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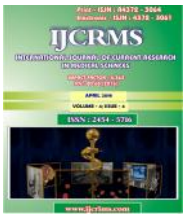

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A review on palm oil supplemented diet and enzymatic antioxidants in aging

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Abstract

The importance of genetics in the regulation of biologic aging is shown by the characteristic longevity of each animal species. Deoxyribonucleic acid (DNA) undergoes continuous changes in response to exogenous agents and intrinsic processes. Stability is maintained by the double-strandedness of DNA and by specific repair enzymes. Somatic mutagenesis, due to either a greater susceptibility to mutagenesis or deficits in repair mechanisms, is a factor in biologic aging. An antioxidant is a substance that when present in low concentrations relative to the oxidizable substrate significantly delays or reduces oxidation of the substrate. The body has developed several endogenous antioxidant systems to deal with the production of Reactive Oxygen Intermediate (ROI). These systems can be divided into enzymatic and non-enzymatic groups. Palm oil consists mainly of glycerides made up of a range of fatty acids. Triglycerides constitute the major component, with small proportions of triglycerides and monoglycerides. Palm oil also contains other minor constituents, such as free fatty acids and non-glyceride components. Crude palm oil is considered to be the richest natural source of carotenoids. Carotenoids also play an important potential role by acting as biological antioxidants, protecting cells and tissues from the damaging effect of free radicals. Feeding experiments using various animal models have highlighted that red palm oil is beneficial to health by reducing oxidative stress. Because of high level of antioxidants content of palm oil, it can enhance longevity of cell as well as the whole body.

Keywords: Palm oil, antioxidants, aging

Introduction

The biologic basis of aging

The importance of genetics in the regulation of biologic aging is shown by the characteristic longevity of each animal species. Several theories of aging have been promulgated. These theories, which have been extensively reviewed (Goldstein, 1971; Goldstein et at., 1983; Goldstein, 1989), fall into two general categories: the accumulation of damage to informational molecule, and the regulation of specific genes.

Deoxyribonucleic acid (DNA) undergoes continuous changes in response to exogenous agents and intrinsic processes. Stability is maintained by the double-strandedness of DNA and by specific repair enzymes. Somatic mutagenesis, due to either a greater susceptibility to mutagenesis or deficits in repair mechanisms, is a factor in biologic aging. In fact, there is a positive correlation of species longevity with repair enzymes. In humans, the spontaneous mutagenesis rate is not adequate for the number of changes that would be necessary to cause aging and there is no evidence that a failure in repair systems underlies this phenomenon (Abrass, 1990).

A related theory, the error-catastrophe theory, proposes that errors occur in DNA, RNA and protein synthesis, each augmenting the others and finally culminating in an error catastrophe. Translation was considered the most likely source of age dependent errors because it was the final common pathway. But increased translational error has been found during aging either in vivo or in vitro (Abrass, 1990).Amino acid substitutions do not increase with aging, although some enzymes activities may be altered by changes in posttranslational modification, such as glycosylation.

At present, the most favoured concept of aging is that it is regulated by specific genes. Support for such a hypothesis has been gained mostly from in vitro models of aging. Adult cells can be placed into three categories based on their replicative capacity: continuously replicating, replicating in response to a challenge and non-replicating. For example, epidermal, gastrointestinal and haemopoietic cells are continuously renewed; the liver can regenerate in response to injury; neurons, cardiac and skeletal muscle do not regenerate. In vitro replication is closely related to in vivo proliferation. Neurons and cardiac myocytes from adults can be maintained in culture but do not divide, whereas hepatocytes, marrow cells, endothelial cells and fibroblasts replicate in vitro. Because they are easily obtained from skin, fibroblasts have been the most extensively studied. Although some cells continuously replicate in vivo, they have a finite replicative life (Hayflick, 1976). For fibroblasts in vitro, this is about 50 times. The replicative life of fibroblasts in vitro correlates with the age of the donor - the older the donor, the fewer the doublings in vitro. With time in culture doubling time decreases and replication eventually stops.

When fibroblasts from younger donors are fused with non replicating senescent cells, DNA synthesis is inhibited in both nuclei. But when protein synthesis is transiently inhibited immediately after fusion, DNA synthesis is increased in both nuclei suggesting that a cytoplasmic protein factor may be involved in inhibiting replication. When senescent cytoplasts - cells without nuclei — are fused with young, dividing cells, DNA synthesis is depressed. Growth arrest both in vivo and in vitro has now been associated with the appearance of a specific protein that may be involved in DNA replication.

These experiments help us understand the finite life span of cells in vitro but do not explain the process of aging in vivo, since organisms do not suddenly die because all their cells stop replicating and die. Nevertheless, factors associated with finite cell replication may have a direct influence on the aging of an organism. For example, fibroblasts aged in vitro or obtained from older adult donors are less sensitive to many growth factors. Such changes may be mediated at both the receptor and post-receptor level. A decrease in the output of growth factors, changes in cell sensitivity to growth factors, or a slowing of the cell cycle may all contribute to impaired wound healing and thus place older persons at greater risk for infection.

For tissues with non-replicating cells, cell loss may lead to a permanent deficit. With aging, dopaminergic neurons are lost, and this loss influences gait, balance and the susceptibility to drug side effects. Similar cell loss, functional deficits or both may occur in other neurotransmitter systems and lead to autonomic dysfunction and to an alteration in mental function and neuro-endocrine control.

The immune system shows similar age-dependent phenomena. Lymphocytes from older adults have a diminished proliferative response to numerous mitogens. This appears to be due to both a decrease in lymphokines and a lessened response to extracellular signals. Basal and stimulatory interleukin-2 (IL-2) production and IL-2 responsiveness also decreases with age. Diminished IL-2 responsiveness appears to be attributed, at least in part, to a decreased expression of IL-2 receptors.In vivo molecular mechanisms such as those described above, contribute to physiologic deficits and altered homeostatic mechanisms that predispose older persons to dysfunction in the face of stress and disease (Finch, 1976; Finch and Schneider, 1985).

Antioxidant

An antioxidant is a substance that when present in low concentrations relative to the oxidizable substrate significantly delays or reduces oxidation of the substrate (Halliwell, 1995). Antioxidants get their name because they combat oxidation. They are substances that protect other chemicals of the body from damaging oxidation reactions by reacting with free radicals and other reactive oxygen species within the body, hence hindering the process of oxidation (Ofor *et al., 2016*; Nwosu *et al.*, 2016; Nwosu *et al.*, 2015).

Antioxidant system

The body has developed several endogenous antioxidant systems to deal with the production of Reactive Oxygen Intermediate (ROI). These systems can be divided into enzymatic and non-enzymatic groups.

The enzymatic antioxidants include superoxide dismutase (SOD), which catalyses the conversion of O^{2-} to H_2O_2 and H_2O_2; catalase, which then converts H_2O_2 to H_2O and O_2; and glutathione peroxidase, also reduces H_2O_2 to H_2O. The non-enzymatic antioxidants include the lipid-soluble vitamins, vitamin E and vitamin A or provitamin A, beta-carotene, and the water-soluble vitamin, vitamin C and Glutathione (GSH). Vitamin E has been described as the major chain- breaking antioxidant in humans (Packer, 1992). Because of its lipid solubility, vitamin E is located within cell membranes, where it interrupts lipid peroxidation and may play a role in modulating intracellular signaling pathways that rely on reactive oxygen intermediates (ROI) (Kagan et al. 1990; Azzietal. 1993).

The enzymatic and non-enzymatic antioxidant systems are intimately linked to one another and appear to interact with one another. For example, glutathione (GSH) - a nonenzymatic antioxidant in human tissues provides reducing equivalents for the glutathione peroxidase (GPx) catalyzed reduction of hydrogen peroxide and lipid hydroperoxides to water. In addition, the trace elements selenium, manganese, copper, and zinc also play important roles as nutritional antioxidant cofactors. Selenium is a cofactor for the enzyme glutathione peroxidase, and manganese, copper, and zinc are cofactors for SOD. Zinc also acts to stabilize the cellular metallothionein pool, which has direct free radical quenching ability (Bray and Bettger, 1990).

Mode of action of antioxidants

There are four routes:

1. Chain breaking reactions, e.g. alpha-tocopherol which acts in lipid phase to trap “ROS” radical.
2. Reducing the concentration of reactive oxygen species e.g. glutathione.
3. Scavenging initiating radicals e.g. superoxide dismutase which acts in aqueous phase to trap superoxide free radicals.
4. Chelating the transition metal catalysts: A group of compounds serves an antioxidant function by sequestration of transition metals that are well-established pro-oxidants. In this way, transferrin, lactoferrin, and ferritin function to keep iron induced oxidant stress in check and ceruloplasmin and albumin as copper sequestrants.

Non-enzymatic antiox1dai'its

Alpha tocopherol (Vitamin E)

Tocopherol is the major lipid soluble antioxidant found in cells. The term tocopherol was used because this compound permitted an animal to have offspring, tocopherol is from the Greek word tokos, meaning childbirth, and the verb phero, means to bring forth. To indicate the alcohol nature of the molecule, ol was added to the ending.

Vitamin E is a generic term that includes all entities that exhibit the biological activity of natural vitamin E, d-alpha-tocopherol. In nature, eight substances have been found to have vitamin E activity: d-alpha-, d-beta, d-gamma- and d-delta-tocopherol and d-alpha-, d-beta-, d-gamma- and d-delta-tocotrienol. Also, the acetate and succinate derivatives of the natural tocopherols have vitamin E activity, as do synthetic tocopherols and their acetate and succinate derivatives.

Of all these, d-alpha-tocopherol has the highest biopotency, and its activity is the standard against which all the others must be compared. It is the predominant isomer in plasma.

Vitamin E is an essential nutrient that functions as an antioxidant in the human body. It is essential, by definition, because the body cannot manufacture its own vitamin E and thus it must be provided by foods and supplements.

Deficiency of vitamin E does not produce a disease with rapidly developing symptoms such as scurvy or beriberi. Overt symptoms due to vitamin E deficiency occur only in cases involving fat malabsorption syndromes, premature infants and patients on total parenteral nutrition. The effects of inadequate vitamin E intake usually develop over a long time, typically decades, and have been linked to chronic diseases such as cancer and atherosclerosis.

Tocopherols are present in oils, nuts, seeds, wheat germ and grains. Absorption is believed to be associated with intestinal fat absorption.

Approximately 40% of the ingested tocopherol is absorbed. Most tocopherols enter the blood via lymph where they are associated with chylomicrons. Vitamin E is stored in adipose tissue. Phospholipids of the mitochondria and endoplasmic reticulum and plasma membranes possess affinities for alpha tocopherol and the vitamin tends to concentrate in these sites.

Mechanisms of action

Vitamin E is more appropriately described as an antioxidant than a vitamin. This is because, unlike most vitamins, it does not act as a co-factor for enzymatic reactions. Its main function is to prevent the peroxidation of membrane phospholipids, and avoids cell membrane damage through its antioxidant action. The lipophilic character of tocopherol enables it to locate in the interior of the cell membrane bilayers (Halliway and Getteridge, 1992; Borg, 1993). Tocopherol-OH can transfer a hydrogen atom with a single electron to a free radical, thus removing the radical before it can interact with cell membrane proteins or generate lipid peroxidation. When tocopherol-OH combines with the free radical, it becomes tocopherol-O, itself a radical. When ascorbic acid is available, tocopherol-O plus ascorbate (with its available hydrogen) yields semidehydroascorbate (a weak radical) plus tocopherol-OH (Halliway and Gutteridge, 1992). Bythis process, an aggressive ROI is eliminated and a weak ROI (debydroascorbate) is formed, and tocopherol-OH is regenerated. Despite this complex defence system, there are no known endogenous enzymatic antioxidant systems for the hydroxyl radical.

Beta carotene

Carotenoids are pigmented micronutrients present in fruits and vegetables. Carotenoids are precursors of vitamin A and have antioxidant effects. While over 600 carotenoids have been found in the food supply, the most common forms are alpha-carotene, beta-carotene, lycopene, crocetin, canthaxanthin, and flicoxanthin. Beta-carotene is the most widely studied. It is composed of two molecules of vitamin A (retinol) joined together. Dietary beta-carotene is

converted to retinol at the level of the intestinal mucosa.

Mechanisms of action

The antioxidant function of beta-carotene is due to its ability to quench singlet oxygen, scavenge free radicals and protect the cell membrane lipids from the harmful effects of oxidative degradation (Krinsky and Deneke, 1982). The quenching involves a physical reaction in which the energy of the excited oxygen is transferred to the carotenoid, forming an excited state molecule (Krinsky, 1993). Quenching of singlet oxygen is the basis for beta-carotene's well known therapeutic efficacy in erythropoietic protoporphyria (a photosensitivity disorder) (Matthews-Roth, 1993). The ability of beta-carotene and other carotenoids to quench excited oxygen, however, is limited, because the carotenoid itself can be oxidized during the process (autoxidation). In 1984, Burton and Ingold showed that beta-carotene autoxidation in vitro is dose-dependent and dependent upon oxygen concentrations. At higher concentrations, it may function as a pro-oxidant and can activate proteases.

In addition to singlet oxygen, carotenoids are also thought to quench other oxygen free radicals. It is also suggested that beta carotene might react directly with the peroxyl radical at low oxygen tensions; this may provide some synergism to vitamin E which reacts with peroxyl radicals at higher oxygen tensions (Cotgreave et al, 1988).

Ascorbic acid (Vitamin C)

Ascorbic acid (vitamin C) is a water-soluble, antioxidant present in citrus fruits, potatoes, tomatoes and green leafy vegetables. Humans are unable to synthesize 1-ascorbic acid from d-glucose due to absence of the enzyme L-gulacolactone oxidase. Hence, humans must therefore obtain ascorbic acid from dietary sources.

Mechanism of action

As an antioxidant, it scavenges free radicals and reactive oxygen molecules, which are produced during metabolic pathways of detoxification. It also prevents formation of carcinogens from precursor compounds (Block and Menkes, 1989). One important property is its ability to act as a reducing agent (electron donor), making it capable of reducing such compounds as a molecular oxygen, nitrate and cytochromes a and c. Donation of one electron by ascorbate gives the semidehydroascorbate radical (DHA). Ascorbate reacts rapidly with $O^{2-}$and even more rapidly with OH to give DHA. DHA, itself can act as a source of vitamin C.

Glutathione (GSH)

GSH is synthesized intracellularly from cysteine, glycine, and glutamate. In addition to its role as a substrate in GSH redox cycle, GSH is also a scavenger of hydroxyl radicals and singlet oxygen. It is capable of either directly scavenging ROI or enzymatically via glutathione peroxidase, as described later. In addition, GSH is crucial to the maintenance of enzymes and other cellular components in a reduced state. GSH also has an important role in xenobiotic metabolism and leukotriene synthesis. It is found in millimolar concentration in all human cells (Halliwell, 1994).

The majority of GSH is synthesized in the liver, and approximately 40% is secreted in the bile. The biologic role of GSH in bile is believed to be defence against dietary xenobiotics and lipid peroxidation in the lumen of the gut and protection of the intestinal epithelium from oxygen radical attack (Aw, 1994),

Mechanism of action

Reduced glutathione, (GSH, a tripeptide with a free thiol group, is a major antioxidant in human tissues that provides reducing equivalents for the glutathione peroxidase (GPx) catalyzed reduction of hydrogen peroxide and lipid hydroperoxides to water. During this process GSH becomes oxidized glutathione (GSSG). The GSSG is then recycled into GSH by glutathione reduetase (GR) and

reduced nicotinamide adenine dinucleotide phosphate (NADPH) as in the equation below.

$$H_2O_2 + 2GSH \xrightarrow{GP_X} GSSG + H_2O_2$$

$$GSSG + NADPH + H \xrightarrow{GP_X} 2GSH + NADP^+$$

Antioxidant enzymes

Superoxide dismutase (SOD)

SOD is an endogenously produced intracellular enzyme present in essentially every cell in the body. Cellular SOD is actually represented by a group of metalloenzymes with various prosthetic groups. The prevalent enzyme is cupro-zinc (CuZn) SOD, which is a stable dimeric protein (32,000 D). SOD appears in three forms: (1) Cu-Zn SOD in the cytoplasm with two subunits, and (2) Mn-SOD in the mitochondrion (Warner, 1994). A third extracellular SOD recently has been described contains Copper (CuSOD).

Mechanism of action

SOD is considered fundamental in the process of eliminating ROI by reducing (adding an electron to) superoxide to form H_2O_2. Catalase and the selenium-dependent glutathione peroxidase are responsible for reducing H_2O_2 to H_2O.

The respective enzymes that interact with superoxide and H_2O_2 are tightly regulated through a feedback system. Excessive superoxide inhibits glutathione peroxidase and catalase to modulate the equation from $H_2O_2$to H_2O. Likewise, increased H_2O_2slowly inactivates CuZn-SOD. Meanwhile, catalases and glutathione peroxidase, by reducing H_2O_2, conserve SOD; and SOD, by reducing superoxide, conserves catalases and glutathione peroxidase. Through this feedback system, steady low levels of SOD, glutathione peroxidase, and catalase, as well as superoxide and H_2O_2 are maintained, which keeps the entire system in a fully functioning state (Fridovich, 1993).

SOD also exhibits antioxidant activity by reducing O^{2-} that would otherwise lead to the reduction of Fe^{3+} to Fe^{2+} and thereby promote OH formation. When the catalase activity is insufficient to metabolize the H_2O_2 produced, SOD will increase the tissue oxidant activity. Hence, it was found that the antioxidant enzymes function as a tightly balanced system, any disruption of this system would lead to promotion of oxidation.

Glutathione peroxidase enzyme

The glutathione redox cycle is a central mechanism for reduction of intracellular hydroperoxides. It is a tetrameric protein 85,000D. It has 4 atoms of selenium (Se) bound as seleno-cysteine moieties that confer the catalytic activity. One of the essential requirements is glutathione as a co-substrate.

Glutathione peroxidase reduces H_2O_2 to $H_2O$by oxidizing glutathione (GSH). Re-reduction of the oxidized form of glutathione (GSSG) is then catalysed by glutathione reductase. These enzymes also require trace metal cofactors for maximal efficiency, including selenium for glutathione peroxidase; copper, zinc, or manganese for SOD; and iron for catalase (Halliwell, 1995).

The catalase enzyme

This enzyme is a protein enzyme present in most aerobic cells in animal tissues. Catalase is present in all body organs being especially concentrated in the liver and erythrocytes. The brain, heart, skeletal muscle contains only low amounts.

Catalase and glutathione peroxidase seek out hydrogen peroxide and convert it to water and diatomic oxygen. An increase in the production of SOD without a subsequent elevation of catalase or giutathione peroxidase leads to the accumulation of hydrogen peroxide, which gets converted into the hydroxyl radical.

Other antioxidants

Coenzyme Q1O

CoQ1O (Coenzyme Q1O) is also known as ubiquinone. It is found in almost every living cell (hence the name “ubiquitous”) and is essential to energy production by the mitochondria. Far beyond producing energy, CoQ1O can protect the body from destructive free radicals and enhance immune defences.

Uric acid

Uric Acid acts as an endogenous radical scavenger and antioxidant. It is present in about 0.5mmol/L in body’s fluids and is the end product of purine metabolism. Uric acid is a powerfull scavenger of singlet oxygen, peroxyl radical (ROO) and OH radical (Halliwell, 1994).

Albumin

Depending on the fact that albumin has one sulfhydryl group per molecule; it scavenges several free radicals (Halliwell, 1994) and thus can be considered as one of the primary extracellular defense systems.

Albumin is an additional sacrificial antioxidant that can bind copper tightly and iron weakly to its surface. The bound metals would still be on its surface. The bound metals would still be available for participation in Haber-Weiss reaction, but any generated OH would immediately react with and be scavenged by albumin. The resultant protein damage is biologically insignificant because of the large amount of available albumin and free radicals would be inactivated before reacting with other more vital protein structures.

Other plasma proteins namely ceruloplasmin and transferrin have also shown antioxidant activity. However, antioxidant supply is not unlimited as one antioxidant molecule can only react with a single free radical. Therefore, there is a constant need to replenish antioxidant resources, whether endogenously or through supplementation.

Palm oil

Palm oil is a dark yellow to yellow-red oil, extracted from the mesocarp (flesh of the fruit) of the oil palm (Elaeis guineensis). It is semi-solid at room temperature. Palm fruit oil is consumed worldwide in more than 100 countries. In some parts, the palm fruit oil is often still consumed in its unrefined state, as an ingredient of traditional dishes, where it contributes its characteristic golden red color and unique flavor. However, to most users, palm oil is more familiar as a refined vegetable oil product purchased at their local store and incorporated into their everyday foods. It is healthful, abundantly available, relatively inexpensive, and technically suitable for most food product,

Composition of palm oil

Palm oil consists mainly of glycerides made up of a range of fatty acids. Triglycerides constitute the major component, with small proportions of triglycerides and monoglycerides. Palm oil also contains other minor constituents, such as free fatty acids and non-glyceride components. This composition determines the oil’s chemical and physical characteristics. Crude palm oil contains approximately 1% of minor components: carotenoids, vitamin E tocopherols and tocotrienols), sterols, phospholipids, glycolipids, and other trace impurities (Goh et al., 1985). The most important are carotenoids and Vitamin E, both of which possess important physiological properties.

Antioxidant in palm oil

Carotenoids

Crude palm oil is considered to be the richest natural source of carotenoids (about 15 times more than in carrots). The human body uses carotenoids as Vitamin A. Carotenoids also enhance immune function by a variety of mechanisms, and can improve cardiovascular health. Carotenoids also play an important potential role by acting as biological antioxidants, protecting cells and tissues from the damaging effect of free radicals. A build-up of free radicals

in the body is associated with degenerative diseases such as heart disease and cancer, as well as general ageing. It is, therefore, in one's own best interest to ensure that one eats a diet rich in antioxidants that will prevent the damage that is done to bodies by free radicals.

Red palm oil is a form of processed palm oil (deacidified and deodorized) which retains 80% of the original carotenoids, making it a remarkable source of Vitamin A. These natural antioxidants act as buffers against free radicals and are believed to play a protective role in cellular ageing.

Tocopherols and Tocotrienols: Natural vitamin E exists in eight different forms or isomers, four tocopherols and four tocotrienols. Natural palm oil contains alpha, beta, gamma, and delta tocopherols and alpha, beta, gamma, and delta-tocotrienols. Tocotrienols in Vitamin E have been found to have antioxidant and anti-cancer activities. Its antioxidant properties bring many benefits to the human body, such as preventing skin aging, preventing fat oxidation, reducing blood pressure etc. Tocotrienol-rich fraction of palm oil is capable of protecting brain against oxidative damage and thereby from the ensuing adverse alterations that accompany aging. The antioxidant ability of gamma-tocotrienol may prevent development of increased blood pressure by reducing lipid peroxides and enhancing the total antioxidant status, including superoxide dismutase activity.

CoQ1O: CoQ1O (Coenzyme Q10) is also known as ubiquinone. It is found in almost every living cell (hence the name 'ubiquitous') and is essential to energy production by the mitochondria. Far beyond producing energy, CoQ1O can protect the body from destructive free radicals and enhance immune defences.

Benefits of palm oil

Feeding experiments using various animal models have highlighted that red palm oil is beneficial to health by reducing oxidative stress (Ebong et al., 1999) It is known to be the richest source of carotenoids in terms of alpha and beta carotenes (Sundram et al., 2003) with its wide range of protective properties against disease, aging as well as being modulators for cellular processes/functions by acting as scavengers of oxygen and peroxyl radicals (Van Rooyen et al, 2008). The antioxidant properties of red palm oil (RPO) has been attributed to the synergistic actions of carotenoids and vitamin E in a natural food environment and might provide the ultimate dietary supplement to fight oxidative stress associated with aging.

Effect of heating on palm oil

It is known that fats and oils, when subjected to prolonged heating (for usually above smoke point; 235°C for palm oil), are subjected to a series of chemical-physical modifications, the effects of which can be observed in the variation of it nutritional characteristics. The main factors influencing the entity of these transformations are represented by the temperature and the time of the treatment, the nature of foods being fried, the presence of metals that catalyze the oxidation phenomena, and the composition of the frying oil. Increasing temperature progressively decrease the beta-carotene content of palm oil and loss of its reddish colour, and consequent adverse nutritive effects (Owojuyigbe, 2003; Nwosu *et al.*, 2016; Obeagu, 2018, Eze *et al.*, 2016). Therefore it is necessary to evaluate the health benefits of red palm oil following frying.

Conclusion

The importance of genetics in the regulation of biologic aging is shown by the characteristic longevity of each animal species. Deoxyribonucleic acid (DNA) undergoes continuous changes in response to exogenous agents and intrinsic processes. Stability is maintained by the double-strandedness of DNA and by specific repair enzymes. An antioxidant is a substance that when present in low concentrations relative to the oxidizable substrate significantly delays or reduces oxidation of the substrate. The body has developed several endogenous antioxidant systems to deal with the production of Reactive Oxygen Intermediate (ROI). Palm oil also contains other minor

constituents, such as free fatty acids and non-glyceride components. Crude palm oil is considered to be the richest natural source of carotenoids. Carotenoids also play an important potential role by acting as biological antioxidants, protecting cells and tissues from the damaging effect of free radicals. Because of high level of antioxidants content of palm oil, it can enhance longevity of cell as well as the whole body.

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