5; γ-T, 0.1; δ-T, 0.03; α-T3, 0.3; β-T3, 0.05; *all rac*-α-T, 0.74; *rac*-α-tocopheryl acetate, 0.67 (NRC, 1989; Pryor, 1995).

Recently, only 2R-stereoisomers (RRR-, RSR-, RRS-, and RSS-) of α-T are recommended to estimate the vitamin E requirement for humans (Institute of Medicine, 2000). This recommendation is based on current information that vitamin E forms are not interconvertible in humans and that the levels of vitamin E forms in human plasma is dependent on the affinity of hepatic α-tocopherol transfer protein (α-TTP) which selectively transfers 2R-stereoisomers of α-T from chylomicron remnants to very low density lipoprotein (VLDL) (Traber, 1999). Therefore, 2S-stereoisomers of α-T, the other tocopherols (β-, γ-, and δ-T), and tocotrienols are excluded to estimate the vitamin E requirement for humans because of their limitation in binding with the α-TTP. For the last two decades, α-TE was used for most nutrient databases as well as nutrition labels without distinguishing between the different tocopherols in foods. The Dietary Reference Intake (DRI) report (Institute of Medicine, 2000) shows the conversion of α-TE to RRR-

Table 1-4. Biological Activity of Vitamin E Homologs and Derivatives

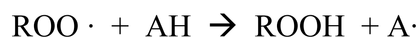
	Activity	
Name	IU/mg	Compared to RRR-α-T
Natural vitamin E		
2R4'R8'R-α-tocopherol	1.49	100%
2R4'R8'R-β-tocopherol	0.75	50%
2R4'R8'R-γ-tocopherol	0.15	10%
2R4'R8'R-δ-tocopherol	0.05	3%
2R-α-tocotrienol	0.75	30%
2R-α-tocotrienol	0.08	5%
2R-α-tocotrienol	Not known	-
2R-α-tocotrienol	Not known	-
Synthetic vitamin E		
2S4'R8'R-α-tocopherol	0.46	31%
2R4'R8'S-α-tocopherol	1.34	90%
2S4'R8'S-α-tocopherol	0.55	37%
2R4'S8'S-α-tocopherol	1.09	73%
2S4'S8'R-α-tocopherol	0.31	21%
2R4'S8'S-α-tocopherol	0.85	57%
2S4'S8'S-α-tocopherol	1.10	60%
RRR-α-tocopheryl acetate	1.36	91%
RRR-α-tocopheryl acid succinate	1.21	81%
All-rac-α-tocopherol	1.10	74%
All-rac-α-tocopheryl acetate	1.00	67%
All-rac-α-tocopheryl acid succinate	0.89	60%

(Pryor, 1995)

-α-T by using the equation (mg of α-T in a meal = mg of α-TE in a meal × 0.8) based on USDA food intake survey data and also recommends abandoning the use of α-TE.
Now, intake of only the 2R-stereoisomers of α-T from foods and supplements is strongly recommended to establish the Dietary Reference Intakes (DRIs) for vitamin E, especially Estimated Average Requirements (EARs), Recommended Dietary Allowances (RDAs), and Adequate Intakes (AIs) (Institute of Medicine, 2000). However, all eight stereoisomers of supplemental α-T are used as the basis for establishing the Tolerable Upper Intake Levels (ULs) for vitamin E.

Functions

Vitamin E functions as an antioxidant to protect cellular membranes from destruction by preventing the oxidation of unsaturated fatty acids in the phospholipids. Its antioxidant function involves the reduction of free radicals, preventing the potentially deleterious reactions of highly reactive oxidizing species (Tappel, 1992). Vitamin E is a primary antioxidant which can break the chain reaction during the propagation of free radical reactions due to the reactivity of the phenolic hydrogen on its hydroxyl group in the chroman ring system (Burton and Ingold, 1989).


ROO · + AH → ROOH + A·

RO · + AH → ROH + A·

R · + AH → RH + A·

In this process, vitamin E (AH) donates the phenolic hydrogen to a fatty acyl free radical (ROO ·, RO ·, R·) to prevent the attack of the free radical on other PUFAs and, then, vitamin E itself is converted to a semi-stable radical intermediate, the tocopheroxyl radical (A·). The tocopheroxyl radical is stabilized by delocalization of the unpaired electron around a phenol ring to form stable resonance hybrids. Tocopheroxyl radicals can interact with other compounds or with each other to form a variety of products (Figure 1-4). Under conditions of low oxidation rates in lipid membrane systems, tocopheroxyl radicals primarily form tocopherylquinone. Tocopherylquinone can be formed when the interaction of two tocopheroxyl radicals with the regeneration of tocopherol (Nawar, 1996). Formation of tocopherylquinone is also thought to occur by

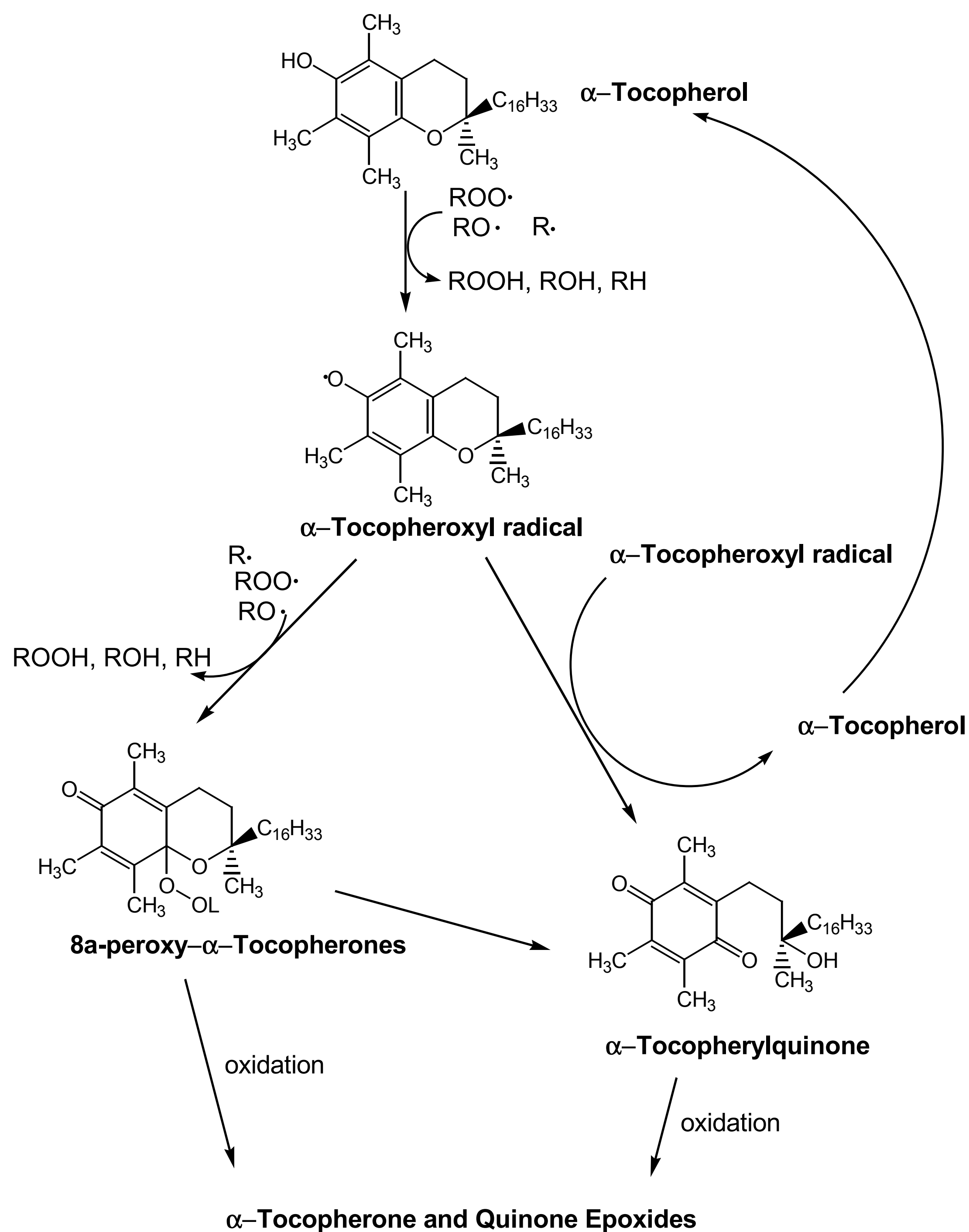


Figure 1-4. Formation of Oxidized α-Tocopherol Products (Kamal-Eldin and Appelqvist, 1996; Decker, 1998).

the transfer of an electron from a tocopheroxyl radical to a phospholipids peroxyl radical, to form a phospholipids peroxyl anion and tocopherol cation.

The tocopherol cation hydrolyzes to 8a-hydroxytocopherone, which rearranges to tocopherylquinone (Liebler and Burr, 1992). Under condition of more extensive oxidation, high concentration of peroxyl radicals favorably form tocopherol-peroxyl complexes. These complexes can hydrolyze to tocopherylquinone. Subsequent hydrolysis leads to the formation of epoxyquinone (Liebler, 1991). The formed tocopherylquinone can be regenerated back to tocopherol in the presence of reducing agents (e.g., ascorbic acid and glutathione) (Figure 5) (Chow, 1991). An additional reaction that can occur is the interaction of two tocopheryl radicals to form tocopherol dimers and trimers (Draper, 1996).

Vitamin E acts as a major peroxyl radical scavenger in biomembranes, resulting in stabilization of membranes. Free radical–induced oxidative damage to membrane lipids is regarded as a critical initiating event leading to cell injury (Tappel, 1972). Biological membranes contain a relatively high portion of polyunsaturated lipids which become susceptible to oxidation. Lipid oxidation is associated with the loss of membrane polyunsaturated fatty acids and the formation of hydroperoxides, free radical intermediates, and other secondary products. This process may disturb the structure of membranes and affect the permeability and functions of the membrane, including activation of phospholipase A2 and disturbance of the critical calcium homeostasis (Ungemach, 1987). Lipid oxidation products including hydroperoxides, aldehydes, and epoxides may react with essential proteins, enzymes, and nucleic acids, causing irreversible damage to the cells. Vitamin E is also believed to act as a membrane stabilizer by forming complexes with the products of membrane lipid hydrolysis such as lysophopholipids and free fatty acids (Wang and Quinn, 2000). Vitamin E and fatty acid molecules are assumed to form a complex by a hydrogen bonding. Shamovsky et al. (1992) stated that the stability of the fatty acid complexes with vitamin E increased as the number of fatty acid double bonds increases.

Oxidized vitamin E, tocopheryl radical, is considered to be regenerated to the tocopherol or tocotrienol form by biochemical mechanisms including ascorbate and, glutathione

(Packer, 1995). The regenerating system is in Figure 1-5 (Chow, 1991). In 1961, Tappel (1972) proposed that ascorbate may function to regenerate oxidized vitamin E. A direct reduction of tocopheroxyl radical by ascorbate was detected in a pure solution in 1979 (Packer, 1979). It is difficult to conceive that this could occur *in vivo* because ascorbate and GSH are water soluble and reside primarily within in the cytosol, and vitamin E is lipid soluble and residues primarily within biomembranes. Vitamin E can be spared by ascorbate or glutathione (GSH) in a number of pure or biological systems. Although previous studies strongly support the hypothesis that repairing mechanisms for oxidized vitamin E do exist in vivo, whether vitamin E is regenerated in vivo is still questioned (Chow, 1991). A model describing the effects of vitamin E in protecting membranes in plant tissue has been proposed by Shewfelt and del Rosario (2000).

Tocopheryl radicals may be regenerated by Maillard reaction products (MRPs) (Beddows et al., 2001; Wagner et al., 2002). MRPs are shown to efficiently suppress oxidation in different foods like cereals, milk, meat, juices or nuts (Anese et al., 1999; Bedinghaus and Ockerman, 1995; Hansen and Hemphill, 1984; Wijewickreme and Kitts, 1998) and in model systems (Jing and Kitts, 2000; Yoshimura et al., 1997). MRPs are found as highly antioxidantive in water-soluble but less effective in water-insoluble conditions. The mechanism of MRPs has been proposed to suppress the peroxide formation as scavenger of free radicals or interact with the remaining tocopherols in lipids and act synergistically by regenerating the oxidized tocopherols. A conceivable mechanism for this hypothesis is shown in Figure 1-6.

The relative effectiveness of tocopherols as antioxidants depends mainly on the chroman structure (Stone and Papas, 1997). α-T3 has similar antioxidant activity as α-T in homogeneous solutions and in low density proteins (Suarna et al., 1993). There was no difference in antioxidant activities between RRR-α-T and *all-rac*-α-T since they have identical chroman structures. The number of methyl groups on chroman structure does not exert a major effect on the antioxidant activities of different tocopherol homologs (Stone and Papas, 1997). α-T3 with three methyl groups on the chroman ring has similar antioxidant activity compared to δ-T with one methyl group against peroxy radicals (Niki, 1987). On the other hand, Giese (1996) found that γ- and δ-T had higher antioxidant

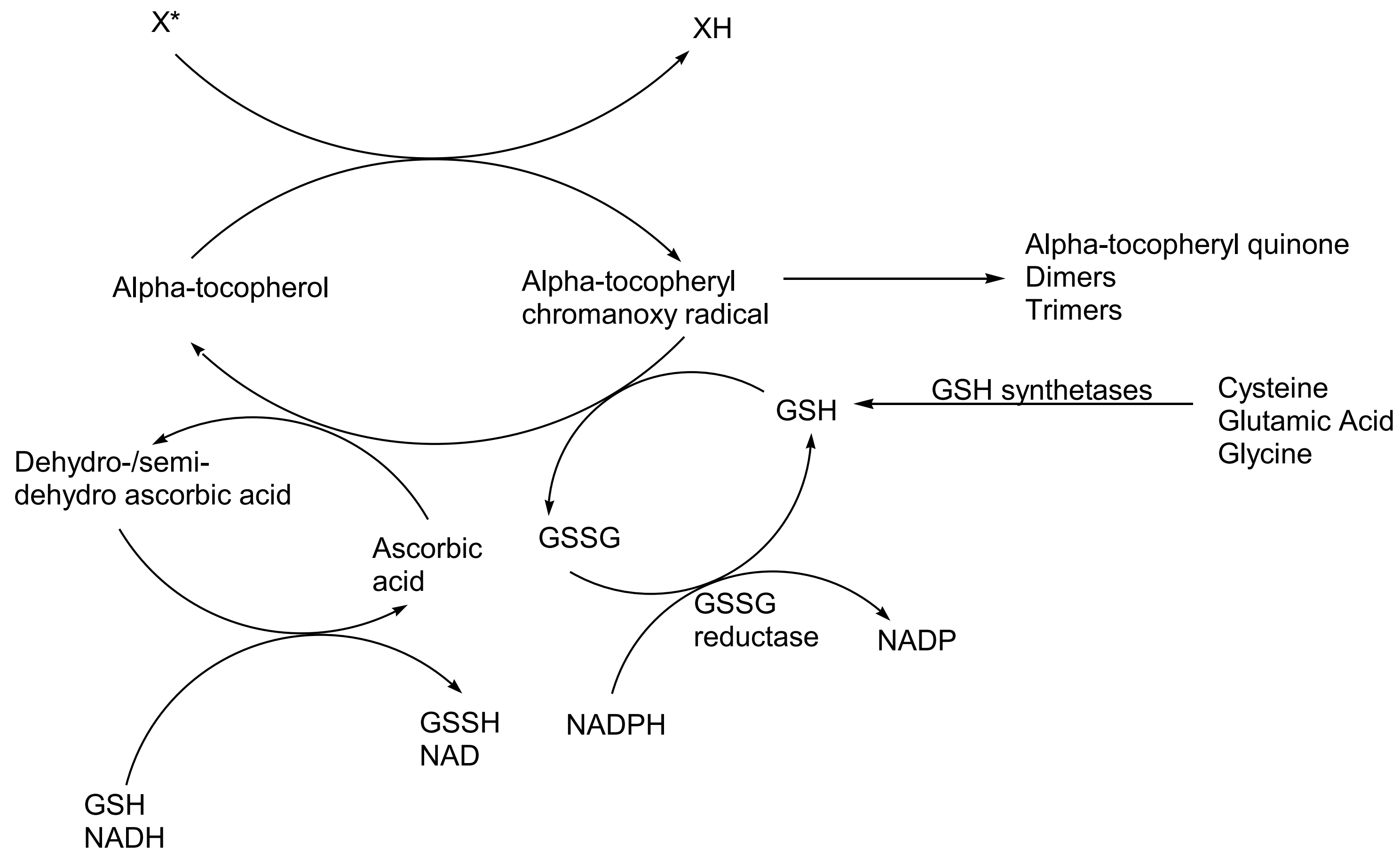


Figure 1-5. A-Tocopherol regeneration systems. X· - free radicals; GSH – reduced glutathione; GSSG – oxidized glutathione; NADH or NAD – reduced or oxidized nicotinamide adenine dinucleotide (Chow, 1991).

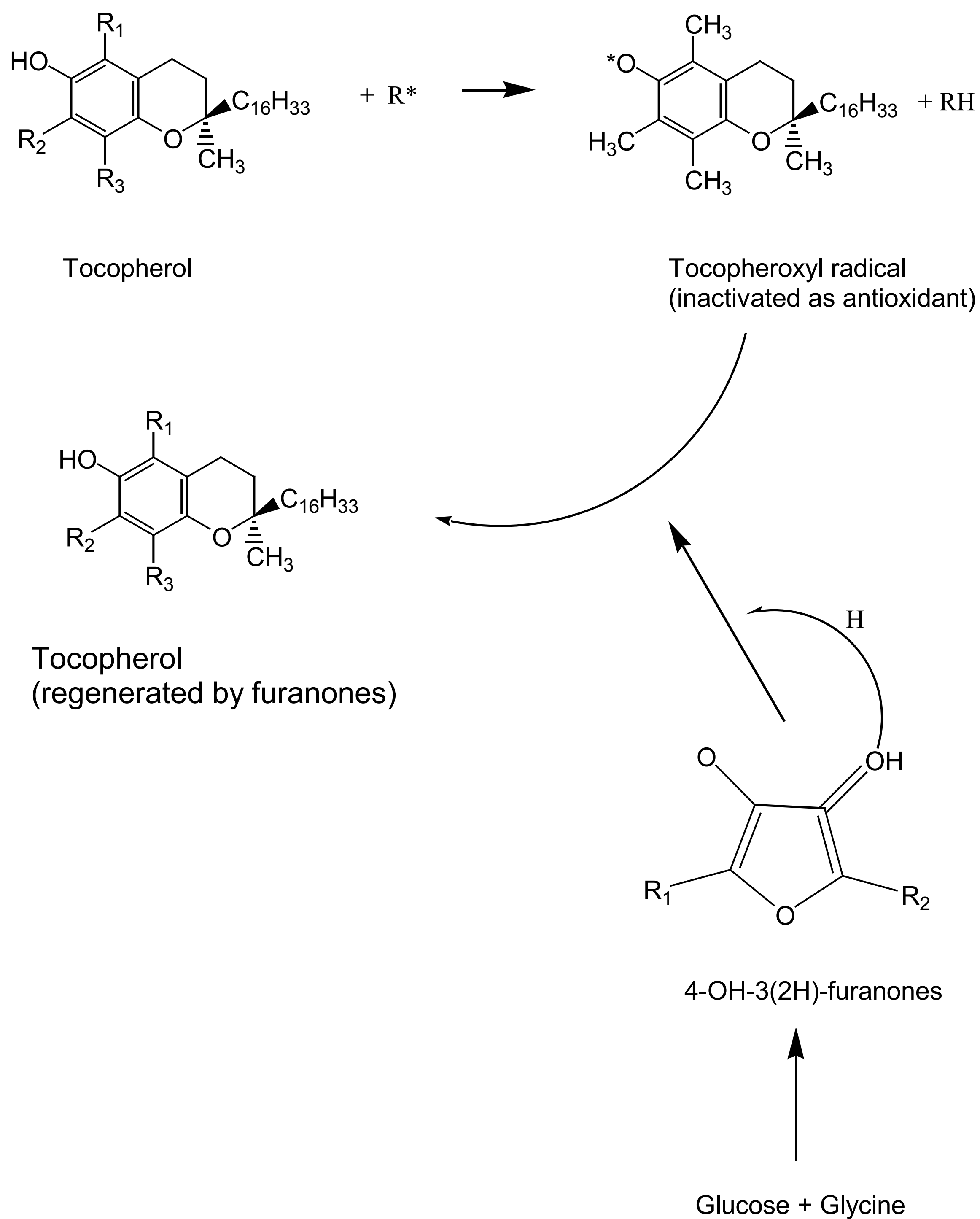


Figure 1-6. Possible pathway for the regeneration of inactivated tocopheryl radicals by Maillard reaction products-4-OH-3(2H)-furanones in the reaction of glucose and glycine (Wagner et al., 2002).

activity than that of γ- and δ-T. The ester forms of α-tocopherol such as α-tocopheryl acetate and succinate are widely used in food fortification due to their increased stability. They have no antioxidant activity but exhibit *in vivo* antioxidant activity as a result of enzymatic cleavage of the ester (Liebler, 1993; Gregory, 1995).

Lipid oxidation is particularly common in cell and organelle membranes, lipoproteins, the adipose tissue, brain, and other tissues where PUFA are abundant. Due to the role of vitamin E as an antioxidant, it is believed that vitamin E reduces various human diseases especially caused by oxidative stress including cancer, cardiovascular and coronary heart diseases (Jacob and Burri, 1996; Leger, 2000; Pruthi et al., 2001; Adams and Best, 2002). The other health benefits of vitamin E include increased resistance to infection (Meydani et al., 1989; Mehta et al., 1998; Han and Meydani, 1999), enhanced immune response (Gaby and Machlin, 1991; Meydani et al., 1997), prevention of atherosclerosis (Verlangiery and Bush, 1992; Neuzil et al., 2001), reduced progression of cataracts (Robertson et al., 1989; Leske et al., 1998), prevention of exercise-induced muscle damage (Gohil et al., 1987; Adams and best, 2002) and haemolytic and sickle cell diseases (Jain, 1989; Phillips and Tangney, 1992; Shukla et al., 2000), and decreased risk of neurological disorders such as Alzheimer disease (Muller, 1986; Rayner et al., 1996; Engelhart et al., 2002) and prevention of progression of kidney disease (Mune et al., 2002) and delayed aging (Bonnefoy et al., 2002). These benefits are highly related to the antioxidant activities of vitamin E.

Physico-Chemical Properties

Vitamin E in the oil form is relatively stable to oxidation if protected from oxidative conditions. It shows good heat stability in the absence of oxidative events. Light is a significant accelerator of lipid oxidation and vitamin E in an oxidation-prone environment decreases more rapidly when subjected to UV compared to visible light. Esters such as *all rac*-α-tocopheryl acetate can not donate the phenolic hydrogen atom required for antioxidant action, therefore, vitamin E esters are more stable under oxidative conditions. Ester forms, due to their oxidative stability, are used for food fortification and in supplements (Johnson, 1995).

Requirement

Vitamin E is essential for normal growth and development. Vitamin E deficiency leads to clinical abnormalities. Based on recent powerful studies that other naturally occurring forms (β-, γ-, and δ-T and tocotrienols) are poorly recognized by the α-T transfer protein in plasma or tissue and then not converted to α-T, resulting in no contribution to the human requirement, only in the 2R-stereoisomers of α-T from foods and supplements is currently recommended to establish the vitamin E requirement for humans (Institute of Medicine, 2000). There are four possible values for vitamin E requirements for humans, which are referred to as the Dietary Recommended Intakes (DRI):

1) Estimated Average Requirement (EAR), the nutrient intake estimated to be adequate for a specific function in half of a given population group.
2) Recommended Dietary Allowance (RDA), the daily intake sufficient to meet the nutrients of nearly all individuals in a given population group.
3) Adequate Intake (AI), an approximation of the average nutrient intake needed by a population group to meet a defined functional requirement.
4) Tolerable Upper Intake Level (UL), the maximal level of intake that is considerable safe.

Traditionally, RDA is used to measure the adequacy of nutrient needs for all healthy persons, but is not a value to cover special needs due to chronic diseases, infections, etc. The RDA for vitamin E is shown in Table 1-5.

The requirement of vitamin E is elevated when the intake of polyunsaturated fatty acids is increased (NRC, 1989). Some researchers suggested that a ratio of at least 0.4 mg α-T per gram of PUFA should be consumed by adults (Bieri and Evarts, 1973; Horwitt, 1974; Witting and Lee, 1975). However, the vitamin E requirement is more affected by several factors than PUFA mass including the degrees of unsaturation, the portion of PUFA deposition in the tissue, and PUFA metabolism (Draper, 1993; Johne and Kubow, 1999). Energy and nutrient status of the organism also influences the vitamin E requirement. When vitamin E intercepts a radical, it is oxidized to the tocopheroxyl

Table 1-5. The Recommenced Daily Allowance (RDA) for Vitamin E

Group	Age (years)	RDA (mg/day of α-tocopherol)
Children	1-3	6
	4-8	7
Boys	9-13	11
	14-18	15
Girls	9-13	11
	14-18	15
Adult men	> 19	15
Adult women	> 19	15
Pregnancy	14-50	15
Lactation	14-50	19

(Institute of Medicine, Food and Nutrition Board, 2000)

radical. The tocopheroxyl radical can be regenerated by biological reducing agents including vitamin C (McCay, 1985), glutathione (Niki, 1987; Chow, 1991), and ubiquinols (Stoyanovsky et al., 1995) through the redox cycling reactions (Figure 5). The recycling reactions are coupled with the energy status of the organism. For example, inadequate production of nicotinamide adenine dinucleotide (NADH), nicotinamide adenine dinucleotide phosphate (NADPH) or glutathione (GSSG) reductase are caused by dietary deficiencies of niacin (component of NADP or NADPH) or riboflavin (cofactor for GSSG reductase), resulting in break of the redox reaction chain. In case of intakes of antioxidant compounds such as plant polyphenols and flavonoids, the total antioxidant pool may be increased. Besides, vitamin requirements can be influenced by age, gender, pregnancy, lactation, diseases, exercise, body size, smoking habits and chronic diseases (Institute of Medicine, 2000).

Toxicity

Vitamin E is one of the least toxic vitamins (Tappel, 1992). There is no evidence of side effects from the consumption of vitamin E naturally occurring in foods. Most studies on vitamin E toxicity are conducted by intake of α-T as a supplement, food fortificant, or pharmacological agent. Animal studies on the toxicity of vitamin E show that α-T is not mutagenic, carcinogenic, or teratogenic (Abdo et al., 1986; Dysmsza and Park, 1975; Krasavage and Terhaar, 1977). Kappus and Diplock (1992) reviewed numerous studies on vitamin E toxicity and concluded that humans show few side effects following supplemental doses below 2,100 mg per day of tocopherol. One study reported a significant 50% increase in mortality from hemorrhagic stroke in Finnish male smokers who consume 50 mg/day of *all rac*-α-T for 6 years (Alpha-Tocopherol, Beta Carotene (ATBC) Cancer Prevention Study Group, 1994). On the other hand, an increase in hemorrhagic stroke was not observed in patients with Alzheimer's disease consuming 2,000 IU (1340 mg)/day of *all rac*-α-T for 2 years (Sano et al., 1997). Most adults appear to tolerate relatively high oral doses without significant evidence of toxicity, but muscle weakness, fatigue, double vision, emotional disturbance, breast soreness, thrombophlebitis, and gastrointestinal distress symptoms including nausea, diarrhea and flatulence were occasionally reported as side effects (NRC, 1989; Groff et al., 1995;

Institute of Medicine, 2000). Based on animal studies, vitamin E toxicity in humans is difficult to project because of the unclear relevance of animal study evidences to human situations. Human studies of short duration provide little information on side effects due to lack of excess dosage and short periods (few weeks or months) of vitamin E supplementation (Institute of Medicine, 2000).

Since side effects by vitamin E overintake have been reported, establishment of the maximum level for vitamin E is needed for a safe intake. For the purpose, an UL was established for vitamin E. This is the highest level of daily nutrient intake that is likely to be at no risk of adverse effects in almost all individuals, while consumption of vitamin E above the UL may be at some risk. The UL for α-T for adults is 1,000 mg/day of all eight stereoisomers of α-T (Institute of Medicine, 2000). Council for Responsible Nutrition (CRN) provides two values for a safe oral intake, the No Observed Adverse Effect Level (NOAEL) and the Lowest Observed Adverse Effect Level (LOAEL). The NOAEL for vitamin E is 1200 IU which was established based on human data; therefore, it requires no safety factor and is considered as safe for long-term use of vitamin E supplements (CRN, 1998). On the other hand, since the LOAEL is the lowest level of intake for which adverse health effects have been reliably reported in humans, a safety factor may be applied to this level for a safe intake. Now, a LOAEL for vitamin E has been not established due to lack of data indicating oral toxicity.

Occurrence in foods

Table 1-6 provides data on the contribution of 15 aggregated food categories to vitamin E in the U.S. diet. Fats and oils are the number-one contributor, providing more than 20% of vitamin E in the U.S. diet. Although nuts and seeds are rich sources of vitamin E, they contribute only 3.8% to the diet because of infrequent consumption (Murphy et al., 1990). One reason for this is they are considered a significant source of fat and calories causing weight gain. More recent data indicates increasing nut consumption as consumers become more and more aware of beneficial health effects (Dahm, 2002). For almonds, five of the 10 food categories in which almonds have a substantial ingredient presence grew at double digit growth rates in 2001 (Dahm, 2002).

Table 1-6. Contribution of Food Categories to Vitamin E in the Adult U.S. Diet

Food category	Percent of total vitamin E
Fats and oils	20.2
Vegetables	15.1
Meat, poultry, and fish	12.6
Desserts	9.9
Breakfast cereals	9.3
Fruits	5.3
Bread and grain products	5.3
Dairy products	4.5
Mixed main dishes	4.0
Nuts and seeds	3.8
Eggs	3.2
Salty snacks	3.0
Legumes	2.1
Soups, sauce and gravies	1.7
Beverages	0.0

(Murphy, 1990)

9. The Effect of Roasting on Vitamin E in Oil Seeds

Vitamin E stability of several oil seeds during roasting has been studied. There are conflicting results to conclude the effect of roasting on vitamin E in oilseeds. Some studies reported a decrease in levels of vitamin E in oils prepared from roasted oilseeds. Shin et al. (1997) noted a progressive decrease in the levels of tocopherols and tocotrienols in rice bran oil that was extruded at increasing temperatures. Yoshida and Takagi (1997) also reported that roasting (160-250 ºC) of sesame seeds decreased the levels of tocopherols, sesamolin and sesamin. The decrease was greater as roasting temperature increased. Yoshida et al. (1995, 1999, 2001, 2002) also reported that roasting in a microwave or electric oven decreased the levels of tocopherols in sesame, soybean and sunflower seed oils. The degree of decrease in tocopherols was greater as roasting temperature increased. Although roasting caused a gradual decrease of vitamin E, the levels of vitamin E were still remained high. Compared to the original levels, more than 80 and 90% of vitamin E was retained in soybeans and sunflower seeds after 20 min of microwave roasting, respectively (Yoshida et al., 1999, 2002). Sesame seeds roasted in an electric oven at 180-220 ºC also showed a decrease in the levels of γ- and δ-T. γ-T was still present at more than 80% of its original value after roasting for 25 min at 180, 200 and 220 ºC (Yoshida et al., 2001).

In contrast, Lane et al. (1997) reported that a heat pre-treatment (100-175 ºC) of rice bran increased the levels of extracted tocopherols, tocotrienols and other components. A similar phenomenon was observed in heated corn fiber (Moreau et al., 1999). They suggested that a significant amount of the tocopherols and tocotrienols are bound to proteins or linked to phosphate or phospholipids. Heat breaks these bonds, resulting in an increase of extractable tocopherols. Kim et al. (2002) also noted that α- and γ-T in rice germ oil gradually increased as roasting temperature (160-180 ºC) and time (0-15 m) increased.

Yen et al. (1990) observed that the level of γ-T in oils increased by roasting temperatures up to 200 ºC over 30 min but fell with higher roasting temperature, resulting in the highest level of γ-T in oils prepared by roasting at 200-220 ºC. Due to variation in roasting conditions (methods, temperature and time), type of samples, and oil extraction methods, it is difficult to compare these observations. Based on the results of the above

studies, high retention levels of tocopherols even after severe roasting indicates that tocopherols are highly stable to heating. Mild heating appears to increase the tocopherol levels, possibly due to improvement of oil extraction, especially tocopherols bound to other components in seeds, by heating.

Longer roasting of peanut kernels increases oxidative stability of the oils (Cheng et al., 1987; Huang et al., 1988; Chiou et al., 1991). Chiou (1992) reported that oil prepared after 90 min of roasting was more stable to oxidative changes than oil prepared from peanuts roasted for less time. Megahed (2001) reported that oil stability decreased by microwave roasting due to the formation of oxygenated compounds in oils extracted from roasted peanuts increased with microwave roasting time. Young (Moss and Otten, 1989) found that the optimum process requires approximately five separate heating zones to raise the temperature of the nuts slowly to the roasting temperature. He also found that peanuts roasted at low temperature for longer time have the best flavor and a longer shelf life due to more even roast. However, the information on the effect of roasting on vitamin E in peanuts is not available.

10. Storage Stability of Vitamin E

Storage of raw and processed foods can produce significant decreases in vitamin E. Packaging methods and materials, length and temperature of storage, characteristics of the food and its susceptibility to lipid oxidation, availability of other natural or synthetic antioxidants in the food and many other factors affect the stability of vitamin E during storage. Ames (1972) in his early review stated that foods are normally exposed to deleterious factors during processing and storage that can lead to large losses in vitamin E. A relationship between tocopherol stability and lipid oxidation has not been clearly established. Normally, storage losses are related to lipid oxidation occurring in the food and the interaction of vitamin E as an antioxidant. Previous reviews (Ames, 1972; Bauernfeind, 1977; Bramley et al., 2000; Kivimäe and Carpena, 1973) have shown that vitamin E stability in various raw and processed foods is quite good if the food is adequately protected from conditions conducive to lipid oxidation. Even under thermal abuse, if lipid oxidation is not proceeding, vitamin E can be expected to remain stable. If, however, oxidation is not controlled, rapid and extensive loss of vitamin E will occur.

The rate of degradation of vitamin E is dependent on the availability of oxygen, storage temperature, and water activity, as well as on the fat content and composition of the food. Alkali, light, and cations such as Fe^{3+} and Cu^{2+} accelerate degradation (Cort et al., 1978; Bauernfeind, 1980; Widicus et al., 1980; Widicus and Kirk, 1981). γ-T is a more effective antioxidant than α-T (Ikeda and Fukuzumi, 1977; Parkhurst et al., 1968). Adnan et al. (1981) stated that the effectiveness of tocopherols as antioxidants is not related to their disappearance from the sample and perhaps γ-T in particular, may be regenerated by other hydrogen donors in the system. α-T is the most stable tocopherol (Carpenter et al., 1976; Lehmann and Slover, 1976). The rate of tocopherol destruction in oxidizing lipids is dependent on the type of hydroperoxides (Frankel et al., 1957; Lips, 1957) and increases with increasing unsaturation.

Lipoxygenase-catalyzed oxidation of linoleic acid can be accompanied by considerable losses of fat-soluble vitamins. Tappel et al. (1953) showed that α-T is destroyed during the lipoxygenase-catalysed oxidation of linoleic acid. Gordon and Barimalaa (1989) also reported that vitamin A, D and E are all susceptible to rapid co-oxidation at neutral pH in the presence of soybean lipoxygenase. Roozen et al (1994) who studied lipid oxidation in low fat foods reported that the presence of α-T has no influence on the rate of enzymatic oxidation but inhibits formation of hexanal, a decomposition product. Chu and Lin (1993) investigated factors affecting the vitamin E content in soybean oils as related to the storage conditions of soybean flour, cracked beans, and flakes with three different thicknesses. Soybean flakes with a thickness of 0.16-0.33 mm showed higher extracted oil yield but a slightly lower tocopherol content of the oils than did cracked beans and thicker bean flakes. Oils from thin flakes were significantly low in tocopherol content compared to those from medium and thick flakes, indicating vitamin E destruction by enzyme action.

Storage studies for edible oils (Chu and Lin, 1993; Jaimand and Rezaee, 1995; Shahidi et al., 1997; Goffman and Möllers, 2000; Cinquanta et al., 2001; Okogeri and Tasioula-Margari, 2002) indicated that vitamin E stability in oils is affected by absence of prooxidants, oxygen, metals, high intensity light and other types of irradiation, proper packaging that prevents oxygen transfer into the oil, and proper temperature. Under proper storage of high quality RBD oil, little loss of vitamin E occurs even after

prolonged storage. Storage studies on several tree nuts show that vitamin E content decreases as storage time increases, possibly as a result of its antioxidant function during lipid oxidation (Fourie and Basson, 1989a; Yao et al., 1992; Erickson et al., 1994; Senesi et al., 1991, 1996; Lavedrine et al., 1997; Lima et al., 1998). Rate of loss is highly affected by storage temperature, the packaging material and availability of oxygen.

Proper packaging could improve vitamin E retention in nuts. Sensi et al. (1991) showed that 94% loss of the initial α-T (35.4 mg/100g of oil) occurred for almond packaged in metallized film under vacuum while 80% loss under nitrogen during 18 month storage at 20 ºC. Tocopherols in unroasted pecans were stable in commercial cellophane packages in air at 0.6°C for 48 weeks. No vitamin E loss was noted in-shelled, roasted, and salted cashew nuts during storage in flexible packaging materials with a lower water vapor permeability rate at 30°C for 1 year (Lima et al., 1998). Lima et al. (1998) reported oscillating changes in tocopherol values of roasted cashew nut during storage time. This was also observed in raw and roasted pecans (Erickson et al., 1994) and pecans (Yao et al., 1992).

Fatty acid composition and geographic origin are also considered to affect tocopherol stability of nuts during storage. Lavedrine et al. (1997) stated that the very high level of polyunsaturated fatty acids (linoleic acid >60%; linolenic acid 10%) in walnuts explained the decrease of tocopherol amounts (about 30%) observed within three months. Such a decrease was not observed in pecans stored at 4°C (Yao et al., 1992), probably because of their low percentage of polyunsaturated fatty acid, linoleic acid (25%) and linolenic acid (1.5%) compared to the high percentage of monounsaturated fatty acids (oleic acid > 60%).

Fourie and Basson (1989) showed that changes in tocopherol content of tree nuts were related to storage stability. The high total tocopherol concentration in almonds accounted for their good storage stability at 33°C and 55% RH for 16 months. Macadamia nuts, with practically no tocopherols, were rancid after 2 months storage (Fourie and Basson, 1989).

11. Vitamin E in Peanuts

Peanuts are an excellent source of vitamin E. One ounce of peanuts provides 25% of daily need of vitamin E (USDA, 1998) (Table 2). The levels of vitamin E in peanuts are influenced by cultivar, genotype, growing conditions, origins, year, regions, and maturity stages (Baurnfeind, 1980; Sturm et al., 1966; Sanders et al., 1992; Hashim et al., 1993a, 1993b; Dutta et al., 1994). Sturm et al (1966) determined tocopherols in peanut oil from 17 varieties of three genotypes grown under the same conditions and found that runner varieties are the richest source for α-, γ-, and δ-T, while Spanish varieties had the lowest α-T. The variations of tocopherol contents among different cultivars are affected by the degree of maturation (Hashim et al., 1993a). Hashim et al (1993a) reported that α-T decreased with maturation of Sunrunner. γ-T decreased with maturation of Florunner peanuts. Sanders et al. (1992) reported significant differences in tocopherol levels among three origins of United States, Argentina and China.

After harvest, raw peanuts are processed to produce various peanut products including peanut butter, salted peanuts, confections, roasting stocks, peanut oil, etc. During processing and further storage of products, the level of tocopherols can be changed by processing itself and by oxidation due to their antioxidant activity. Because of the high content of unsaturated fatty acids in peanut lipids, vitamin E plays as integral role in controlling oxidation and maintaining quality of raw and processed peanut products. Some studies on lipid oxidation in peanuts have been reported (St. Angelo and Ory, 1977a; Evranz, 1993; Divino et al., 1996); however, little information is available on the vitamin E stability in peanuts related to lipid oxidation during storage. Tocopherol destruction was accelerated following the induction period of lipid oxidation in reconstituted partially defatted peanuts (RPDP) and blanched whole peanuts (BWP) during storage, suggesting that tocopherol stability in peanuts is closely related with lipid oxidation (Adnan et al., 1981). The study showed that more than 95% of α-T was destroyed for RPDP while 28% for BWP during 40 days of storage at 50°C.

Storage studies on raw and roasted peanuts usually indicate that roasted whole peanuts are less stable to oxidation than raw peanuts because of disruption of fat bodies and exposure of membrane phospholipids. One would surmise that vitamin E is, therefore, less stable in roasted peanuts compared to raw peanuts during storage, although little

literature is available to document this postulation. Changes in vitamin E during storage have been documented on several tree nuts including almond, macadamia, pecan, almond, walnut and cashew nut (Fourie and Basson, 1989a; Yao et al., 1992; Erickson et al., 1994; Senesi et al., 1991, 1996; Lavedrine et al., 1997; Lima et al., 1998). However, no information is available on raw and roasted peanuts.

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CHAPTER III

EFFECT OF PEANUT BUTTER MANUFACTURE ON VITAMIN E CONTENT OF PEANUTS AND ANALYSIS OF COMMERCIAL PEANUT PRODUCTS FOR VITAMIN E

INTRODUCTION

Numerous studies strongly suggest that frequent nut consumption protects against coronary heart disease (CHD) by decreasing low-density lipoprotein (LDL) cholesterol. The protective effect of nut consumption against CHD was first indicated by The Adventists Health study based upon dietary information on 31, 208 non-Hispanic Seventh-Day Adventists living in California. Consumption of nuts at more than four times per week decreased fatal CHD events when compared to subjects who consumed nuts less than once per week (Fraser et al., 1992). Consumer and health science interest in the dietary benefits of nut consumption increased with subsequent positive epidemiological studies including the Adventists Health Study (Fraser et al., 1995, 1997a, 1997b), the Iowa Women's Health Study (Prineas et al., 1993; Kushi et al., 1996), the Nurses' Health Study (Hu et al., 1998), the Cholesterol and Recurrent Events (CARE) Study (Brown et al., 1999), and the Physicians' Health Study (Albert and Willett, 2002). Due to a large number of participants who varied in age, sex, race, smoking habits, alcohol use, exercise habits, diet and lifestyle, the extended statistical strength led to the authors conclude that frequent nut consumption protects against CHD.

In clinical studies, high mono- and polyunsaturated fatty acid (MUFA and PUFA) diets supplemented with nuts including peanuts showed decreases in the total and LDL cholesterol levels which are risk factors for CHD (Spiller et al., 1992, 1998; Sabaté et al., 1993; Sabate and Hook, 1996; Lavedrine et al., 1997; Morgan and Clayshulte, 2000; Zambón et al., 2000; Curb et al., 2000; Rajaram et al., 2001; Almario et al., 2001; Hyson et al., 2002). O´Byrne et al. (1997) reported that a low fat diet supplemented with peanuts improved serum lipoprotein profiles when compared to a regular low fat diet for postmenopausal women with high serum cholesterol levels. Kris-Etherton et al. (1999a) indicated that high-MUFA diets are preferable to a low fat diet for lowering serum

cholesterol and that high consumption of peanuts and nuts might be a preferable dietary approach to low-fat diets to reduce CHD risk.

While the exact reasons are not known to explain the beneficial effects of nut consumption on blood lipid profiles, explanations offered in the literature include the unsaturated fatty acid profile of nut lipids, replacement of more hypercholesterolemic lipids in the diet by the lipids present in nuts and significant amounts of micronutrients considered beneficial to heart health (arginine, sterols, magnesium, copper, potassium, fiber, folate and vitamin E) (Sabate and Hook, 1996; Wolk et al., 1999; Kris-Etherton et al., 1999b, 2001).

One of the beneficial components in peanuts is vitamin E. There is evidence that diets high in vitamin E are protective against heart disease, possibly by preventing the oxidation of LDL cholesterol (Parthasarathy, 1992; Stampfer, 1993; Meyer, 1996). The minimum amount of vitamin E needed to inhibit LDL oxidation appears to be 400IU per day (Jailal, 1995). According to the Second National Health and Nutrition Examination Survey (NHANES II) (1976-1980), about 70% of people failed to meet 100% of the RDA for vitamin E (Kant, 1994). The most recent USDA nationwide survey (1987-1988) found that 34% of men and 41% of women among ages 25-50 intake under 75% of the RDA for vitamin E (Murphy et al., 1992). This may be due to a low fat diet trend, resulting in low intake of fat soluble vitamins including vitamin E based on the public’s mind that reduced fat or fat-free diet is good for health, especially lowering risk of CHD and inhibiting obesity. Some people avoid peanuts because peanuts are high in calories due to high fat content (about 50%). Fortunately, about 85% of the lipid in peanuts is unsaturated fat known as “good fat”. In addition, recent findings show that regular peanut consumption is an effective way to control hunger without significant weight gain (Kirkmeyer and Mattes, 2000; Alper, 2001; Mattes and Lokko, 2002; Alper and Mattes, 2002).

Overall, peanuts and peanut products are considered to be excellent sources of

vitamin E to the U.S consumer. Most recent USDA data obtained from the 1994-1996 Continuing Survey of Food Intakes by Individuals (CSFII, 1994) shows that smooth peanut butter by itself ranks as the 8th most significant source of vitamin E in the US diet, providing 2.3% of the total vitamin E as measured by alpha-tocopherol equivalents (α-TE) (D. Haytowitz, private communication).

Levels of vitamin E in peanuts and peanut products are influenced by cultivar, growing condition, and maturity (Sturm et al., 1966; Hashim et al., 1993a, 1993b). After harvesting, the vitamin E level of peanuts can be influenced by processing, storage, and marketing. Roasting imparts flavor and inactivates lipoxygenase in peanuts. Information on the nutrient compositional changes of peanuts associated with oil, protein, carbohydrate, mineral and water-soluble vitamin contents during roasting have been reported (Derise et al., 1974; St. Angelo et al., 1977; Oupadissakoon et al., 1984; Damame et al., 1990). Variable tocopherol levels of raw and roasted peanuts and peanut butter were reported by some researchers (Hashim et al., 1993a; Piironen et al., 1986; Lee et al., 1998; Wyatt et al., 1998). However, no information is available on the effect of peanut butter manufacture on vitamin E content of peanuts.

The purpose of this study was to provide more complete data on the vitamin E content of commercial raw, dry-roasted peanuts and peanut products including peanut butter and peanut oils and to investigate the effect of peanut butter manufacture on vitamin E content of peanuts.

MATERIALS AND METHODS

Samples

All raw samples (runner type) used for peanut butter manufacture were of mixed cultivars but were mostly comprised of Georgia Green. After harvest, peanuts were stored

in-shell in silos at ambient temperature (55-70 ºF). After shelling, peanuts were stored at 34-41 ºF with relative humidity of 55-70% (NPC, 2002). Shelled peanuts were shipped in 2000 lb containers and transported to Tara Foods, Albany, Ga. Prior to shipment, peanuts were tempered at 55 ºF for 48-72 h to avoid condensation. At Tara Foods, peanuts were stored for less than 2 weeks at ambient temperature and then processed into peanut butter.

Peanut Butter Manufacture

Peanut butter was commercially manufactured at Tara Foods. Roasting was conducted at 270 to 290 °F in the 1st zone and 340 to 385 °F in the 2nd zone in a Proctor-Schwartz gas-fired roaster at Tara Foods. Total roasting time was 18-20 min. After roasting, roasted peanuts were cooled down below 100 ºF. Roasting conditions were adjusted by Tara Foods to achieve Hunter color L value of 49 ± 1 (medium roasting). Roasted peanuts were dry blanched by passing through the blancher to remove the skins. Poor quality nuts or other undesirable matter were removed by screening and inspection. Peanut butter was produced by a two-stage milling process. During milling, the peanut butter is heated to the range of 140-180 °F. Following deaeration, the peanut butter was filled into commercial 18 ounce jars.

Sampling

Samples were obtained directly from the processing line at Tara Foods. Raw peanuts and peanut butter samples of two crop years are from different processing runs (total runs = 12). When blends of raw peanuts were used for the processing run, each raw peanut sample used in the blend was individually sampled prior to blending. One to two kg of each raw peanut sample and four 18 ounce jars of the finished peanut butter were shipped by overnight express to the University of Georgia for analysis the next day. In some cases, university personnel obtained the samples on the day of processing and

transported the samples to Athens, Ga for analysis. Raw peanuts and peanut butter samples were stored at -16 ºC until assay.

To investigate contribution of other ingredients to tocopherol levels of peanut butter, stabilizer and peanut oil were assayed for vitamin E. A hydrogenated rapeseed oil stabilizer was obtained from Tara Foods. Commercial raw and roasted peanuts, peanut butter and peanut oils were purchased locally and assayed for vitamin E.

Vitamin E Analysis

Assay of vitamin E was completed by direct solvent extraction coupled with HPLC that was optimized for assay of peanuts and peanut products (Lee et al., 1998). Validation parameters obtained with this procedure in the current study are given in Table 2-1. Linearity tests of the standard curves were completed over ranges of 0.0 to 36.4, 0.0 to 1.8, 0.0 to 27.6 and 0.0 to 3.4 ng/20 μL injection volume for α-tocopherol (α-T), β-tocopherol (β-T), γ-tocopherol (γ-T) and δ-tocopherol (δ-T), respectively. Excellent linear relationships between the peak areas and the amount of injected tocopherol were observed (r^2 = 0.999). The limit of detection limit (LD) was calculated based on the detector's signal-to-noise (S/N) ratio. To obtain the detection limit, the standard deviation of S/N ratio was multiplied by 3 as a factor and then added to the average of the S/N ratio. The LDs were 0.44, 0.05, 0.16, and 0.07 ng/20 μL for α-, β-, γ-, and δ-T, respectively. For the limit of quantitation (LQ), 10 was use as a multiplying factor (*Food Chemicals Codex*, 1996). The LQs were 0.54, 0.09, 0.23, and 0.12 ng/20 μL.

Repeatability (RT) and reproducibility (RD) were determined to evaluate the precision of the vitamin E assay (Table 1). The coefficient of variations (CV) of RT and RD were less than or about 5% for all homologs, showing the high precision of the analysis. The accuracy expressed as recovery was also excellent. The % mean recoveries ± S.D.
Table 2-1. Precision and Accuracy of Assay

		Precision		Accuracy [a]
Homologs [b]	Parameters	Repeatability [c]	Reproducibility [d]	Recovery
		(mg/100 g)		(%)
α-T	Mean [e]	11.60	11.86	97.8
	S.D. [f]	0.14	0.23	1.64
	CV [g], %	1.22	1.90	1.68
β-T	Mean	0.42	0.44	95.8
	S.D.	0.02	0.02	3.03
	CV, %	3.58	4.09	3.17
γ-T	Mean	9.98	9.70	98.0
	S.D.	0.24	0.27	2.12
	CV, %	2.36	2.83	2.16
δ-T	Mean	1.00	0.97	97.0
	S.D.	0.05	0.07	2.81
	CV, %	5.23	6.75	3.93

[a] Accuracy is a measure of the closeness of the analytical result to the true value evaluated by analyzing a spiked sample.
[b] Corresponding tocopherols.
[c] Repeatability refers to the results of independent determinations carried out on a sample by analyzing five replicates of the sample on same day.
[d] Reproducibility refers to the results of independent determinations carried out on a sample by analyzing five replicates of the sample at different periods of time.
[e] n = 5.
[f] Standard deviation.
[g] Coefficient of variation which calculated by the standard deviation divided by the mean.

were 97.8 ± 1.64, 95.8 ± 3.03, 98.0 ± 2.12, and 97.0 ± 2.81 for α-, β-, γ-, and δ-T, respectively, based upon five assays. Concentrations of the tocopherols were calculated from the peak area determined by the Waters 764 integrator (Millipore Corp., Cary, N.C., USA).

Statistical Analysis

Statistical analysis (two-way analysis of variance, ANOVA) was performed by using the SAS Statistical Analysis System (SAS Ins., 1990). Statistical significance between groups was determined by Duncan's test at $\alpha = 0.05$.

RESULTS AND DISCUSSION

Effect of peanut butter manufacture on vitamin E

The tocopherol composition of raw peanuts and peanut butter is given in Table 2-2. In raw runner peanuts, α- and γ-T were the major vitamin E forms present, comprising 94.6% of the total vitamin E on a weight basis. The mean α- and γ-T levels were 11.0 and 10.3 mg/100 g. This value, based on 27 samples, falls in the mid-range of previously reported α-T levels for raw, runner peanuts (Hashim et al., 1993a, 1993b; Lee et al., 1998). Statistical differences did not exist based upon the two crop years for any of the tocopherols and total tocopherol in raw peanuts ($p > 0.05$).

There were no significant differences between raw peanuts and peanut butter for any of the tocopherol levels except for γ-T ($p > 0.05$). Overall mean γ-T value of peanut butter was lower by 6% than that of raw peanuts. A statistical significant decrease in total tocopherol level was observed for peanut butter of 1998 crop year ($p < 0.05$), but it was numerically minor (about 3%). No statistical difference or little changes in tocopherol

Table 2-2. Vitamin E Contents of Runner-Type Raw Peanuts and Peanut Butter (mg/100g edible wt) [I]

Samples	Crop year	α-T	β-T	γ-T	δ-T	Total
Raw peanuts	1998 (n=13)	10.8 ± 0.75^{aA}	0.4 ± 0.05^{aA}	10.2 ± 0.92^{aA}	0.9 ± 0.12^{aA}	22.2 ± 1.16^{aA}
	1999 (n=14)	11.2 ± 0.99^{aB}	0.4 ± 0.10^{aB}	10.5 ± 0.86^{aB}	0.7 ± 0.43^{aB}	22.8 ± 2.40^{aC}
	Overall mean [II]	11.0 ± 0.89^{C}	0.4 ± 0.09^{C}	10.3 ± 0.89^{C}	0.8 ± 0.33^{C}	22.5 ± 1.35^{D}
Peanut butter	1998 (n=7)	10.4 ± 0.57^{bA}	0.5 ± 0.16^{bA}	9.7 ± 0.94^{bA}	0.9 ± 0.15^{bA}	21.5 ± 1.81^{bB}
	1999 (n=5)	9.9 ± 1.27^{bB}	0.4 ± 0.06^{bB}	9.6 ± 0.42^{bB}	0.7 ± 0.45^{bB}	20.6 ± 2.20^{bC}
	Overall mean	10.2 ± 0.91^{C}	0.4 ± 0.13^{C}	9.7 ± 0.74^{D}	0.8 ± 0.30^{C}	20.1 ± 1.69^{D}

[I] All samples were assayed in duplicate.
Values in the same column of each sample (raw peanuts, peanut butter) with the same superscript small letter are not statistically different ($p > 0.05$). Values in the same column with the same superscript capital letter of one crop year are not statistically different ($p > 0.05$). Overall mean values in the same column with the same superscript capital letter are not statistically different ($p > 0.05$).
[II] Mean and standard deviation were calculated by using all sample data of 1998 and 1999 crop years.

levels between raw peanuts and peanut butter indicates that little loss is attributable to the roasting and milling steps.

To investigate the degree of contribution of other ingredients to the tocopherol levels of peanut butter, tocopherols of peanut oils and stabilizer (hydrogenated rapeseed oil) were determined (Table 2-3). α- and γ-T of stabilizer were 12.0 and 16.0 mg/100 g, respectively. The mean α- and γ-T levels of were 11.0 and 10.3 mg/100 g for six commercial RBD peanut oils. Based on this experimental data, actual tocopherol loss of peanuts by peanut butter manufacture was calculated (Table 2-4). All information on the formulation was provided by Tara Foods. Since other ingredients including peanut oil, stabilizer, sucrose and salt were added at the final milling stage step before filling, it was assumed that net tocopherol loss is from tocopherol losses of raw peanuts only before mixing with other ingredients and the tocopherol losses of ingredients are excluded. Although stabilizer and peanut oils have high levels of α- and γ-T, their contribution to the final peanut butter was about 4 and 5% for α- and γ-T of peanut butter, respectively, due to low percentage of composition. Calculated tocopherol losses of peanuts by peanut butter manufacture were below 6% for all tocopherols. In other words, about 94% vitamin E present in raw peanuts was retained in peanut butter. β- and δ-T seem to be more stable to peanut butter manufacture compared to α- and γ-T. Calculated values of vitamin E retention based on the data collected on 12 processing runs over two years convincingly shows that vitamin E of peanuts are highly stable to peanut butter manufacture comprising roasting which is conducted at the range of 275 to 385 °F and milling.

Vitamin E composition of commercial peanuts and peanut products

Tocopherols of commercial peanuts and peanut products are shown in Table 2-3. All commercial peanuts and peanut products were high in α-T although their α-T levels

Table 2-3. Tocopherol Content of Commercial Peanut Products and Stabilizer (mg/100g edible wt)[a]

Sample	α-T	β-T	γ-T	Δ-T	Total
Raw peanuts (n=5)	8.2 ± 2.39	0.3 ± 0.19	7.5 ± 2.32	0.6 ± 0.42	16.6 ± 5.17
Roasted peanuts (n=14)	4.1 ± 1.50	0.2 ± 0.11	6.2 ± 1.75	0.6 ± 0.31	11.3 ± 3.53
Peanut butter (n=7)	9.4 ± 1.47	0.3 ± 0.17	9.7 ± 0.57	0.8 ± 0.14	20.3 ± 1.99
Reduced fat peanut butter (n=4)	5.5 ± 2.20	0.2 ± 0.05	7.9 ± 0.87	0.7 ± 0.22	14.3 ± 2.94
RBD Peanut oil [b] (n=6)	12.3 ± 1.07	0.6 ± 0.21	11.8 ± 2.64	1.0 ± 0.28	25.7 ± 5.99
Peanut butter stabilizer (n=1) [c]	12.0	0.1	16.0	2.3	30.4

[a] Mean of duplicate measurements.
[b] Refined, bleached and deordorized peanut oil.
[c] The stabilizer (hydrogenated rapeseed oil) used for peanut butter manufacture.

Table 2-4. Estimated Percentage of Vitamin E Retention of Peanuts during Peanut Butter Manufacture [a]

	Unit	Tocopherols [b]				
		α-T	β-T	γ-T	δ-T	Total
Ingredients (%) [c]		----------------------		mg/unit weight (dry wt)	--------------------	
Peanuts (88.0)[d]	88.0 g	10.30	0.37	9.64	0.75	21.07
Peanut oil (2.0) [e]	2.0 g	0.25	0.01	0.24	0.02	0.51
Stabilizer (1.7) [f]	1.7 g	0.20	-	0.27	0.04	0.52
Sucrose (7.1)	7.1 g	-	-	-	-	-
Salt (1.2)	1.2 g	-	-	-	-	-
Peanut Butter (100) [g]	100 g	10.75	0.39	10.15	0.81	22.10
Manufactured peanut butter [h]	100 g	10.2	0.4	9.7	0.8	21.1
% Vitamin E loss [i]	%	5.32	-	4.69	1.07	4.74
% Vitamin E retention [j]	%	94.7	100.0	95.4	98.9	95.3

[a] Vitamin E retention of peanuts during peanut butter manufacture was calculated based on the mean of experimental data in this study.
[b] Tocopherols in corresponding portion of ingredients. Data was calculated from the mean of experimental data in table 2-2 and 2-3.
[c] Ingredients of commercial peanut butter. Formulation of ingredient was provided by Tara Foods, Albany, Ga.
[d] Overall mean of raw peanuts in Table 2-2 was used. Moisture content (6.5%) was used to calculate the values in dry basis.
[e,f] Tocopherols of corresponding amount of ingredients based on data in Table 2-3.
[g] Ideal peanut butter. Numerical sum of all ingredients.
[h] Overall mean of peanut butter in Table2-3.
[i] Tocopherol loss (%) of peanuts by peanut butter manufacture. Value was calculated by (g-h)/d*100 based on the assumption that tocopherol loss during peanut butter manufacture occurs only in tocopherols from raw peanuts.
[j] % Vitamin E retention = 100 - % vitamin E loss.

were slightly lower than those of freshly manufactured products. The mean α-T values (mg/100 g) were 8.2 for commercial raw (n=5), and 4.1 for roasted peanuts (n=14), 9.4 for peanut butter, 5.5 for reduced fat peanut butter (60% peanut) and 12.3 for peanut oils. The α-T levels obtained in this study for raw and roasted peanuts and peanut products are in the midrange of those reported by others: 3.55-7.22 for dry-roasted peanuts and 8.15-12.27 for peanut butter (Lee et al., 1998); 10.05-12.21 for raw peanuts (Hashim et al., 1993a); 12.93 and 10.72 for Florunner and Sunrunner raw peanuts (Hashim et al., 1993b); 10.89 for raw peanuts (Piironen et al., 1986). USDA Nutrient Data Bank (2002) reported 9.13 for raw peanuts, 7.41-7.81 for roasted peanuts and 10.0 for peanut butter in α-TE unit/100g.

ACKNOWLEDGEMENTS

This study was supported by funds received from the Georgia Traditional Industries Program and from the University of Georgia, College of Agricultural and Environmental Sciences. The authors thank Robert Karn of Tara Foods for advice and Tara Foods/The Kroger Company for raw peanuts and peanut products of known history.

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CHAPTER IV

EFFECTS OF DRY ROASTING ON VITAMIN E CONTENT OF PEANUTS AND PEANUT OILS PREPARED FROM ROASTED PEANUTS AND MICROSTRUCTURE OF RAW AND ROASTED PEANUTS

INTRODUCTION

Nut consumption is related to low incidence of coronary heart disease (Fraser et al., 1992, 1995; Sabatè and Fraser 1993, 1994; Hu et al., 1998, 1999, 2001; Kris-Etherton, 2001; Ellsworth et al., 2001; Albert and Willet, 2002). Especially, the Nurses' Health Study (Hu et al., 1998) showed that frequent peanut consumption is associated with a low risk of CHD. O'Byrne et al. (1997) reported that a low fat diet supplemented with peanuts improved serum lipoprotein profiles when compared to a regular low fat diet for postmenopausal women with high serum cholesterol levels. The effects of nuts on CHD risk may be through multiple mechanisms due to the variety of nutrients and bioactive substances present in nuts (Kris-Etherton et al., 2001). Favorable fatty acid composition and other intrinsic components including folate, vitamin B6, vitamin E, selenium, sterols and phytochemicals are considered as possible components in peanuts showing the protective effects.

Peanuts contain approximately 50% oil composed of 81% of unsaturated fatty acids of which about 39% is polyunsaturated (USDA, Nutrient Data Bank, 2002). Recent reports (Kris-Etherton et al., 1999; Hu et al., 2001) indicated that replacing saturated fat with unsaturated fat is more effective in lowering risk of CHD than simply reducing total fat consumption. Characteristics of peanut lipids with high mono- and polyunsaturated fatty acid contents are desirable to decrease CHD risk but make peanuts susceptible to oxidation. Peanuts are an excellent source of vitamin E. Due to the role of vitamin E as an antioxidant, vitamin E stability may be related to oxidative stability of peanuts which can be affected by processing and storage.

Peanuts are processed into peanut butter, salted products, confections, roasting stocks and peanut oils. To produce these peanut products, peanut kernels are subjected to roasting. Roasting develops desirable flavor that ultimately enhances the overall palatability of the peanut products. Although the roasting reduces moisture content and

inactivates lipoxygenase present in raw peanuts which cause lipid oxidation, it can destroy nutrient components and decrease shelf-life (St. Angelo et al., 1975, 1977; Oupadissakoon and Young, 1984; Basha and Young, 1985). Roasting decreases the levels of essential amino acids in peanuts such as lysine, threonine, and methionine (McOsker, 1962; Basha and Young, 1985; Damame et al., 1990) and destroys the thiamin up to 90%, while niacin, choline, and riboflavin are little affected by roasting (Fournire et al., 1949). The nutrient compositional changes of peanuts associated with oil, protein, carbohydrate, mineral and water-soluble vitamin contents during roasting have been reported (Derise et al., 1974; Damame et al., 1990).

There are conflicting results on the effect of roasting on vitamin E stability of oilseeds. The levels of vitamin E in rice bran, sesame, soybean and sunflower oils that was reported to decrease by roasting (Shin et al., 1997; Yoshida and Takagi, 1997; Yoshida et al., 1995, 1999, 2001, 2002). Usually, the levels of vitamin E were still high (> 80% retention) although the tocopherol loss increased as roasting temperature increased. In contrast, increases in vitamin E levels of rice bran, corn fiber, and rice germ oils were observed (Lane et al., 1997; Moreau et al., 1999; Kim et al., 2002). On the other hand, Yen et al. (1990) observed both phenomenon when sesame seeds were at 180, 200, 220, 240 and 260 ºC for 30 min. They reported that the level of γ-T in oils increased by roasting temperatures up to 200 ºC but fell with higher roasting temperature, resulting in the highest level of γ-T in oils prepared from seeds roasted at 200-220 ºC (Yen et al., 1990). However, no information is available on effect of roasting on tocopherol levels of peanuts.

Storage studies on raw and roasted peanuts usually indicate that roasted peanuts are less stable to oxidation than raw peanuts (St. Angelo and Ory, 1972, 1975; St. Angelo et al., 1977; Yuki et al., 1978). This may be due to both chemical - destruction of natural antioxidants, breakdown of fatty acids (Damame at al., 1990) and physical changes - disruptions of lipid bodies, membranes and cellular compartmentalization (Young and

Schadel, 1993) - of peanuts during roasting. Young and Schadel (1990, 1993) showed the changes in microstructure of peanuts – cell wall, cytoplasmic network, protein bodies, starch granules, parenchyma tissue, epidermal tissue, surface of cotyledons – affected by roasting time at 160 ºC and roasting methods (oven and oil roasting). Vitamin E stability was not studied in this research.

The purpose of this study was to investigate changes in vitamin E content and microstructure of peanuts roasted at different temperatures (140, 150 and 160 ºC) and relate the changes to other physical characteristics including moisture and color. In addition, vitamin E content of peanut oils prepared from the roasted peanuts was determined.

MATERIALS AND METHODS

Sample preparation

Runner-type raw peanuts (halves) from the 2001 crop year were obtained from Tara Foods, Albany, GA. Raw peanuts were stored at –16 ºC. Before roasting, peanuts were allowed to equilibrate to room temperature overnight. The equilibrated peanuts (300g) were then placed in a single layer in a plate and roasted in a preheated air forced oven (Lincoln Impinger-Model 1450, Foodservice Products, Inc., Fort Wayne, IN, USA). Roasting was at 140, 150, and 160 ºC for 10, 15, and 20 min, separately. Roasted peanuts were cooled at room temperature and then stored in a freezer at -16ºC until assay. All procedures were duplicated. Moisture contents were determined for raw and roasted peanuts by using AOAC Official Method 925.40 (1995).

Color measurement

Hand-blanched raw and roasted peanuts (30 g) were ground in a coffee mill for 90 s to produce a peanut paste with a uniform texture like peanut butter. The peanut paste was compacted in three disposable tissue culture dishes (35 × 10 mm (diameter × depth), Becton Dickinson, NJ, USA) without air holes on the surface. Color of the surface of the peanut paste was measured using a Minolta CR-300 colorimeter (Minolta Corp., Ramsey, NJ, USA) which directly gives CIELAB color values-L*, a*, b*. L* value represents light- and darkness with a range black (0) to white (100), a* value represents the green-red spectrum with a range of green (-100) to red (+100), while b* value represents blue-yellow spectrum with a range of blue (-100) to yellow (+100).

Vitamin E Extraction

To determine tocopherols in peanuts, direct solvent extraction was coupled with HPLC (Lee et al., 1998). Peanut samples (30 g) were ground in a coffee mill for 10-20 s and 1.0 – 1.5 g of ground sample was weighed into a 125 mL round bottom glass bottle. Four millilitres of hot deionized water (80ºC) were added to the sample and then mixed with a spatula. Ten millilitres of isopropanol were added to the mixture and thoroughly mixed. Approximately, 5 g of anhydrous magnesium sulphate were added followed by 25 mL of extraction solvent (hexane:ethylacetate, 90:10, v/v), containing 0.01% butylated hydroxytoluene (w/v). The mixture was homogenized with a Polytron ^{R} homogenizer for 90 s at medium speed. The homogenized mixture was filtered through a medium porosity glass filter in a vacuum bell jar filtration apparatus (Kontes, Vineland, NJ, USA). The filter cake was transferred to the same extracting bottle and then 5 mL of isopropanol and 30 mL of extracting solvent were added. Homogenization and filtration were repeated. The combined filtrate was transferred to a 100 mL volumetric flask and adjusted to 100 mL with extracting solvent. After mixing, a 1.0 mL aliquot of the combined filtrate was taken into a 10 mL test tube and evaporated under nitrogen gas. The solids were

redisolved with 2 mL of *n*-hexane and injected onto the HPLC system after filtration through a 0.45 μm nylon membrane filter (MSI Inc., Westboro, MA). Peanut oils were prepared by hydraulic pressing of raw and roasted peanut samples in a Carver press (Carver, Inc., Wabash, IN, USA). About 30 g of peanut samples were placed in the cylinder of the Carver press and pressed to obtain the oil under pressure of 70-100 kg/cm^2 for 30 min. Tocopherols in peanuts oils were directly determined after appropriate dilution with in *n*-hexane. All steps were done under yellow light and all solvents were HPLC grade.

HPLC

The normal phase HPLC system consisted of a LC-6A pump, equipped with a Shimadzu RF-10A spectrofluorometric detector (Shimadzu Corp., Columbia, Md., U.S.A.), a Spectra Series AS 100 autosampler (Thermo Separation Products Inc., San Jose, Calif., U.S.A.), and a 25 cm× 4 mm, 5 μm Li-Chrosorb Si60 column (Hibar Fertigsaule RT. Darmstadt, F.R. Germany) equipped with a precolumn packed with Perisorb A 30-40 μm (Darmstadt, F.R. Germany). The isocratic mobile phase contained 0.8% isopropanol in *n*-hexane (HPLC grade, J.T. Baker Chemical Co., Phillipsburg, N.J.). Prior to use, the mobile phase was filtered through a 0.22 μm nylon membrane filter (MSI Inc., Westboro, MA) and de-gassed by stirring under vacuum. The flow rate was 1.0 mL/min. For the determination of tocopherol homologs, the excitation and emission wavelengths were set at 290 and 330 nm, respectively. The tocopherols in samples were identified by comparison with standard tocopherols. Tocotrienols were not found in peanuts. Concentrations of tocopherols were calculated from the chromatographic peak area determined by the Waters 764 integrator (Millipore Corp., Cary, NC, USA).

Fixation and sample preparation for scanning electron microscopy (SEM)

After hand blanching, 3 mm cube blocks were cut from raw and roasted peanuts and fixed in a Karnovsky's solution (1965) as modified by Young and Schadel (1989). The sample blocks were fixed with a freshly made fixative (3.0% formaldehyde and 4.0% glutaraldehyde in 0.1 M sodium phosphate buffer, pH 7.0) for 36 h at 4ºC (Young and Schadel, 1989). After four washes of 0.1M sodium phosphate buffer (PBS), the samples were post-fixed for 1 hr in PBS buffered 1.0% osmium tetroxide at 4ºC followed by three washes of distilled water. The samples were dehydrated at room temperature at 15 min intervals in a graduated series of aqueous ethanol (10, 25, 50, 70, 85 and 95%). Finally, the samples were dehydrated in absolute ethanol twice at 30 min intervals. Dehydrated peanut samples were dried to the critical point in a SamDri 805 CPD (Tousimis), mounted on aluminum specimen stubs, and coated with a layer of gold palladium to 20 nm in thickness. The tissue was viewed with a LEO 982 field-emmission SEM (LEO Ectron Microscopy, Inc., Thornwood, NY, USA).

Statistics

The experiment was duplicated and all assays were completed in triplicate. Data was expressed in the mean of determinations ± S.D. Experimental data were analyzed by ANOVA and Duncan's multiple range test at the level of $\alpha = 0.05$.

RESULTS AND DISCUSSION

Moisture

Moisture contents of raw and roasted peanuts were shown in Table 3-1. Moisture content of raw peanuts was 6.5 ± 0.03%. During the initial 10 min of roasting, moisture of raw peanuts rapidly decreased and gradually decreased after 10 min. The rate of

Table 3-1. Moisture Content in Raw and Roasted Peanuts [I, II]

Samples		Moisture Content (%)
Raw peanuts		6.5 ± 0.03
Roasted peanuts		
Roasting temp	Roasting time	
140 °C	10 min	2.8 ± 0.10
	15 min	2.1 ± 0.04
	20 min	1.6 ± 0.03
150 °C	10 min	1.9 ± 0.09
	15 min	1.4 ± 0.10
	20 min	1.0 ± 0.02
160 °C	10 min	1.6 ± 0.14
	15 min	1.2 ± 0.08
	20 min	0.6 ± 0.04

[I] Mean ± SD of three determinations from duplicate experiments.

decrease was more rapid at higher roasting temperatures. After 20 min of roasting, moisture contents of roasted peanuts were 1.6 ± 0.03, 1.0 ± 0.02 and 0.6 ± 0.04% at 140, 150 and 160 ºC, respectively. Each moisture content for raw and roasted peanuts was used to give vitamin E content on a dry weight basis. Since roasting is conducted at high temperature over the boiling point of water, moisture is removed as steam. During moisture removal, Young and Shadel (1991) reported that pock-marks were observed on the surface of peanuts presumably due to the escape of internal steam.

Color

Changes in color of peanuts during roasting were determined and expressed as CIELAB L*, a*, b* values (Table 3-2). The initial L*, a* and b* values of the raw peanuts were about 65, 0 and +15, respectively, indicating that raw peanuts have a quite light color with a yellow hint. The L* value of peanuts decreased with roasting time, indicating darkening. The rate of darkening increased with roasting temperature. Moss and Otten (1989) who studied the relationship between color development and moisture content during roasting of peanuts reported an induction period for the L* value of roasted peanuts at initial stages of roasting followed by a decrease, fitting a 2nd degree polynomial. During initial roasting at 140ºC, the L* value slightly increased followed by a decrease, indicating the induction period. Since the length of the induction period for color formation is inversely proportional to roasting temperature (Moss and Otten, 1989; Özdemir and Devres, 2000), the induction period for 150 and 160ºC should be shorter than 10 min, the first point of observation in this study. That is why L* value decreased over roasting time without an initial increase at 150 and 160ºC. In general, the L* color scale, relative lightness of a product, is used to determine the degree of roast of the peanuts in industry.

Browning of peanuts during roasting was associated with an increase in a* (from from near 0 in unroasted samples to 8.3 at the highest level of roasting) and an increase in

Table 3-2. Color of raw and roasted peanuts in CIELAB color scale (L*a*b*) [I, II]

Samples		CIELAB color values		
		L*	a*	b*
Raw peanuts		65.6 ± 0.03	0.3 ± 0.01	15.2 ± 0.05
Roasted peanuts				
Temp (°C)	Time (min)			
140°C	10	66.5 ± 0.49	0.5 ± 0.15	19.7 ± 0.17
	15	65.3 ± 0.36	1.8 ± 0.06	22.9 ± 0.33
	20	64.1 ± 0.38	2.6 ± 0.04	24.4 ± 0.31
150°C	10	64.4 ± 0.50	2.7 ± 0.06	24.5 ± 0.22
	15	62.7 ± 0.51	3.7 ± 0.29	25.6 ± 0.65
	20	61.3 ± 0.27	4.5 ± 0.29	26.8 ± 0.38
160°C	10	60.3 ± 0.78	5.5 ± 0.15	28.0 ± 0.72
	15	56.7 ± 0.22	7.2 ± 0.32	29.4 ± 0.77
	20	54.8 ± 0.46	8.3 ± 0.08	30.1 ± 0.40

[I] Mean ± SD of three determinations from duplicated experiments.
[II] Values in the same column with different superscript letters are significantly different ($p < 0.05$).

b*. Higher temperatures showed the rapid rates of both a* and b*color changes. A tendency of a little drop in the a* and b* values for the first few minutes of roasting which was observed by Moss and Otten (1989) was not noted in this study because the first sampling time was at 10 min. Moss and Otten (1989) also reported that there was a high correlation between drying and color b* value of peanuts during roasting at the range of 157 to 171 ºC ($b^* = 25.3\ (M/Mo)^2 - 37.93\ (M/Mo) + 30.97$, $R^2 = 0.9462$, where M = moisture of roasted peanuts, Mo = moisture of raw peanuts). In this study, a similar phenomenon was observed between M/Mo and b* value (Figure 3-1). Moisture ratio (M/Mo) and color b* value of peanuts roasted at 140 to160 ºC showed a correlation of $b^* = 21.61\ (M/Mo)^2 - 40.62\ (M/Mo) + 34.12$ ($R^2 = 0.9123$). Correlation coefficients for both a* and L* values with moisture ratio were relatively low ($R^2 < 0.82$).

Tocopherols of peanuts

Tocopherol contents of raw and roasted peanuts were determined by using a direct solvent extraction and HPLC (Table 3-3). Oil extraction yields (%) by direct extraction for raw and roasted peanuts were 55.8 ± 0.02 for raw, 55.8 ± 0.74 for roasted peanuts (55.8 ± 0.68 for 140ºC, 55.6 ± 0.85 for 150ºC, and 56.3 ± 0.62 for 160ºC). There was no significant difference in oil extraction yield between raw and roasted peanuts ($p > 0.05$). The initial values of α-, β-, γ- and δ-T in raw peanuts were 10.9, 0.5, 10.8 and 1.1 mg/ 100g (dry basis), respectively. Overall effects of roasting temperature and time were significant on tocopherols except for β-T ($p < 0.05$). No significant change was observed in the levels of tocopherols in peanuts during roasting at 150 ºC for 20 min. At 160 ºC, the level of total tocopherol significantly decreased during the initial 10 min of roasting while there was no significant loss with roasting time up to 20 min. About 5, 12, 20, and 10% losses of α-, β-, γ- and δ-T occurred during 20 min of roasting, respectively. α-T was

Figure 3-1. Relationship between moisture content ratio (M/Mo) and CIE b* values of peanuts during roasting at 140, 150 and 160 ºC.

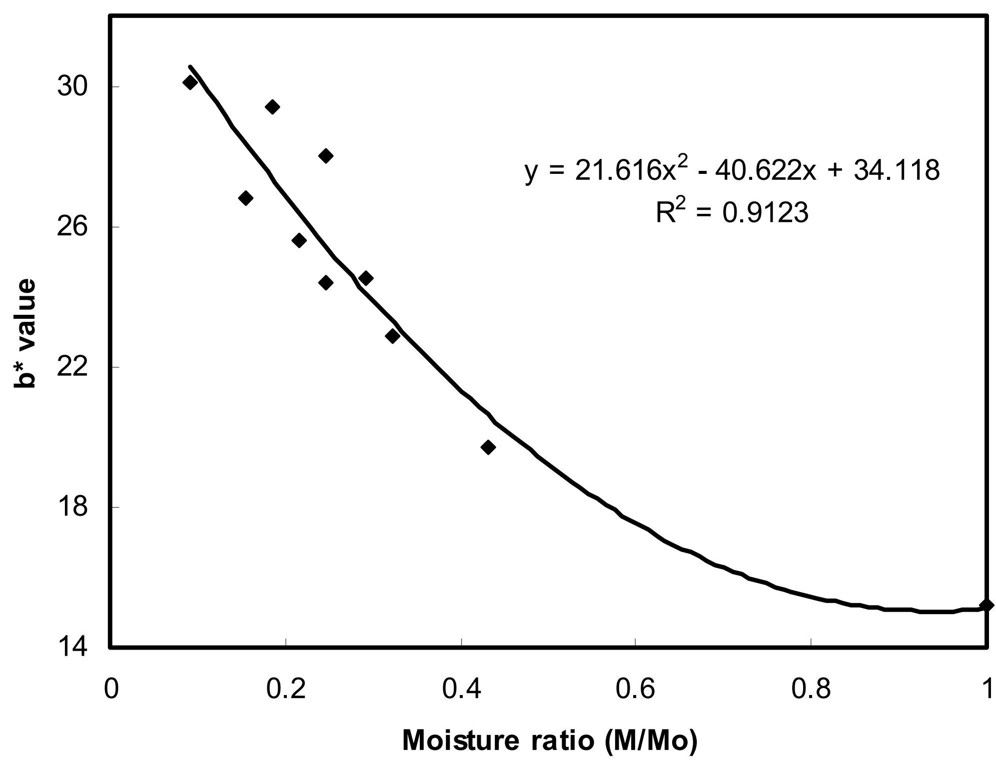

y = 21.616x2 - 40.622x + 34.118
R2 = 0.9123
b* value
Moisture ratio (M/Mo)
30
26
22
18
14
0
0.2
0.4
0.6
0.8
1

Table 3-3. Vitamin E Content of Raw and Roasted Peanuts by Direct Solvent Extraction [I, II]

Peanut samples		Vitamin E content (mg/100g, dry basis)				
		α-T	β-T	γ-T	δ-T	Total-T
Raw		10.9 ± 0.18 (100)[III] [a]	0.5 ± 0.04 (100) [ab]	10.8 ± 0.16 (100)[b]	1.1 ± 0.04 (100)[bc]	23.3 ± 0.32 (100)[b]
Roasted						
Temp (°C)	Time (min)					
140°C	10	10.5 ± 0.39(96) [abc]	0.5 ± 0.02 (100)[ab]	11.0 ± 0.31 (102)[b]	1.1 ± 0.04 (100)[bc]	23.2 ± 0.66 (100)[b]
	15	10.9 ± 0.37 (100) [ab]	0.5 ± 0.08 (100)[a]	11.9 ± 0.38 (110)[a]	1.4 ± 0.06 (127)[a]	25.1 ± 0.54 (108)[a]
	20	10.9 ± 0.37 (100) [ab]	0.5 ± 0.09 (100)[a]	12.3 ± 0.54 (114)[a]	1.2 ± 0.04 (109)[b]	25.3 ± 0.71 (109)[a]
150°C	10	10.7 ± 0.33 (98) [abc]	0.5 ± 0.03 (100)[ab]	10.6 ± 0.55 (97)[b]	1.0 ± 0.10 (91)[cd]	22.8 ± 0.39 (98)[b]
	15	10.5 ± 0.10 (96) [abc]	0.5 ± 0.02 (100)[ab]	10.6 ± 0.32 (97)[b]	1.1 ± 0.03 (100)[cd]	22.6 ± 0.19 (97)[b]
	20	10.6 ± 0.16 (97) [abc]	0.5 ± 0.04 (100)[ab]	10.7 ± 0.20 (98)[b]	1.1 ± 0.04 (100)[bc]	22.9 ± 0.07 (98)[b]
160°C	10	10.4 ± 0.26 (95) [bc]	0.5 ± 0.06 (100)[ab]	8.4 ± 0.58 (78)[c]	1.0 ± 0.04 (91)[d]	20.3 ± 0.87 (87)[c]
	15	10.2 ± 0.08 (94) [c]	0.4 ± 0.03 (80)[b]	8.4 ± 0.43 (78)[c]	0.9 ± 0.04 (82)[d]	19.9 ± 0.17 (85)[c]
	20	10.2 ± 0.26 (94) [c]	0.5 ± 0.06 (100)[ab]	8.7 ± 0.31 (81)[c]	1.0 ± 0.14 (91)[d]	20.4 ± 0.57 (88)[c]

[I] Mean ± SD of three determinations from duplicated experiments.
[II] Values in the same column with different superscript letters are significantly different ($p < 0.05$).
[III] % retention in parameters

the most stable during roasting while γ-T was the least. On the other hand, roasting at 140 ºC caused a significant increase in the levels of tocopherols of peanuts over roasting time up to 20 min ($p < 0.05$). Increases of 6, 14 and 11% increases compared to the initial values of β-, γ- and δ-T were observed.

A similar phenomenon was observed by Yen et al. (1990) who roasted sesame seeds at 180, 200, 220, 240 and 260 ºC for 30 min. They reported that the level of γ-T in sesame seed oils increased by roasting temperatures up to 200 ºC but fell with higher roasting temperature, resulting in the highest level of γ-T in roasted sesame at 200-220 ºC. Lane et al. (1997) and Moreau et al. (1999) reported increases in the levels of extracted tocopherols and tocotrienols in rice bran and corn fiber by heat pre-treatment at 100-175 ºC. They suggested that a significant amount of these tocopherols and tocotrienols are bound to proteins or linked to phosphate or phospholipids. Heat breaks these bonds, resulting in an increase of extractable tocopherols. Kim et al. (2002) also noted that α- and γ-T in rice germ oil gradually increased as roasting temperature (160-180 ºC) and time (0-15 m) increased.

In contrast, several studies reported decreases in the levels of tocopherols and tocotrienols in oil seeds and soybeans during roasting. Shin et al. (1997) noted a progressive decrease in the levels of tocopherols and tocotrienols in rice bran oil by extrusion at increasing temperature. Yoshida and Takagi (1997) also reported that the levels of tocopherols in sesame seeds decreased by roasting at 160-250 ºC and the decrease was greater as roasting temperature increased. Yoshida et al. (1995, 1999, 2001, 2002) showed decreased levels of tocopherols in sesame, soybean and sunflower seed oils as roasting time increased.

Due to variations in roasting methods (oven, microwave, extrusion), roasting temperature and time, types of samples and oil extraction methods, it is difficult to compare these observations. The above studies suggest that mild heating might increase the extractable tocopherol levels, possibly due to an increase in recovery of tocopherols

bound to other components in oil seeds, but a gradual decrease of tocopherol contents would occur by more severe heat treatments. The degree of roasting such as mild or severe may depend on heat sensitivity of samples due to different sample matrix and components. Based on the hypothesis above, the degree of roasting at 140 and 150 ºC for 20 min is not severe enough to cause a significant vitamin E loss by heating; therefore, any loss of vitamin E may be overshadowed by an increase in amount of extractable tocopherols which is difficult if not impossible to experimentally quantify.

Tocopherols of peanut oils

Oil extraction yields (%) by pressing were 35.9 ± 0.02 for raw and 36.8 ± 1.77 for roasted peanuts. Tocopherol contents of the raw and roasted peanut oils prepared by hydraulic pressing are shown in Table 3-4. The tocopherol contents of the pressed raw peanut oil were 22.7, 1.0, 19.2, and 2.0 mg/100g of oil for α-, β-, γ- and δ-T, respectively. The levels of tocopherols in pressed peanut oils were significantly decreased by roasting at all temperatures ($p < 0.05$). After roasting for 20 min, α-T loss was constant by 16% at all temperature while γ-T loss was greater at higher temperature, resulting in 8, 15 and 19% losses of the initial values at 140, 150 and 160 ºC, respectively. Loss by roasting was the greatest for γ-T followed by α-T, indicating less stability of γ-T to roasting compared to α-T.

The phenomenon of a slight increase in tocopherol levels at 140 ºC was not observed in pressed peanut oils. It may be due to variation in the oil extraction method. Pressing mainly extracts the oils in spherosomes (oil reserve bodies) of the peanut kernels, causing decreased recoveries of membrane bound tocopherols. If a significant amount of tocopherols were in areas from the oil reserve bodies or bound with other components present in peanuts, the levels of extractable tocopherols should decrease, resulting in no apparent increase in tocopherol contents by roasting.

Table 3-4. Vitamin E Content in Oils Extracted from Raw and Roasted Peanuts by Pressing [I, II]

Peanut oil samples		Vitamin E content (mg/100g, dry basis) [II]				
		α-T	β-T	γ-T	δ-T	Total-T
Raw		22.7 ± 0.30 (100) [IIIa]	1.00 ± 0.15 (100) [a]	19.2 ± 0.68 (100) [a]	2.0 ± 0.22 (100) [a]	44.9 ± 1.31 (100) [a]
Roasted						
Temp (°C)	Time (min)					
140°C	10	19.2 ± 0.67 (85) [b]	0.9 ± 0.02 (90) [ab]	18.6 ± 1.09 (97) [ab]	1.9 ± 0.14 (95) [ab]	40.6 ± 1.71 (90) [b]
	15	18.9 ± 0.41 (83) [b]	0.9 ± 0.04 (90)[ab]	18.8 ± 0.84 (98) [ab]	2.0 ± 0.06 (100) [a]	40.6 ± 1.14 (90) [b]
	20	18.7 ± 0.24 (82) [b]	0.9 ± 0.01 (90) [ab]	17.6 ± 0.16 (92) [b]	1.9 ± 0.02 (95) [ab]	39.1 ± 0.41 (87) [bc]
150°C	10	19.5 ± 0.24 (86) [b]	0.9 ± 0.14 (90) [ab]	18.1 ± 1.09 (94) [ab]	1.9 ± 0.12 (95) [ab]	40.4 ± 1.32 (90) [b]
	15	19.1 ± 0.72 (84) [b]	0.9 ± 0.13 (90) [ab]	18.2 ± 0.41 (95) [ab]	1.8 ± 0.08 (90) [abc]	40.0 ± 1.34 (89) [b]
	20	18.9 ± 0.28 (83) [b]	0.9 ± 0.03 (90) [ab]	16.3 ± 0.07 (85) [c]	1.5 ± 0.04 (75) [de]	37.6 ± 0.33 (84) [cd]
160°C	10	19.2 ± 0.22 (85) [b]	0.8 ± 0.16 (80) [ab]	15.9 ± 1.03 (83) [c]	1.8 ± 0.15 (90) [bc]	37.6 ± 1.46 (84) [cd]
	15	19.0 ± 0.81(84) [b]	0.7 ± 0.05 (70) [b]	15.0 ± 0.50 (78) [c]	1.4 ± 0.03 (70) [e]	36.2 ± 1.35 (81) [d]
	20	19.0 ± 0.16 (84) [b]	0.8 ± 0.03 (80) [ab]	15.6 ± 0.71 (82) [c]	1.7 ± 0.03 (85) [cd]	37.0 ± 0.83 (82) [cd]

[I] Mean ± SD of three determinations from duplicated experiments.
[II] Values in the same column with different superscript letters are significantly different ($p < 0.05$).
[III] % Retention in parameters

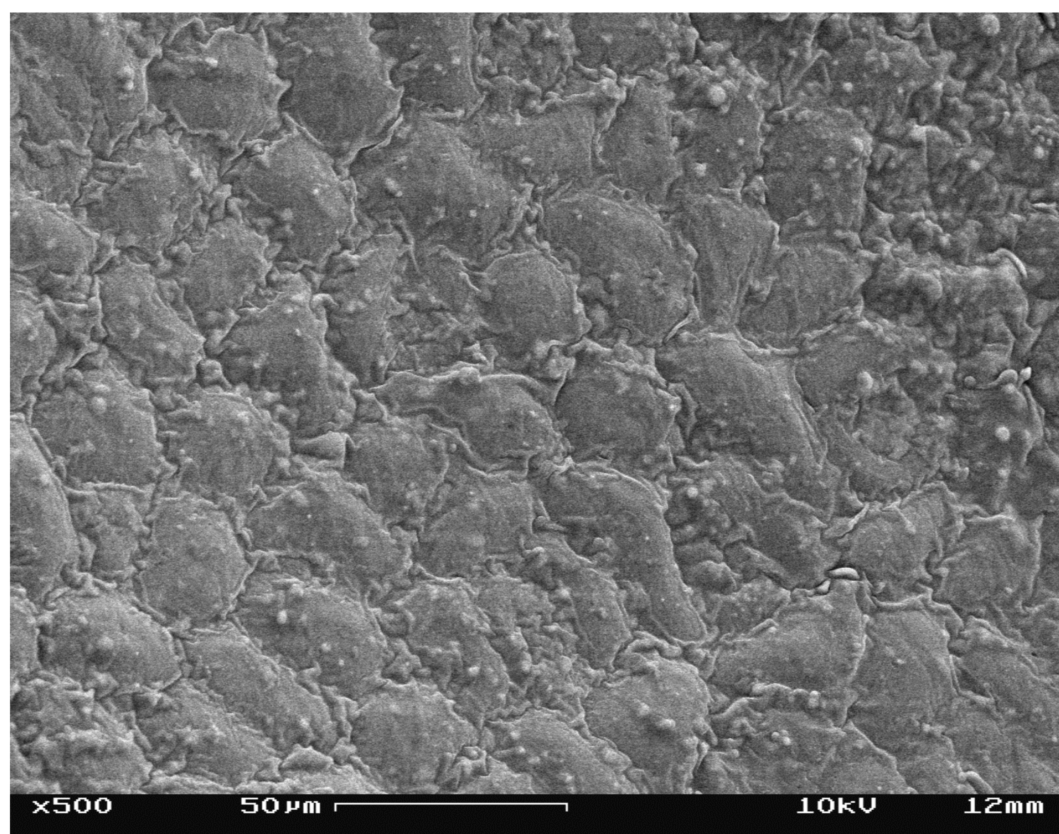
Structure

Changes in microstructures of peanuts roasted at different temperatures (140, 150 and 160 ºC) were examined by using SEM. The raw peanut kernels consist of two cotyledons and the germ. The cotyledon consists of epidermis, vascular and parenchymal tissue. The epidermal tissue is a layer of cells compressing the cotyledon surface which is under layered by the vascular tissue. The parenchyma tissue constitutes the greatest part of the kernel and contains large, isodiametric cells. Figure 3-2 showed the effect of roasting on epidermal cells of the inner surface of peanut cotyledon. Destruction in epidermal cells was more severe at higher roasting temperatures. After roasting at 140 ºC for 20 min, epidermal cells on the surface of a cotyledon became collapsed as some portion of epidermal cells lost their shape (Figure 3-2-B). For roasting at 150 ºC for 20 min, swollen cells were found and no boundary between epidermal cells existed (Figure 3-2-C). After 15 min of roasting at 160 ºC, the characteristic irregular shape of the epidermal cells still remained unchanged although swollen epidermal cells were found (Figure 3-2-A, raw; Figure 3-2-D, roasted). After roasting at 160 ºC for 20 min, it was difficult to recognize individual epidermal cells. Swelling of epidermal cells became severe at high temperatures (Figure 3-2-E).

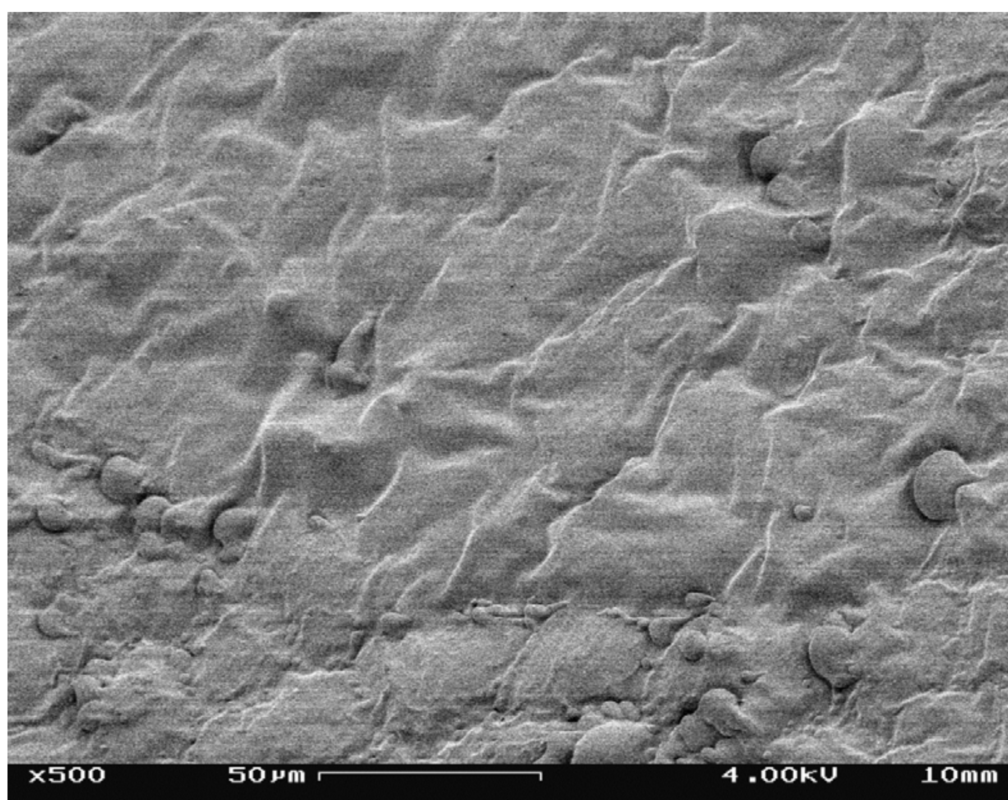
Figure 3-3 shows the cross section of parencyma cells in mid regions of raw and roasted peanut cotyledons. Each parenchyma cell contains lipid bodies, aleurone protein bodies and starch granules. Figure 3-3-(A) showed that parenchyma cells of the cotyledons of raw peanuts had isometric shapes separated by cell walls containing the cellular organells. Middle lamella present between the cell walls makes adjacent parenchyma cells stick together. Cytoplasmic network surrounds these cellular organelles. After roasting at 140 ºC for 20 min, broken cell walls were observed. However, some of cytoplasmic network remained (Figure 3-3-B). At 150 ºC, coagulated suborganells and broken cell wall were observed (Figure 3-3-C). There was no big difference in structure

Figure 3-2. Scanning electron micrograph of epidermal cells of the inner surface of raw and roasted peanuts. (A) - raw, (B) ~ (E) - roasted peanuts: (B) 140 ºC, 20 min, (C) 150 ºC, 20 min, (D) 160 ºC, 15 min, and (E) 160 ºC, 20 min.

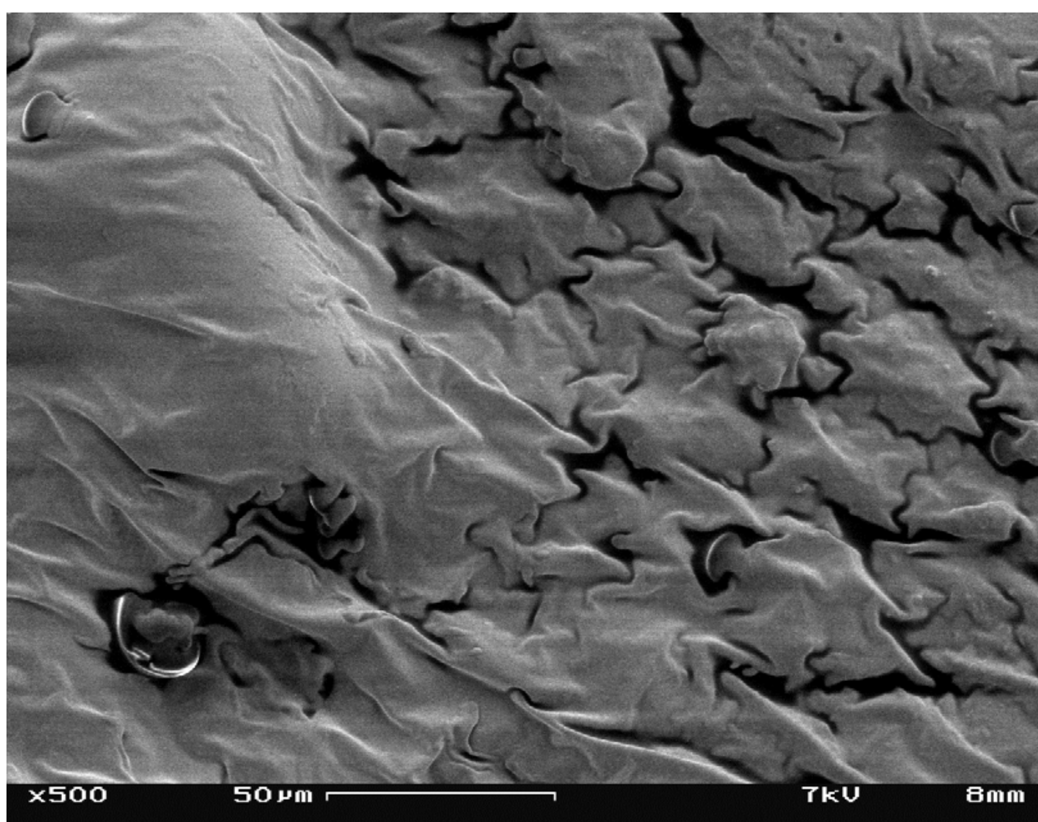
(A) Raw



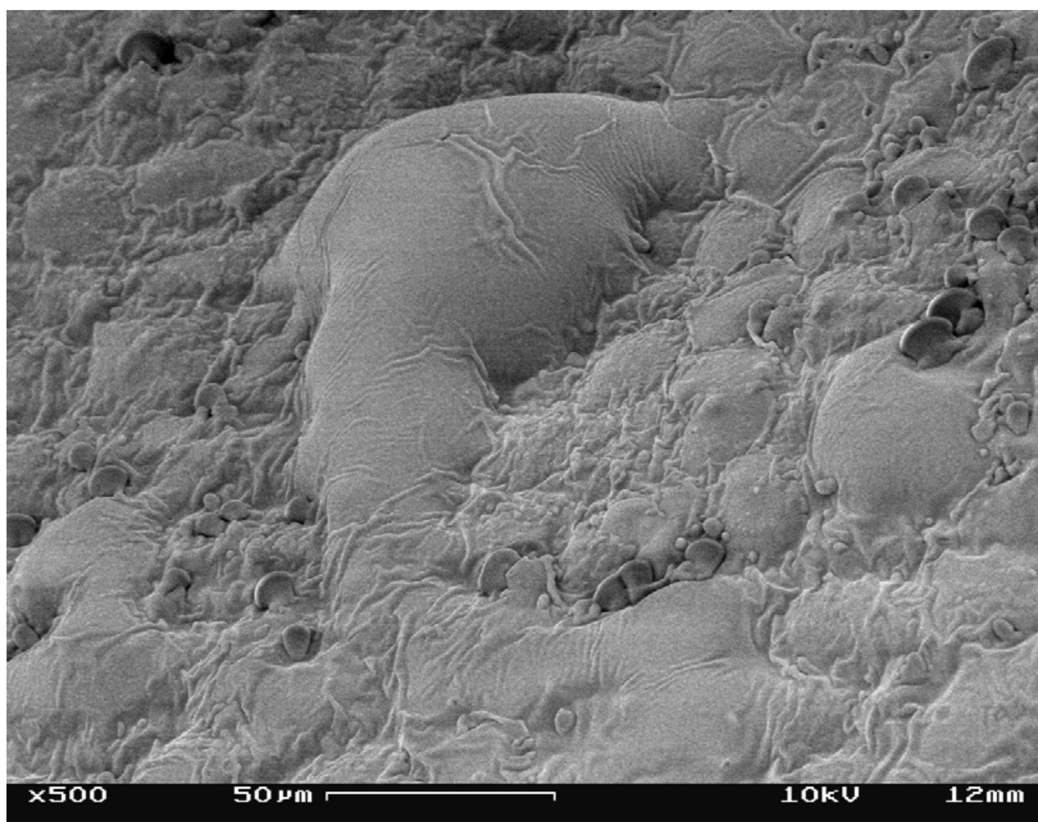
(B) 140ºC-20 min



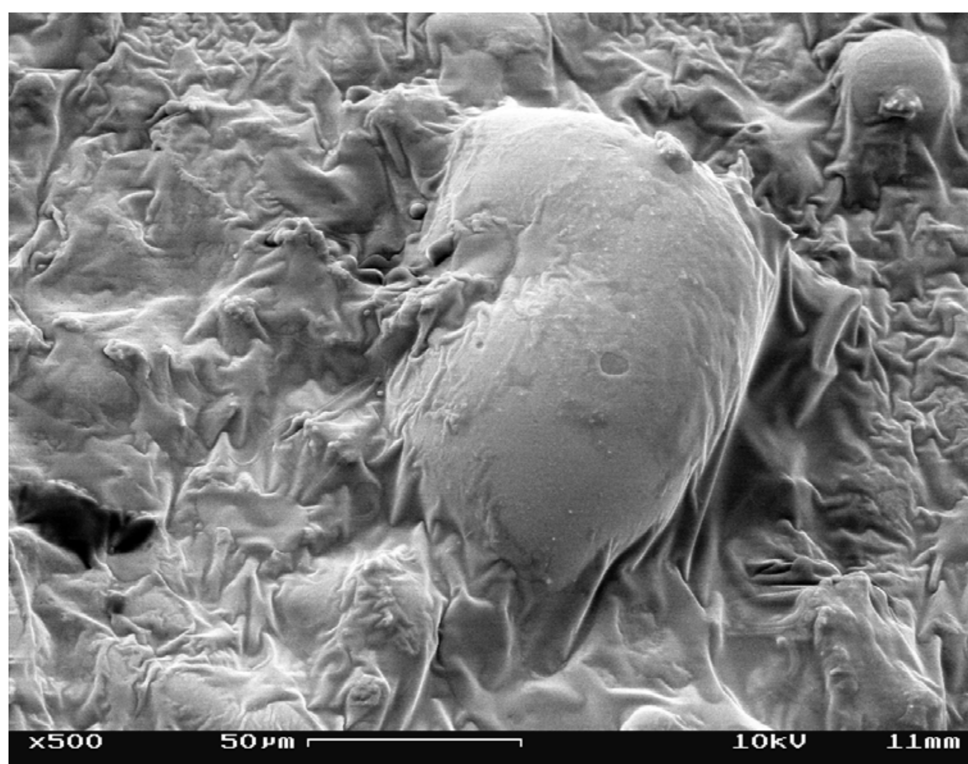
(C) 150ºC-20 min



(D) 160ºC-15 min



(E) 160ºC-20 min



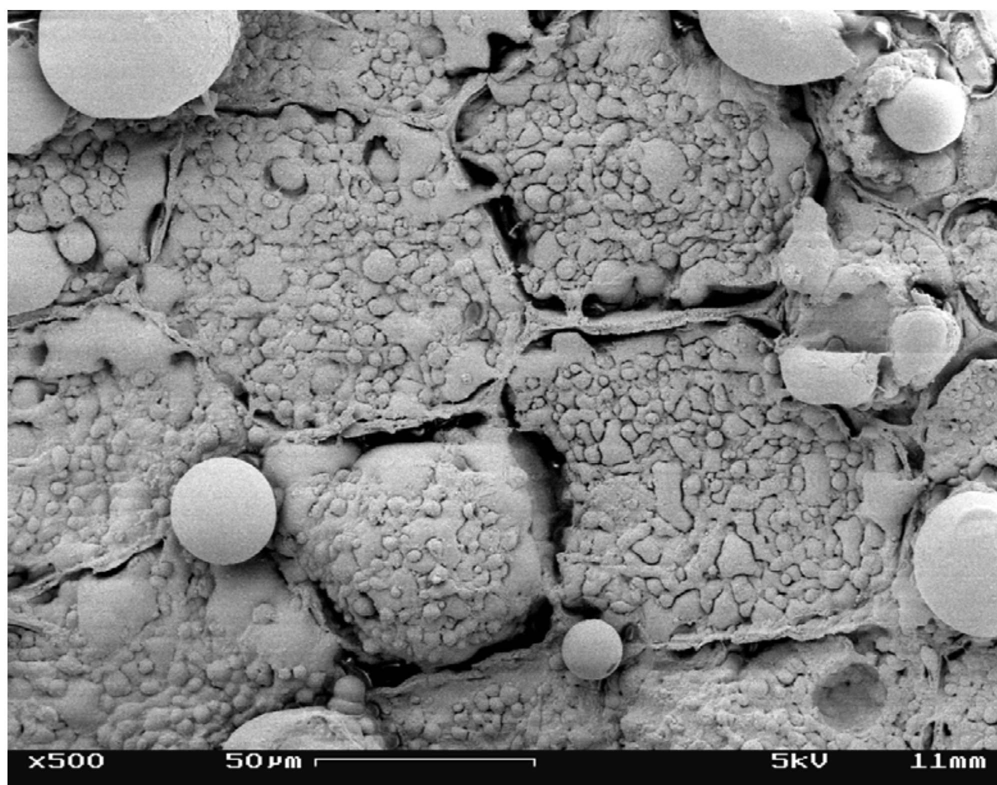
between roasted peanuts at 150 ºC for 20 min and at 160 ºC for 15 min (Figure 3-3-C and D). After roasting at 160 ºC for 20 min, thin and broken cell walls were found due to modification of lamella between cell walls (Figure 3-3-E). Coagulated suborganells were noted in peanuts roasted at 160 ºC.

These thermal modifications in peanut microstructure were in agreement with Young and Schadel's studies (1990, 1993) who studied the microstructure changes in peanut cotyledons affected by roasting time and methods at 160 ºC (oven roasting and oil cooking) with SEM. They showed that microstructure of peanuts was severely destroyed by oil roasting compared to oven roasting. The degree of destruction was great with roasting time. After oven roasting at 160°C for 19 min, disruption of the cytoplasmic network, protein body distension, and the presence of cell wall separation were observed along the middle lamellae (Young and Schadel, 1993). Changes in microstructure of peanuts may be related to vitamin E stability during and after roasting. However, since effects of roasting on characteristics of peanuts can be affected by peanut cultivars, kernel size, roasting conditions and roaster types, it is difficult to directly relate Young and Schadel's observations (1993) on microstructure of roasted peanuts to results on vitamin E stability in this study. In this study, microstructure of peanuts roasted at different temperatures in SEM was studied in order to investigate the relationship between structural damage and vitamin E stability.

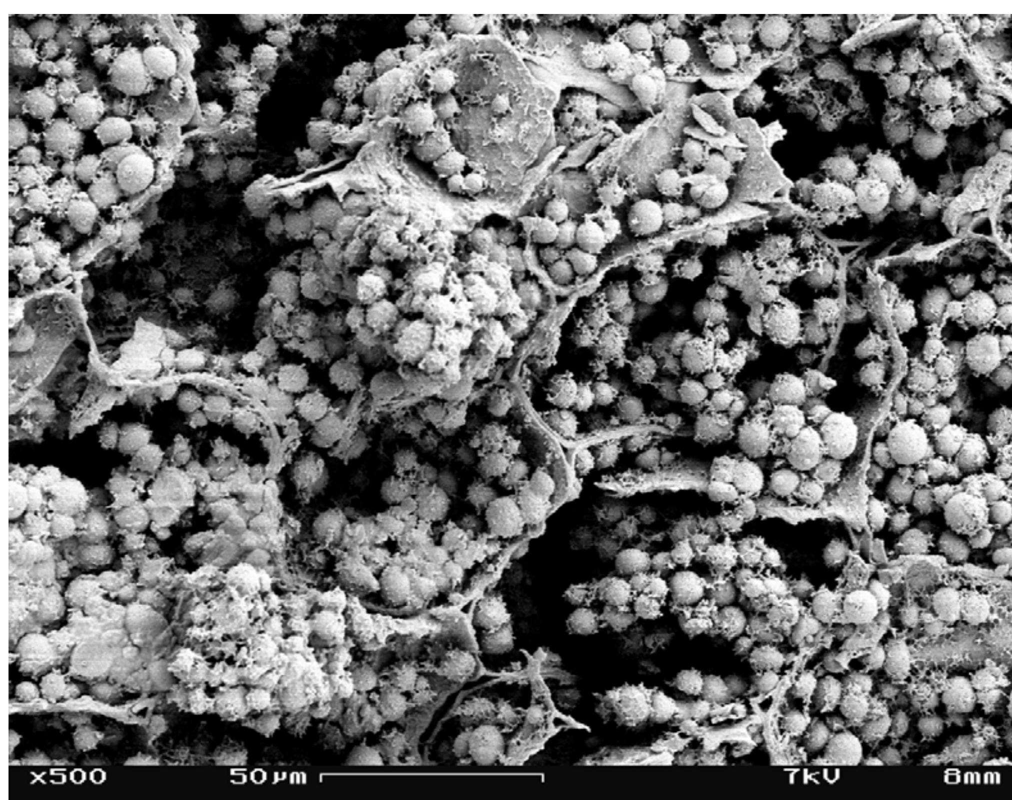
Observations in this study indicate that vitamin E is highly stable to roasting although microstructures of peanuts are severely damaged with increasing roasting temperature. According to Woodroof and Leahy (1940), oil droplets in peanuts are fused into larger and fewer droplets within cells and a small amount of oil escaped into the cell as free oil by dry roasting. These physical changes of peanuts during roasting may contribute much to their susceptibility to oxidation and vitamin E stability during further storage of peanuts. Especially, lipid bodies opened to cytoplasmic environment due to broken cytoplasmic network and cell walls make contact with oxygen more certain. Consequently, although only a slight loss might occur in vitamin E content during

Figure 3-3. Scanning electron micrograph of cross-sections of parenchyma cells of raw and roasted peanut cotyledons. (A) - raw, (B) ~ (E) - roasted peanuts: (B) 140 ^{o}C, 20 min, (C) 150 ^{o}C, 20 min, (D) 160 ^{o}C, 15 min, and (E) 160 ^{o}C, 20 min.

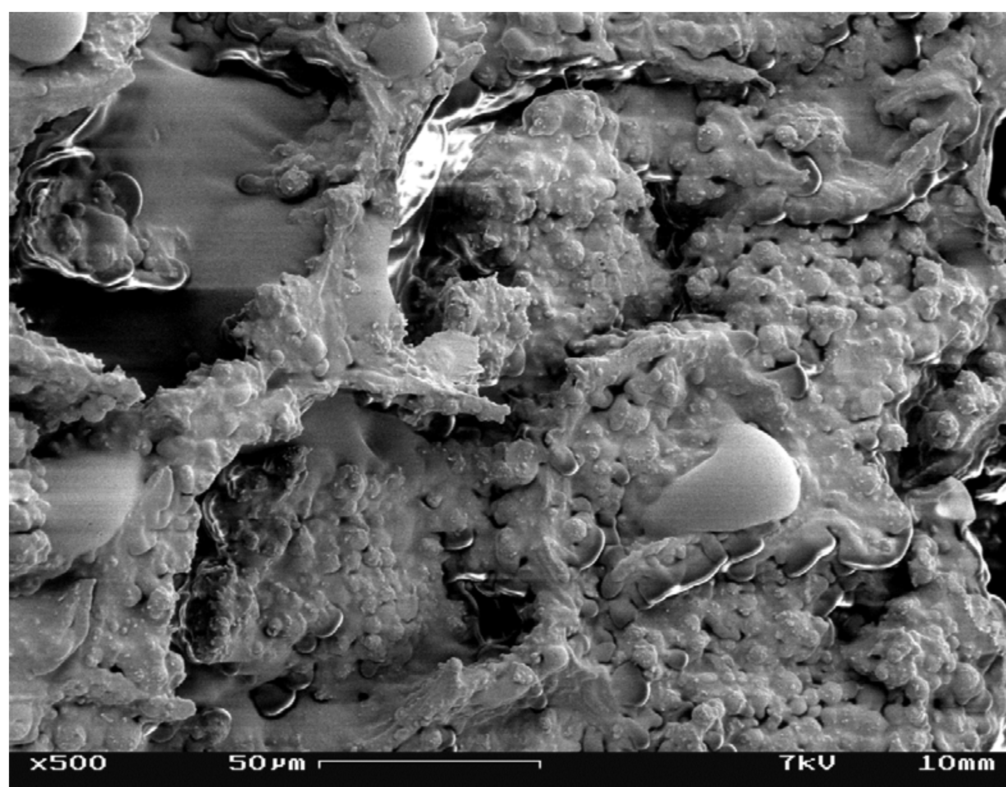
(A) Raw



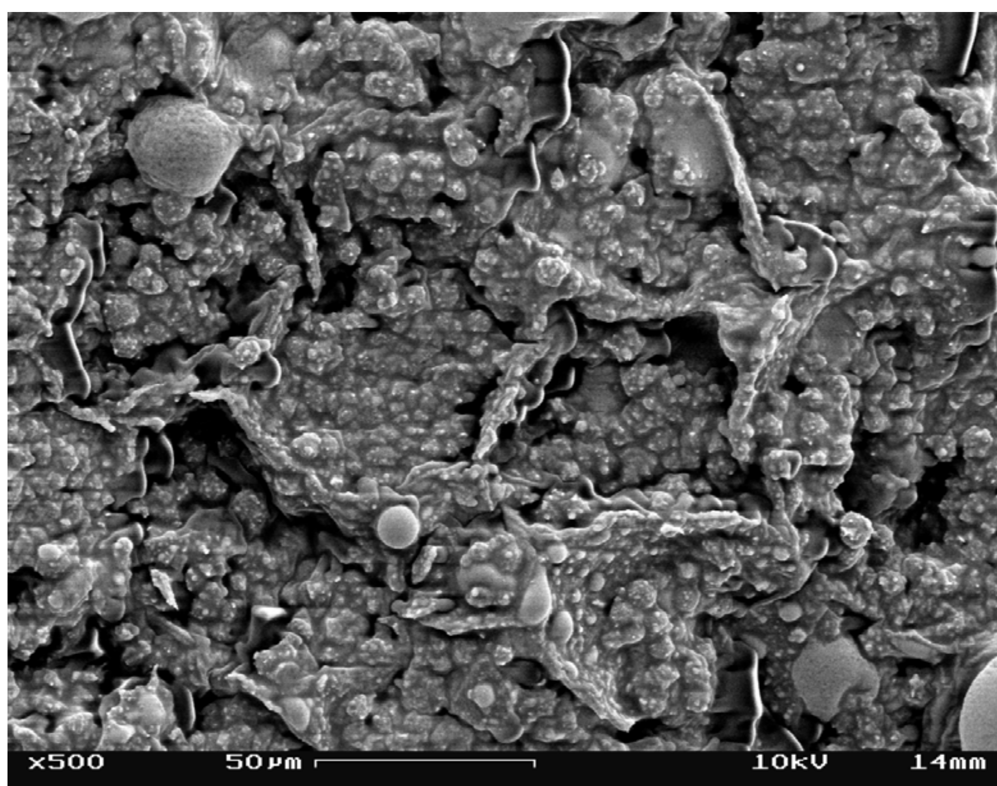
(B) 140ºC-20 min



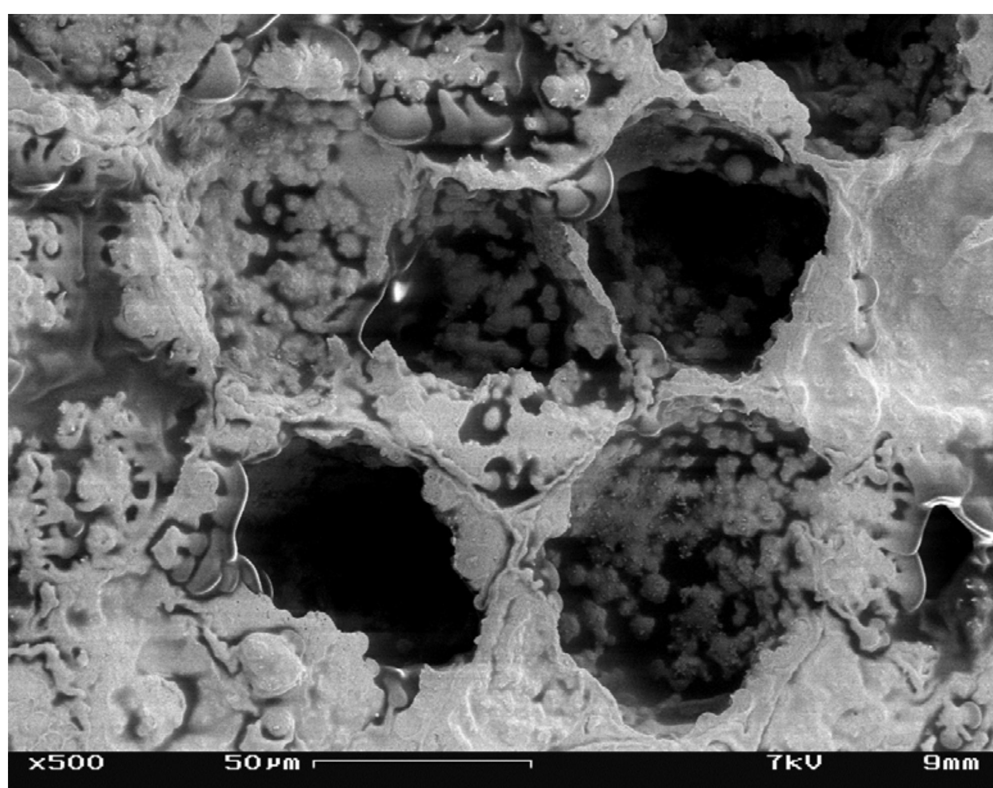
(C) 150ºC-20 min



(D) 160ºC-15 min



(E) 160ºC-20 min



roasting, vitamin E loss would be increased during storage due to damage in microstructure by roasting.

On the other hand, higher oil extraction yield of the pressing method noted for roasted peanuts compared to raw peanuts is due to these physical changes. However, the increase may be from the portion of oil reserve bodies and not bound tocopherols. Roasting itself may either decrease vitamin E levels or increase some portions of vitamin E levels by releasing bound tocopherols through breaking their bonds to other components in peanuts.

CONCLUSIONS

Oven roasting at 140, 150, and 160 ºC up to 20 min decreased moisture content of peanuts and developed the formation of a yellowish brown color, a characteristic of roasted peanuts. The rate of moisture removal and color formation were greater at higher temperature. Moisture ratio (M/Mo) and color b* value of peanuts roasted at 140 to160 ºC showed a correlation of $b^* = 21.61\,(M/Mo)^2 - 40.62\,(M/Mo) + 34.12$ ($R^2 = 0.9123$). Peanuts showed high vitamin E stability to roasting. During roasting, the level of measurable tocopherols in peanuts significantly increased at 140 ºC, remained constant at 150 ºC and decreased at 160 ºC ($p < 0.05$). Tocopherol contents of pressed peanut oils significantly decreased at all roasting temperature ($p < 0.05$). After roasting peanuts at 160 ºC for 20 min, about 93 and 84% of initial α-T was retained in roasted peanuts and peanut oils, respectively. γ-T was the least stable to roasting. Degree of tocopherol loss was higher when measured in peanut oils obtained by pressing than whole peanuts. Microstructure was severely destroyed by roasting. Changes in microstructure of peanuts noted by scanning electron microscopy during roasting would contribute much to their susceptibility to oxidation and vitamin E stability during further storage.

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CHAPTER V

VITAMIN E AND OXIDATIVE STABILITY DURING STORAGE OF RAW AND DRY ROASTED PEANUTS

INTRODUCTION

Tree nuts and peanuts are becoming established as significant dietary components contributing to lower risk of coronary heart disease (CHD) (Fraser et al., 1992, 1995; Sabatè and Fraser 1993, 1994; Hu et al., 1998, 1999, 2001; Kris-Etherton, 2001; Ellsworth et al., 2001; Albert and Willet, 2002). Clinical studies showed that high mono- and polyunsaturated fatty acid (MUFA and PUFA) diets supplemented with nuts including peanuts decreased the total and low density lipoprotein (LDL) cholesterol levels which are risk factors for CHD levels (Spiller et al., 1998; Zambón et al., 2000; Curb et al., 2000; Erario et al., 2001; Rajaram et al., 2001; Hyson et al., 2002). Kris-Etherton et al. (2001) suggested that effects of nuts on CHD risk may be through multiple mechanisms due to the variety of nutrients and bioactive substances present in nuts. Folate, vitamin B-6, vitamin E, selenium, sterols, fiber and other phytochemicals as well as MUFA and PUFA in peanuts are considered as possible components to have protective effects on health (Sabate and Hook, 1996; Wolk et al., 1999; Kris-Etherton, 1999).

Peanuts are an important oilseed crop throughout the world. Peanuts contain about 50% oil composed of 81% of unsaturated fatty acids of which about 39% is polyunsaturated (USDA, Nutrient Database, 2002) Peanut lipids with high percentages of unsaturated fatty acids are desirable in lowering the risk of CHD, but make peanuts susceptible to lipid oxidation which is the most common cause of quality loss during storage. The quality loss of lipids due to oxidation is of economic and nutritional importance to the peanut industry because of off-flavor development and the loss of nutrients such as essential fatty acids, amino acids, and lipid-soluble vitamins.

Peanuts are an excellent source of vitamin E. As an antioxidant and an important nutrient, vitamin E stability of peanuts is important to both the consumer and peanut industry. Tocopherols in peanuts are influenced by cultivar, genotype, growing conditions, origins, year, regions, and maturity stages (Bauernfeind, 1980; Sanders et al.,

1992; Hashim et al., 1993a, 1993b). After harvesting, levels in raw peanuts can be influenced by processing, storage and marketing. Storage studies on several tree nuts including almond, macadamia, pecan, walnut and cashew indicated that vitamin E content decreased as storage time increased, possibly as a result of its antioxidant function during lipid oxidation. The decrease is highly affected by storage temperature, the packaging material and availability of oxygen (Fourie and Basson, 1989; Yao et al., 1992; Erickson et al., 1994; Senesi et al., 1991, 1996; Lavedrine et al., 1997; Lima et al., 1998). Storage studies on raw and roasted peanuts usually indicated that roasted peanuts are less stable to oxidation than raw peanuts (St. Angelo and Ory, 1972, 1975; St. Angelo et al., 1977, 1979). Therefore, one would surmise that vitamin E is less stable in roasted peanuts compared to raw peanuts during storage, although little literature is available on the vitamin E stability during storage related to oxidative stability in raw and roasted peanuts.

The purpose of this study was to clearly show the changes in vitamin E content during storage of raw and roasted peanuts as affected by oxygen and to relate these changes to oxidative stability of peanuts.

MATERIALS AND METHODS

Sample Preparation

Runner-type raw and roasted peanuts from the 2000 crop year were obtained immediately after dry roasting at Tara Foods, Albany, GA. Raw peanuts were commercially dry roasted at 275 ºF in 1st zone and at 375 ºF in 2nd zone in a Proctor-Schwartz gas-fired roaster. Total roasting time was 20 min including each roasting time in the 1st and 2nd zones. After roasting, peanuts were cooled down to below 100 ºF. Roasting conditions were adjusted by Tara Foods to achieve Hunter color L value of 49 ±

1 (medium roasting). After blanching, peanuts were directly obtained from the processing line and transported to the University of Georgia, Ga for packaging on the same day.

Storage Conditions

Raw and roasted peanuts were each divided into two groups and then several sub-bags (150g) from each were packed under air and vacuum in heat sealable metallized polyester barrier film bags (KAPAK Co., Mineapolis, MN). The packaged samples were stored at 21 ± 1°C for 38 wks. Packaging and storage were carried out on the same day of roasting. Sampling was done on days 2, 4, and 7 for only the first week of storage and then the sampling intervals were extended. All samples were assayed in triplicate.

Vitamin E Extraction

To determine tocopherols in peanuts, direct solvent extraction and HPLC was used (Lee et al., 1998). Stored peanut kernels (50g) from each of three sub-samples were ground in a coffee mill for 10-20 s and 1.0 – 1.5 g of ground sample was weighed into a 125 mL round bottom glass bottle. Four millilitres of hot ionized water (80ºC) were added to the sample and then mixed with a spatula. Ten millilitres of isopropanol were added to the mixture and then thoroughly mixed. Approximately, 5 g of anhydrous magnesium sulphate were added followed by 25 mL of extraction solvent (hexane:ethylacetate, 90:10, v/v) containing 0.01% butylated hydroxytoluene (w/v). The mixture was homogenized with a Polytron ^{R} homogenizer for 90 s at medium speed. The homogenized mixture was filtered through a medium porosity glass filter in a vacuum bell jar filtration apparatus (Kontes, Vineland, NJ, USA). The filter cake was transferred to the same extracting bottle and then 5 mL of isopropanol and 30 mL of extracting solvent were added. Homogenization and filtration were repeated. The combined filtrate was transferred to a 100 mL volumetric flask and adjusted to 100 mL with extracting solvent. After mixing, a 1.0 mL aliquot of the combined filtrate was taken into a 10 mL

test tube and then evaporated under nitrogen gas. The remaining analytes in the test tube were redissolved with 2 mL of *n*-hexane and injected onto the HPLC column after filtration using 0.45 µm nylon membrane filter (MSI Inc., Westboro, MA).

HPLC

The normal phase HPLC system consisted of a LC-6A pump, equipped with a Shimadzu RF-10A spectrofluorometric detector (Shimadzu Corp., Columbia, Md., U.S.A.), a Spectra Series AS 100 autosampler (Thermo Separation Products Inc., San Jose, Calif., U.S.A.), and a 25 cm× 4 mm, 5 µm Li-Chrosorb Si60 column (Hibar Fertigsaule RT. Darmstadt, F.R. Germany) equipped with a precolumn packed with Perisorb A 30-40 µm (Darmstadt, F.R. Germany). The isocratic mobile phase contained 0.8% isopropanol in *n*-hexane (HPLC grade, J.T. Baker Chemical Co., Phillipsburg, N.J.). Prior to use, the mobile phase was filtered through a 0.22 µm nylon membrane filter (MSI Inc., Westboro, MA) and de-gassed by stirring under vacuum. The flow rate was 1.0 mL/min. For the determination of tocopherols, the excitation and emission wavelengths were set at 290 and 330 nm, respectively. The tocopherols in samples were identified by comparison of retention times with standard tocopherols. Tocopherol standards (α-, β-, γ- and δ-tocopherols) were obtained from Sigma (St. Louis, MO). Tocotrienols were not found in peanuts. Concentrations of tocopherols were calculated from the chromatographic peak area determined by the Waters 764 integrator (Millipore Corp., Cary, N.C., U.S.A.).

Chemical Measurement

Oil samples were prepared by hydraulic pressing of the stored peanut samples in a Carver press (Carver, Inc., Wabash, IN, USA). About 50 g of peanut samples were placed in the cylinder of the Carver press and pressed to obtain the oil under pressure of 70-100

kg/cm^2 for 30 min. Peroxide value of the oil was determined by acetic acid-chloroform method (Cd 8-53, AOCS 1997). For conjugated diene (CD) determination, a spectrophothometric method using isooctane was used (Erickson, 1994; AOCS 1997, Ti 1a-64). Accurately, 0.9-1.2 g of the pressed oil sample was weighed into a 100 mL volumetric flask and brought to volume with isooctane. The oil sample was completely dissolved by rotating the flask. After allowing the diluted oil to adjust to room temperature, the solution was diluted with isooctane to give a final concentration of about 0.01 g of the sample/L. The absorbance of the solution was determined at 233 nm against an isooctane blank. Concentration of conjugated dienes was calculated by the following equation:

$$CD\% = 0.84[(A/bc)-0.07]$$

where A = absorbance at 233 nm

b = cell length (1cm)

c = concentration of sample (g lipid/L) in final dilution

Oxidative stability of raw and roasted peanuts during storage under air and vacuum packaging was monitored by PV (meq/kg oil) and CD%. Since the limiting PV critical for acceptability for roasted peanuts or peanut oil are 20-30 meq/kg and edible quality of roasted peanuts is lost at PV equal or greater than 42-47 meq/kg oil (St. Angelo et al., 1977; Balasubramanyam et al., 1983; Narasimhan et al., 1986; Evranuz, 1993), sampling was stopped when PV values approximately 47 meq/kg oil. All assays were done in triplicate.

Statistics

Data was analyzed using a Statistical Analysis System package based on SAS System for Linear Models (SAS Institute Inc., 1986). Statistical significance of variables and possible relationships between parameters such as tocopherols and oxidation

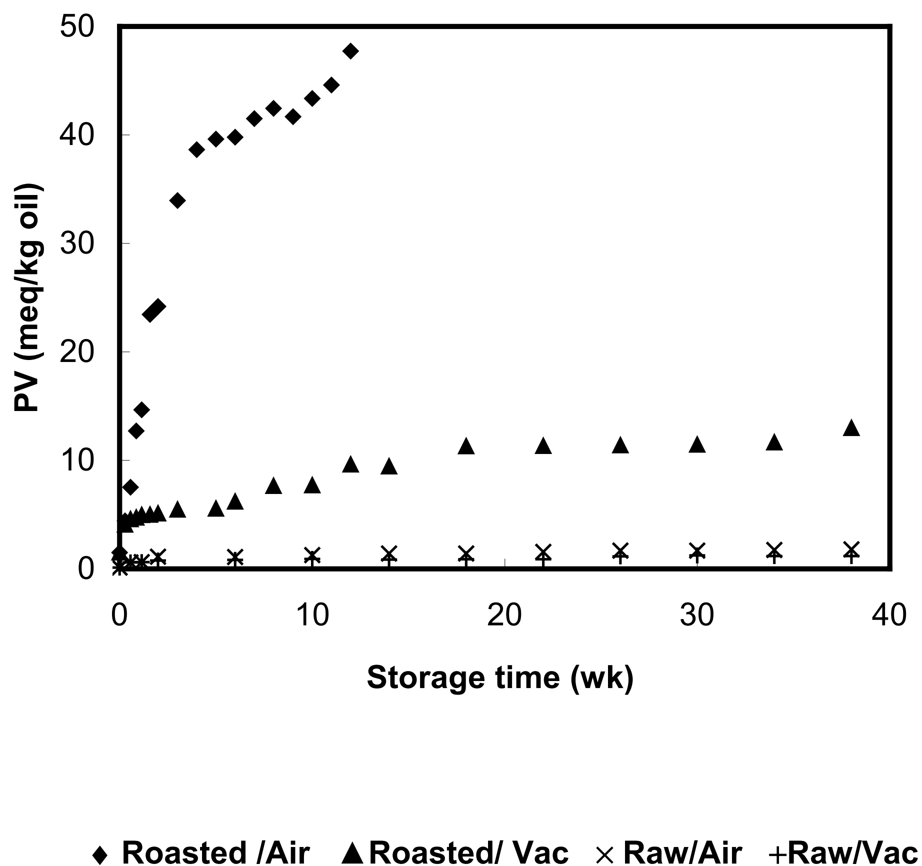
parameters (PV, CDPH) were analyzed using the General Linear Models (GLC) for Multiple Regression.

RESULTS AND DISCUSSION

PV

Changes in PV for raw and roasted peanuts during storage at 21 ºC under air and vacuum are shown in Figure 4-1. The initial PV at 0 day of storage were 0.11 and 1.49 for raw and roasted peanuts, respectively. The rate of PV increase for roasted peanuts was much faster than that of raw peanuts under both air and vacuum, indicating low oxidative stability of roasted peanuts ($p < 0.05$). In the presence of air, PV for roasted peanuts rapidly increased and reached 47 meq/kg by 12 weeks of storage while that for raw peanuts was still below 2 meq/kg until the end of storage (38 weeks). Under vacuum, lipid oxidation was significantly retarded for roasted peanuts ($p < 0.05$). PV for roasted peanuts after 38 weeks of storage under vacuum was about 13 meq/kg oil which was similar to PV values of roasted peanuts at one week of storage under air. The effect of vacuum packaging on lipid oxidation was also statistically significant for raw peanuts ($p < 0.05$) although there was no noticeable difference in PV for stored raw peanuts stored under air and vacuum (Figure 4-1).

Figure 4-1. Changes in peroxide value of raw and roasted peanuts during storage at 21ºC under air and vacuum.



CDV and PV

Changes in conjugated diene concentration for stored raw and roasted peanuts are shown in Figure 4-2. The conjugated diene values (CDV) for all stored peanut samples were strongly correlated with PV. During storage at 21°C a linear relationship existed between PV and CDV: PV = 115.4CDV - 20.457 (R^2 = 0.9809) (Figure 4-3). This strong

linear correlation between PV and CDV was observed in peanut butter (St. Angelo et al., 1975) and fish oil (Kulås and Ackman, 2001) with $R^2 \geq 0.98$.

Tocopherols

Tocopherols in raw and roasted peanuts at 0 day of storage were 10.5 and 10.0 for α-T, 0.5 and 0.5 for β-T, 9.0 and 8.4 for γ-T, and 1.1 and 1.1 for δ-T in mg/100g, respectively. Changes in tocopherol levels for raw and roasted peanuts during storage at 21ºC under air and vacuum were shown in Figure 4-4 and 4-5. Tocopherols of raw peanuts gradually decreased over the storage time, resulting in about 24 and 20% total tocopherol losses during 38 weeks of storage under air and vacuum. In the presence of air, tocopherols of roasted peanuts, especially α- and γ-T, dramatically decreased during initial four weeks of storage, causing about 84 and 68% losses for α- and γ-T, respectively, and then during further storage up to 12 weeks under air, gradually decreased, resulting in 90 and 70% total losses for α- and γ-T, respectively (Figure 4-5A). The rate of α-T loss was faster than that of γ-T. The same order (α-T > γ-T) was found in roasted peanuts (Inoue et al., 1988), partially defatted peanuts (Adnan et al., 1981) and rapeseed (Goffman and Möllers, 2000). Adnan et al. (1981) reported about 95% of α-T and 27% of γ-T losses in reconstituted partially defatted peanuts during storage. Oscillating changes in tocopherols levels were reported in raw pecans and roasted cashew during storage (Yao et al., 1992; Erickson et al., 1994; Lima et al., 1998).

Figure 4-2. Changes in conjugated diene concentration of raw and roasted peanuts during storage at 21ºC under air and vacuum.

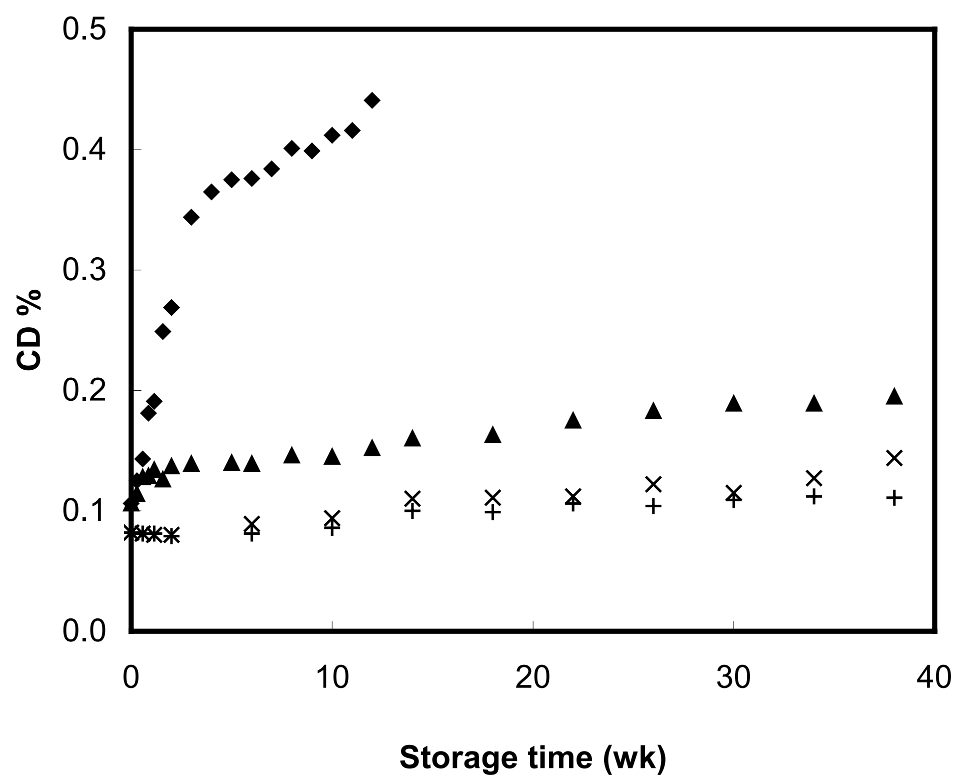
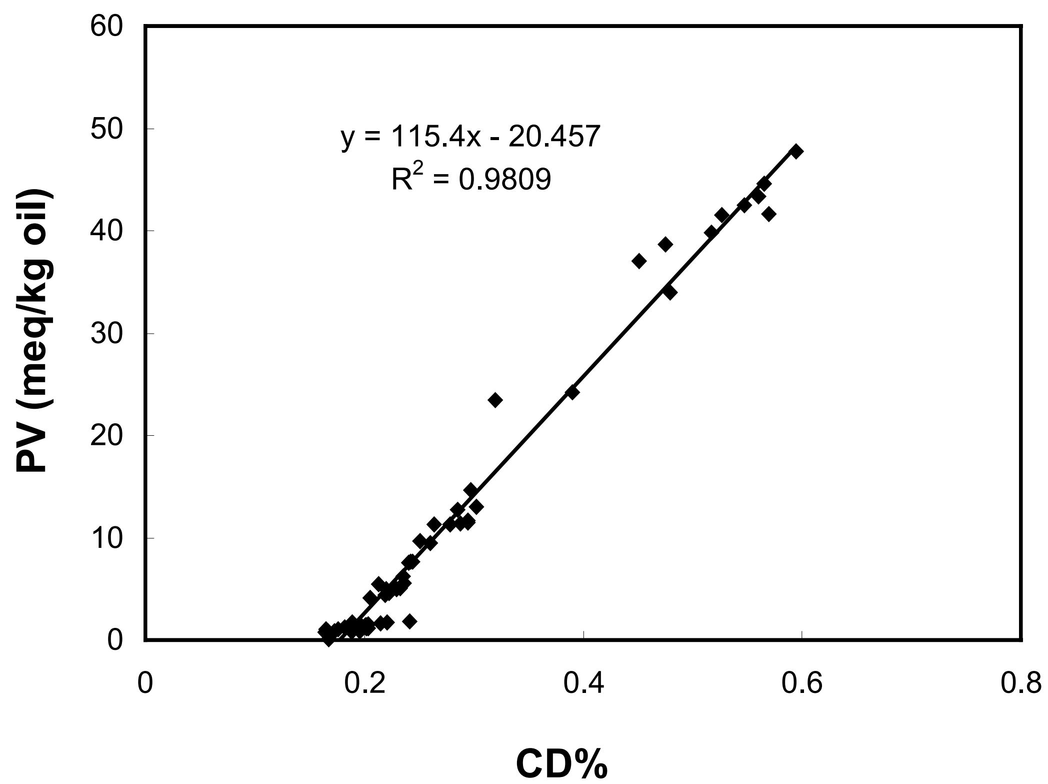

0.5
0.4
0.3
0.2
0.1
0.0
CD %
0
10
20
30
40
Storage time (wk)
Roasted /Air
Roasted/ Vac
Raw/Air
Raw/Vac

Figure 4-3. The relationship between PV and CDV for raw and roasted peanuts during storage at 21°C under air and vacuum.

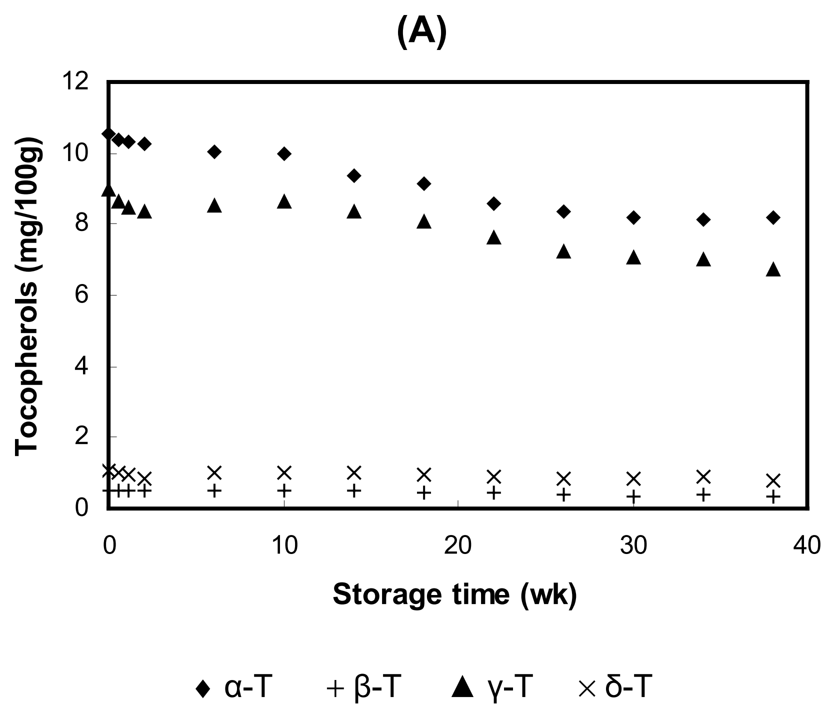

y = 115.4x - 20.457
R2 = 0.9809
PV (meq/kg oil)
CD%
60
50
40
30
20
10
0
0
0.2
0.4
0.6
0.8

Vacuum packaging significantly decreased tocopherol losses for both raw and roasted peanuts over storage time ($p < 0.05$) (Figure 4-4B and 4-5B). Table 1 shows retention of all tocopherols in raw and roasted peanuts during storage at 21 ºC. After 12 weeks of storage, more than 50% of the initial α-T level remained for roasted peanuts stored under vacuum while about 10% α-T was retained under air. Further storage under vacuum extended α-T loss of roasted peanuts, resulting in 74% loss of α-T at the end of storage. The other tocopherols in roasted peanuts, however, remained at more than 50% of initial levels after 38 weeks of storage. For raw peanuts, although remaining tocopherols were slightly higher under vacuum than under air, more than 70% of all tocopherols were retained after 38 weeks of storage under both air and vacuum (Table 4-1). Sensi et al., (1991) reported that during 18 month of storage at 20 ºC, 94% loss of the initial α-T (35.4 mg/100g of oil) occurred in almonds packaged in metallized film under vacuum while 80% loss occurred under nitrogen. Under vacuum, no significant vitamin E loss was noted inshelled, roasted, and salted cashew nuts during storage at 30°C for 1 year in flexible packaging materials with a low water vapor permeability rate (Lima et al., 1998).

Tocopherols and Lipid Oxidation

Tocopherol losses were highly correlated with lipid oxidation based on PV and CDV for all stored peanuts, indicating antioxidant function of vitamin E during lipid oxidation. As PV increased, the level of α-T in stored peanuts decreased exponentially with a high correlation coefficient of 0.9246 ($\alpha\text{-T} = 8.8956\ e^{-0.0468\ PV}$) (Figure 4-6). The level of total tocopherols in stored peanuts also showed a high correlation with PV ($\text{Total-T} = 18.339\ e^{-0.0336\ PV}$, $R^2 = 0.9374$). Evranuz (1993) reported shelf lives of salted roasted peanuts of 28 days at 15°C, 10 days at 25°C, and 11 days at 35°C under the assumption that the products remain acceptable until the PV reaches 25 meq/kg oil.

Figure 4-4. Changes in tocopherol contents of raw peanuts during storage under air (A) and vacuum (B).


(A)
Tocopherols (mg/100g)
Storage time (wk)
0
2
4
6
8
10
12
0
10
20
30
40
α-T
β-T
γ-T
δ-T

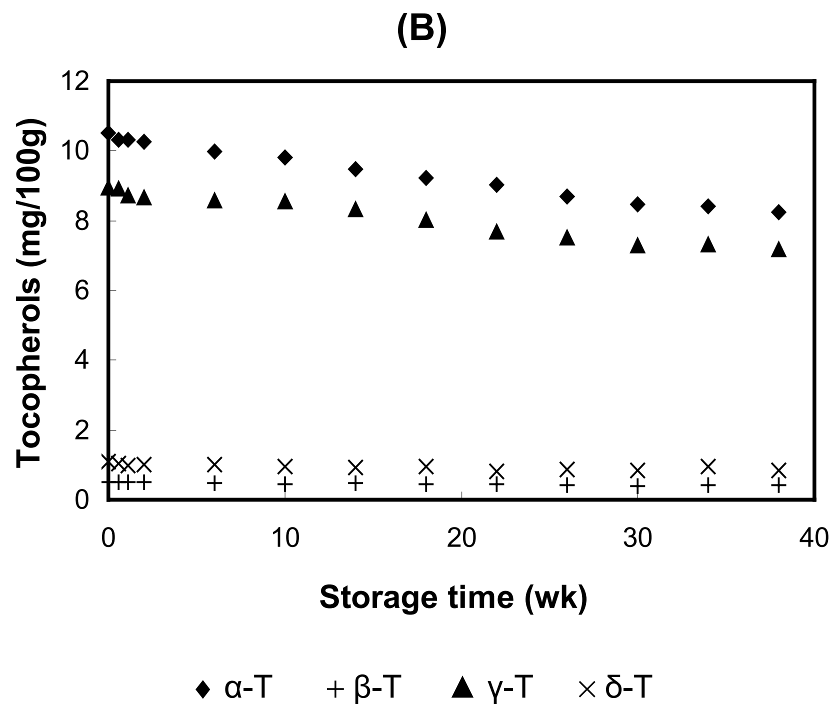
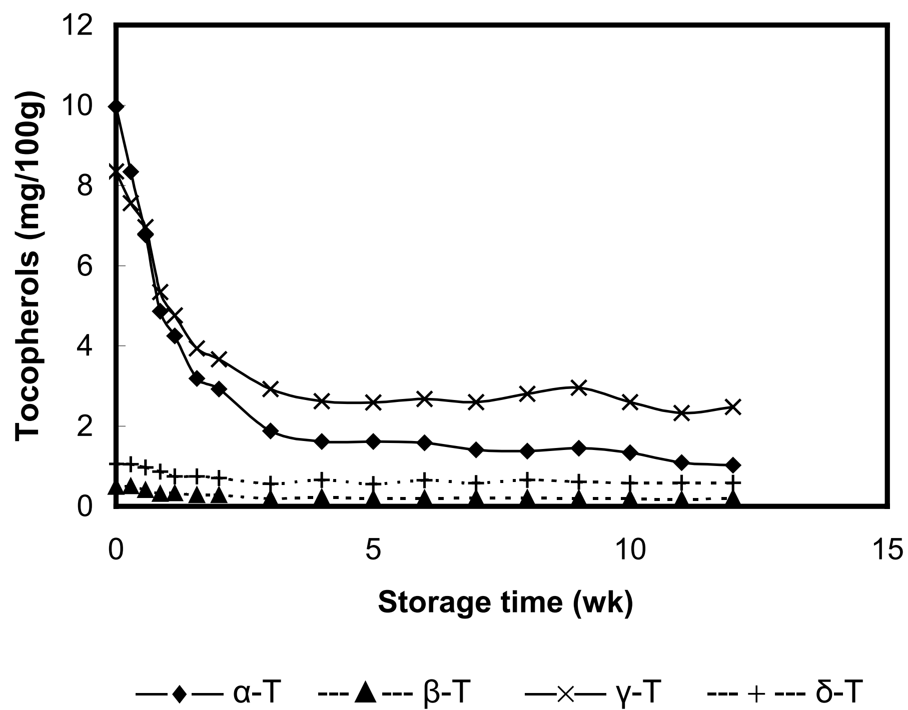

(B)
Tocopherols (mg/100g)
Storage time (wk)
0
2
4
6
8
10
12
0
10
20
30
40
α-T
β-T
γ-T
δ-T

Figure 4-5. Changes in tocopherol contents of roasted peanuts during storage under air (A) and vacuum (B).

(A)



(B)

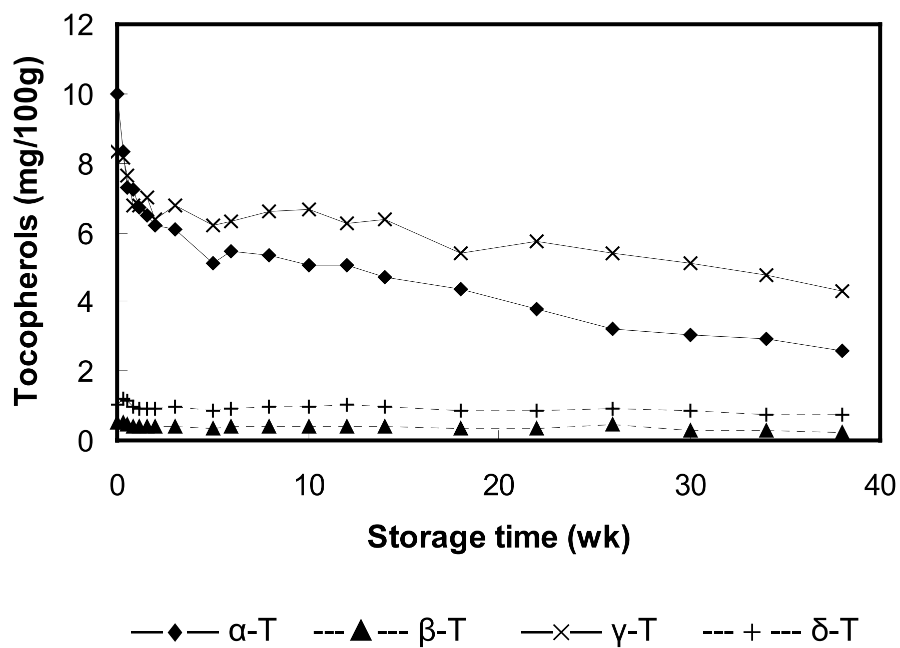


Table 4-1. Retention of Vitamin E in Raw and Roasted Peanuts during Storage under Air and Vacuum at 21°C

Samples	Packaging	Tocopherols	Retention of Tocopherols (%) [a]						
			------------------------ Storage time (wks) ------------------------						
			0	2	6	12	22	30	38
Raw	Air	α-T	100	97.6	95.5	91.9	81.7	77.9	77.9
		β-T	100	98.0	98.0	90.0	84.0	77.0	70.0
		γ-T	100	95.4	95.4	94.9	85.4	79.0	75.4
		δ-T	100	95.3	95.4	91.7	83.3	78.7	70.4
	Vacuum	α-T	100	97.5	95.0	91.7	85.8	80.5	78.2
		β-T	100	100	98.0	98.0	92.0	86.0	82.0
		γ-T	100	96.7	96.0	94.4	85.8	81.8	80.3
		δ-T	100	93.5	92.6	88.0	87.0	78.7	76.9
Roasted	Air	α-T	100	29.5	16.0	10.3	-	-	-
		β-T	100	57.1	40.8	42.9	-	-	-
		γ-T	100	44.0	32.1	29.7	-	-	-
		δ-T	100	67.0	61.3	55.7	-	-	-
	Vacuum	α-T	100	62.2	55.0	50.5	38.2	30.3	26.0
		β-T	100	81.6	79.6	85.7	73.5	59.2	51.0
		γ-T	100	76.3	75.8	74.6	68.5	61.3	51.5
		δ-T	100	84.9	85.9	95.3	86.8	83.0	70.8

[a] Mean of three determinations. Data is not available for roasted peanuts after 12 weeks due to no sampling.

Figure 4-6. The relationship between α-tocopherol and PV for raw and roasted peanuts during storage at 21ºC under air and vacuum.

$y = 8.8956e^{-0.0468x}$
$R^2 = 0.9246$

Alpha tocopherol (mg/100 g)

PV (meq/kg oil)

Applying the same assumption to this study, shelf life of dry roasted peanuts is about 2 weeks when stored at 21 °C under air. At this point, about 30% of α-T remained.

CONCLUSIONS

Vitamin E stability of raw and roasted peanuts during storage at 21 °C was closely correlated with lipid oxidation based on PV and CDV. Tocopherols of peanuts exponentially decreased with increasing PV during storage. Roasting significantly decreased oxidative stability of peanuts, and consequently, lowered vitamin E retention ($p < 0.05$). Vacuum packaging was effective in maintaining the quality of dry roasted peanuts based on oxidative changes and retention of vitamin E.

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CHAPTER VI

SUMMARY AND CONCLUSIONS

SUMMARY AND CONCLUSIONS

The purpose of this research was to investigate vitamin E stability of peanuts during processing and storage and to provide more complete data on the vitamin E content of peanuts and peanut products.

In the first experiment, vitamin E contents of peanuts and peanut products were determined by using a direct solvent extraction method and HPLC. The mean α-T values (mg/100 g) of commercial products were 8.2 for raw peanuts, 4.1 for roasted peanuts, 9.4 for peanut butter, 5.5 for reduced fat peanut butter and 12.3 for peanut oils. Precision and accuracy of the analytical method were very good. The mean recoveries were over 95% for all tocopherols. The effect of peanut butter manufacture on vitamin E content of peanuts of two crop years (runner-type) was studied. No significant difference was observed in any other tocopherols values between 1998 and 1999 crop raw peanuts ($p > 0.05$). Mean α- and γ-T values of raw peanuts were 11.0 and 10.3 in mg/100g. Differences in all tocopherol levels (mg/100g edible portion) except for γ-T between raw peanuts and peanut butter manufactured from the raw peanuts were not significant ($p > 0.05$). Contribution of the oil and stabilizer added to the roasted peanuts during peanut butter manufacture was calculated to be 4% of α- and 5% of γ-T in peanut butter. Tocopherol losses in peanuts by commercial peanut butter manufacture were below 6% for all tocopherols, indicating high stability of vitamin E in peanuts to roasting which is conducted at high temperature and milling.

In the second experiment, the effect of roasting on vitamin E contents of peanuts and peanut oils prepared by pressing the roasted peanuts were studied. Changes in color and microstructure of peanuts were also investigated by using a CIE LAB system and scanning electron microscopy (SEM). Oven roasting at 140, 150, and 160 ºC for 20 minrapidly decreased moisture content of peanuts and developed the formation of a golden brown color of roasted peanuts. The rate of moisture removal and color formation were greater at higher temperature. Moisture ratio (M/Mo) and color b* value of roasted

peanuts showed a correlation of $b^* = 21.61 (M/Mo)^2 - 40.62 (M/Mo) + 34.12$ ($R^2 = 0.9123$). The tocopherol contents of raw peanuts were 10.9, 0.5, 10.8 and 19.2 mg/ 100g (dry basis) for α-, β-, γ- and δ-T, respectively. Based on Duncan's test, the tocopherol levels of peanuts slightly increased at 140 ºC, were constant at 150 ºC, and decreased at 160 ºC during roasting. At 160 ºC, tocopherol losses mainly occurred during initial 10 min of roasting with extensive loss occurring after 10 min, resulting in about 5, 12, 20, and 10% losses of initial levels of α-, β-, γ- and δ-T, respectively. Tocopherol contents of peanut oils prepared by pressing the roasted peanuts significantly decreased at all roasting temperatures with roasting time ($p < 0.05$). After roasting peanuts at 160 ºC for 20 min, about 93 and 84% of initial α-T in peanuts and peanut oils were retained, respectively. α-T was the most stable to roasting while γ-T was the least. Microstructure observed by SEM was severely destroyed with increasing roasting temperature. Swollen epidermal cells on the surface and rupture of parenchyma tissue of peanut cotyledon were observed for peanuts roasted for 20 min at all temperature, which would contribute much to their susceptibility to oxidation and decreased vitamin E stability during further storage.

In the third experiment, vitamin E stability of raw and roasted peanuts during storage at 21ºC under air and vacuum was investigated and related to oxidative stability of the peanuts. Lipid oxidation rapidly progressed in roasted peanuts compared to raw peanuts based on PV and CDV. In the presence of air, PV for roasted peanuts was rapidly increased and reached 47 meq/kg by 12 weeks of storage while the PV for raw peanuts was still below 2 meq/kg until the end of storage (38 weeks). Under vacuum, lipid oxidation was significantly retarded for raw and roasted peanuts ($p < 0.05$). CDV for stored peanut samples were directly proportional to PV ($PV=115.4CDV-20.457$, $R^2=0.98$). α-T levels of raw and roasted peanuts exponentially decreased with increasing PV during storage ($\alpha\text{-T}=8.8956e^{-0.0468PV}$, $R^2 = 0.9246$). For raw peanuts, more than 70% of all tocopherols were retained after 38 weeks of storage under both air and vacuum. Under air, α- and γ-T of roasted peanuts dramatically decreased during the initial four weeks of storage, resulting in about 84 and 68% losses for α- and γ-T, respectively.

Vitamin E gradually decreased during storage up to 12 weeks, resulting in 90 and 70% total losses for α- and γ-T, respectively. α-T showed the least stability during storage. Vacuum storage greatly decreased the rate of oxidation in roasted peanuts. After 12 weeks of storage, more than 50% of the initial α-T level remained for roasted and stored under vacuum compared to about 10% for those stored under air.

In conclusion, peanuts and peanut products are good sources of vitamin E. Vitamin E stability of peanuts is high during roasting and milling, resulting in more 90% of retention for α-T. Roasting significantly decreases oxidative stability of peanuts. Since vitamin E stability is closely related with oxidative stability of peanuts during storage, vacuum packaging to decrease the rate of lipid oxidation is an effective way to retain high vitamin E content, especially α-T.