CHAPTER II
LITERAURE REVIEWS

The contents of the literature review were divided to 4 parts

Part I Skin

Skin is the largest organ of the integumentary system made up of multiple layers of epithelial tissues that guard underlying muscles and organs[12]. Skin pigmentation varies among populations, and skin type can range from dry skin to oily skin [13].

Skin performs the following functions [12, 14, 15]**:**

1. Protection: an anatomical barrier from pathogens and damage between the internal and external environment in bodily defense.
2. Sensation: contains a variety of nerve endings that react to heat and cold, touch, pressure, vibration, and tissue injury.
3. Heat regulation: the skin contains a blood supply far greater than its requirements which allows precise control of energy loss by radiation, convection and conduction.
4. Control of evaporation: the skin provides a relatively dry and impermeable barrier to fluid loss. Loss of this function contributes to the massive fluid loss in burns.
5. Aesthetics and communication: others see our skin and can assess our mood, physical state and attractiveness.
6. Storage and synthesis: acts as a storage center for lipids and water, as well as a means of synthesis of vitamin D by action of UV on certain parts of the skin.
7. Excretion: Sweat contains urea, hence excretion by sweating is at most a secondary function to temperature regulation.

8. Absorption: oxygen, nitrogen and carbon dioxide can diffuse into the epidermis in small amounts. In addition, medicine can be administered though the skin. The skin is an important site of transport in many other organisms.
9. Water resistance: the skin acts as a water resistant barrier so essential nutrients aren't washed out of the body

Skin structure

Skin is composed of three primary layers [15]:

- the **epidermis**, which provides waterproofing and serves as a barrier to infection;
- the **dermis**, which serves as a location for the appendages of skin; and
- the **hypodermis** *(subcutaneous adipose layer)*.

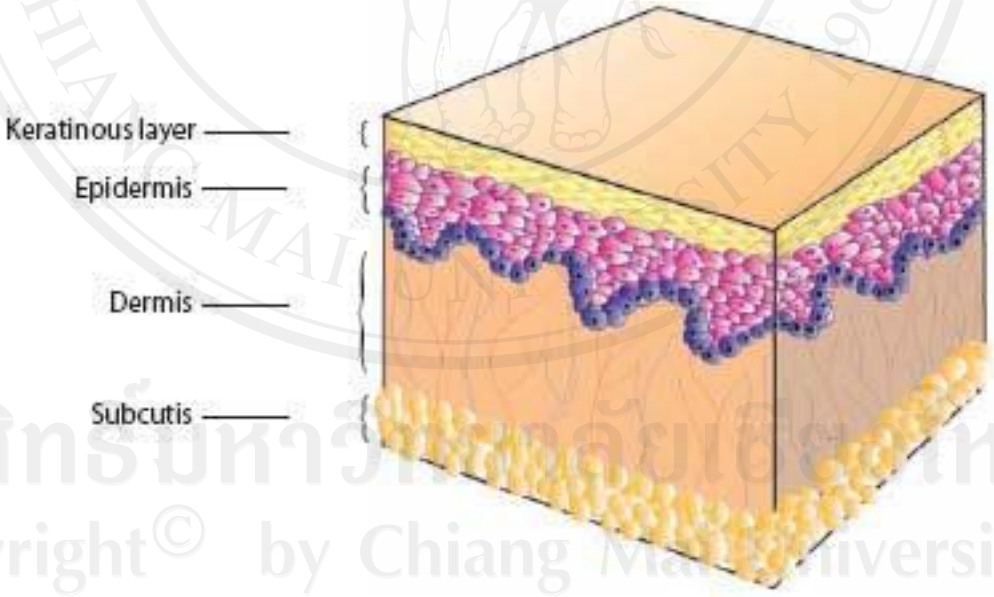

Figure 2.1 Structure and layers of the skin [12].

Epidermis

Epidermis is the outermost layer of the skin [14]. It forms the waterproof, protective wrap over the body's surface and is made up of stratified squamous

epithelium with an underlying basal lamina. Epidermis is divided into the following 5 sublayers or strata [16] as shown in figure 2.1.

- Stratum corneum (top layer)
- Stratum licidum
- Stratum granulosum
- Stratum spinosum
- Stratum basale (or stratum germinativum)

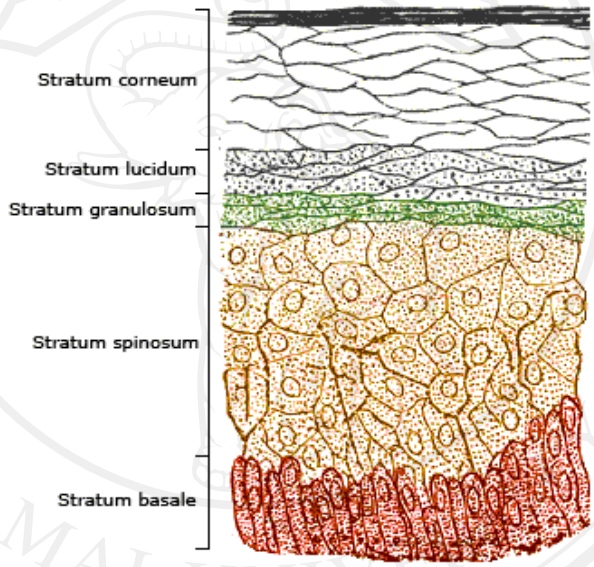

Figure 2.2 Layers of the epidermis (copy from http://en.wikipedia.org)

The stratum corneum is the outermost layer of the epidermis, composed of large, flat, polyhedral, plate-like envelopes filled with keratin, which is made up of dead cells that have migrated up from the stratum granulosum.

The stratum lucidum is a layer that found only in palmoplantar skin (the thicker skin of the palms and soles), between the stratum granulosum and stratum corneum layers. It is composed of three to five layers of dead, flattened keratinocytes .

In the stratum granulosum (or granular layer), keratinocytes are now called granular cells, and contain keratohyalin and lamellar granules.

The stratum spinosum also referred to as the "spinous" or "prickle-cell" layer. Keratinization begins in the stratum spinosum.

Stratum basale (or stratum germinativum) is the deepest layer of the epidermis. The basal cells of this layer can be considered the "stem cells" of the epidermis, undifferentiated, proliferating, and creating daughter cells that migrate upward, beginning the process of differentiation [17].

Dermis

The dermis is a layer of skin between the epidermis and subcutaneous tissues, and is composed of two layers, the papillary and reticular dermis. Structural components of the dermis are collagen, elastic fibers, and extrafibrillar matrix (previously called ground substance). It contains the hair follicles, sweat glands, sebaceous glands, apocrine glands, lymphatic vessels and blood vessels [15-17].

Layer of dermis

- Papillar dermis: the upper layer, contains a thin arrangement of collagen fibers.
- Reticular dermis: The lower, is thicker and made of thick collagen fibers that are arranged parallel to the surface of the skin.

Hypodermis (Subcutaneous)

The hypodermis is the innermost and thickest layer of the skin. It invaginates into the dermis and is attached to the latter, immediately above it, by collagen and elastin fibers. It is essentially composed of a type of cells specialised in accumulating and storing fats, known as adipocytes. These cells are grouped together in lobules separated by connective tissue [12-15].

The hypodermis acts as an energy reserve. The fats contained in the adipocytes can be put back into circulation, via the venous route, during intense effort or when there is a lack of energy providing substances, and are then transformed into energy. When we speak of "burning up calories", we are burning up fats in particular. The

hypodermis participates, passively at least, in thermoregulation since fat is a heat insulator [14].

Part II Skin aging

Aging can be viewed as the accumulation of changes in cell and tissues resulting from a greater disorderliness of regulatory mechanism [18] that result in reduced robustness of the organism to encountered stress and disease [19]. It is a consequence of both genetic program and cumulative environmental effects. Skin transformations are the most perceptible signs of aging [7]. The multifaceted functions of the skin include acting as an entry barrier to compounds, regulation body temperature and fluid and electrolyte balance, and providing receptors for sensations such as touch, pain and pressure [20]. Cutaneous aging is a mix of instrinsic aging (due to inherent genetics) and extrinsic aging (due to environmental conditions such as solar exposure) [21, 22]. During this process of aging, the skin becomes thinner, wrinkled and saggy with graying of hair. This also has ramifications on the permeability characteristics and various functions of the skin.

Free radical theory highlights the role and function of the external factors [23]. According to this theory, aging results from accumulation of cellular damage produced by excess reactive oxygen species (ROS) that are generated as a consequence of oxidative metabolism [24]. Age-associated cellular damage includes oxidation of DNA resulting in mutations, oxidation of proteins causing their reduced function, and oxidation of membrane lipids affecting transport efficiency and possibly transmembrane signaling [25]. The main source of excess ROS implicated in aging is mitochondrial oxidative energy generation [26]. Antioxidizing nutrients are believed to play a role in the prevention and treatment of a variety of chronic diseases [27]. The proposed mechanism by which antioxidants protect cells from oxidative stress is by scavenging free radicals and halting lipid peroxidation chain reactions, which can cause DNA damage [28].

Instrinsic aging

In the skin both genetic (intrinsic) and exogenous factors contribute to the phenotypic and functional changes occurring with age. Chronologically aged skin is dry, lax and atrophic with fine wrinkles and a variety of benign neoplasms [7, 29]. The most consistent histological changes of intrinsic cutaneous aging include flattening of the dermal-epidermal junction. This results in a considerably smaller contact surface between the epidermis and dermis and presumably less communication and nutrient transfer.

Functional changes in skin during intrinsic aging include slow wound healing due to decreased keratinocyte and fibroblast proliferating ability, reduced cytokine production, and delayed recovery of barrier function after damage [30]. The barrier to water loss is more easily disturbed, in part because of decreased lipid synthesis.

The activity of enzymes involved in synthesis and degradation of extracellular matrix proteins is affected by aging. While expression of collagenases and metalloproteinases increases, the level of the tissue inhibitor of metalloproteinases 1 is decreased [31]. Therefore, a shift in balance between synthesis and degradation of collagen occurs that causes a reduction in dermal thickness.

Extrinsic aging

Since skin is in direct contact with environment, it undergoes changes as a consequence of external factors. Among harmful environmental factors that contribute to the extrinsic aging of the skin, exposure to UV light (photoaging) is considered to be the most significant and well recognized. The term photoaging has been coined by Kligman in 1989. Photoaging refers to the effects of long-term UV exposure superimposed on intrinsically aged skin [3, 32]. Photodamaged skin appears sallow, irregularly pigmented, wrinkled, atrophic, with multiple telangiectases, and variety of premalignant lesions [33]. Histological changes in photodamaged skin include thickening of the epidermis, disorganization and cytologic atypia of the keratinocytes, uneven distribution of melanocytes in basal layer with significant decrease of Langerhans cells and masses of amorphous elastic material in the papillary dermis [34]. Deposition of amorphous elastic material in the papillary dermis instead of a normal connective tissue is considered to be the principal element differentiating chronological aging from photoaging. Damage to the collagenous matrix is thought to

underlie the course, rough, wrinkled appearance of photodamaged skin [35, 36]. The main changes in skin components of intrinsically aged and photoaged skin are summarized in table 2.1.

Table 2.1 Changes in intrinsic and photodamaged skin [37]

Skin components and cells	Intrinsic aging	Photoaging
Epidermis	decrease [38] or constant [39]	increase
Dermis	decrease	decrease
Dermal-epidermal junction	normal to flat	flat
Keratinocytes	decrease in proliferative capacity	loss of polarity, atypia
Melanocytes	decrease	increase
Langerhans cell	decrease	decrease
Collagen	disorganized	severely disorganized
Elastic fibers	decrease	increase, abnormal
Blood vessels	loss of vascular loops	ecstatic vessels with atrophic walls
Fibroblasts	decrease	decrease
Inflammatory infiltrate	absent	present
Matrix metalloproteinase activity	increase	increase
Inhibitors of matrix metalloproteinase activity	decrease	decrease

Mechanism of photodamage

UVB irradiation is absorbed maximally by DNA and creates photoproducts, such as cyclobutane pyrimidine dimers and pirymidine pyrimidone photoproducts [40]. These mutations are clinically relevant to premalignant cutaneous tumors and skin malignancies. However, their relevance to other clinical manifestations of

photoaging, such as wrinkles, is not completely elucidated. The role of UVA in skin aging is carried out through the generation of ROS. These unstable molecules damage the DNA, cellular membranes, lipids and proteins [41]. The marker of the UVA damage is considered to be a 'common deletion' in the mitochondrial DNA [33]. Since mitochondria have the highest ROS turnover in the cell, mutations in the mitochondrial genome may be associated with the changes seen with UVA-induced photoaging. Ultraviolet irradiation invokes a complex sequence of specific molecular responses that damage skin connective tissue as shown in figure 2.3 [37]. UV irradiation disrupts the skin collagen matrix by two interdependent pathways: stimulation of collagen degradation and inhibition of collagen production [42].

The primary mechanism by which UV irradiation initiates molecular responses in the skin is by photoproduction of ROS, which induce signaling pathways such as intracellular kinases [43]. Activated kinases upregulate expression and activation of transcription factors, such as activated protein 1 (AP-1) and nuclear transcription factor-κB (NF-κB). Nuclear transcription factor AP-1 stimulates transcription of genes for matrix-degrading enzymes such as metalloproteinase (MMP) 1 (collagenase), MMP3 (stromelysin 1), and MMP9 (92-kDa gelatinase) [44, 45]. Ultraviolet-induced MMP1 initiates cleavage of type I and III collagens in skin [46]. Once cleaved by MMP1, collagen can be further degraded by elevated levels of MMP3 and MMP9 [37]. Thereby, UV irradiation degrades skin collagen and impairs the structural integrity of the dermis. Collagen VII reduction was also found in photodamaged skin [33]. Collagen VII composes anchoring fibrils and, thus, is important in maintaining dermal-epidermal junction integrity.

NF-κB is also activated by UV light [47]. This transcription factor stimulates the transcription of inflammatory cytokines, such as interleukin I, VI, and tumor necrosis factor-α and therefore is involved in attraction of neutrophils containing preformed neutrophil collagenases [48]. In addition to degrading mature dermal collagen, UV irradiation impairs ongoing collagen synthesis. It has been found that collagen I formation is significantly decreased in the papillary dermis of photodamaged skin primarily through downregulation of type I and type III procollagen gene expression [49]. The two mechanisms contributing to reduction in procollagen gene expression are induction of transcription factor AP-1 and

downregulation of type II transforming growth factor-β (TGF-β) receptor. Finally, damaged collagen itself downregulate new collagen synthesis. Poor adhesion of fibroblasts to damaged collagen causes a decreased neocollagenesis [50].

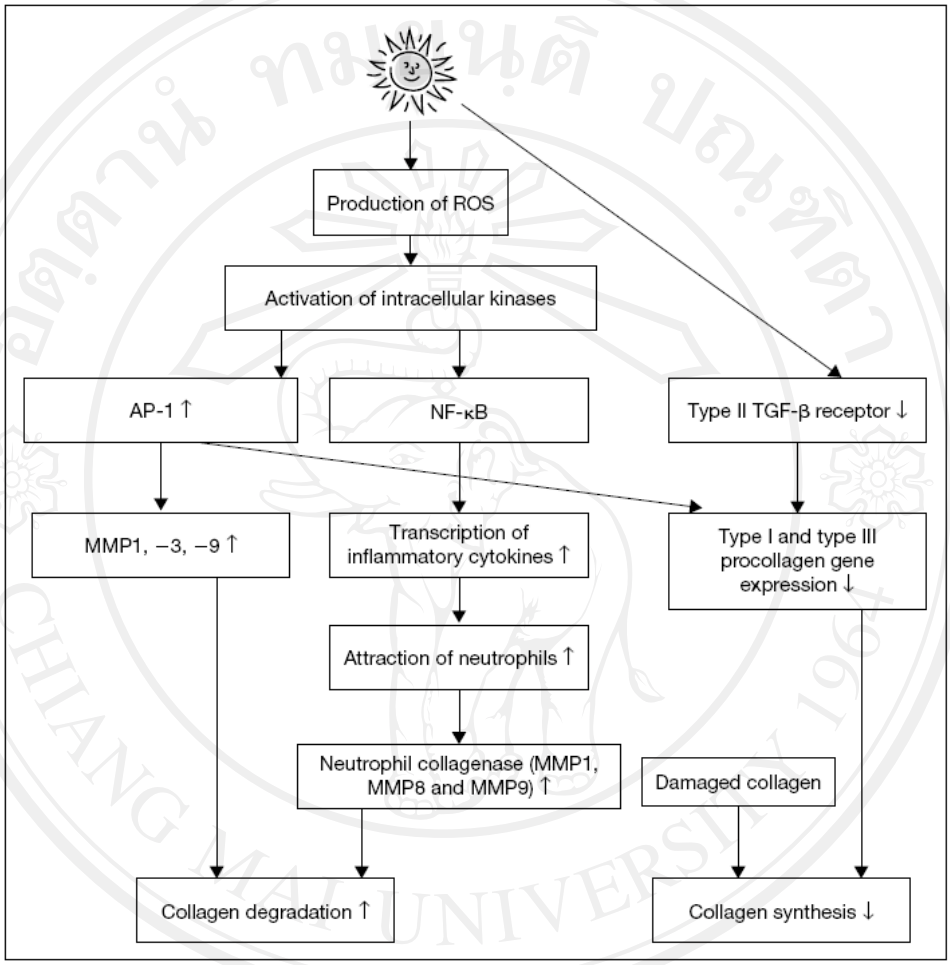

Figure 2.3 The effect of UV light on collagen metabolism [37].

Wrinkle

Wrinkles are the most obvious—perhaps one of the most disliked—aspects of facial aging. Considering this, it is surprising that relatively little is known about what causes a wrinkle, what is its physical structure, and why it is so hard to erase a wrinkle once it is formed. The key findings of winkle are as follows:

- The epidermis thins with age and the properties of the stratum corneum change in a multitude of ways including reduced barrier repair properties and decreased elasticity. These changes are particularly

important in causing ‘‘fine lines’’ and are the changes that are countered by the vast majority of effective topical moisturizing products.

- The intricate, interconnected, elastic fiber network in the dermis degrades and is replaced (especially in the case of sun-exposed skin) by large deposits of poorly organized elastin deep in the dermis. This leads directly to loss of resiliency—the ability of the skin to quickly return to its original shape after distortion. In extreme cases, the large elastin deposits (so-called elastosis) can cause the skin to look sallow and yellow in color.
- Collagen fibers become less well organized and the collagen itself undergoes chemical changes that reduce its mechanical flexibility. Repeated imperfect collagen repair can lead to ‘‘scar like’’ patches of stiff, aligned collagen.
- Glycosaminoglycans composition changes. In sun-exposed skin with chronic (i.e., years) of photodamage there is a marked increase in certain types of sulfated glycosaminoglycans (versican) at the expense of others (decorin). Since these molecules can hold as much as 1000 times their own weight of water in an elastic gel these changes hugely impact the water content of the dermis.
- The subcutaneous fat layer (especially in women) decreases dramatically in thickness.

That skin which has undergone all these deleterious changes is more prone to wrinkles is self-evident—however, these changes are not sufficient to cause some types of wrinkling to occur. It is quite possible to find individuals and areas of skin where all these histological and biochemical changes can be seen but no wrinkles are visible. The extra factor that seems to be necessary is the repeated movement of the skin causing folding of the skin. Over time, and combined with the overall aging changes set out above, wrinkles form and progressively get worse. Thus, wrinkling can be seen on the face where ‘‘expression lines’’ form but not on the upper body even though the histological degradation of the skin may be worse in areas of the body. Around the mouth, smoking also makes a marked and very adverse contribution

to wrinkling.

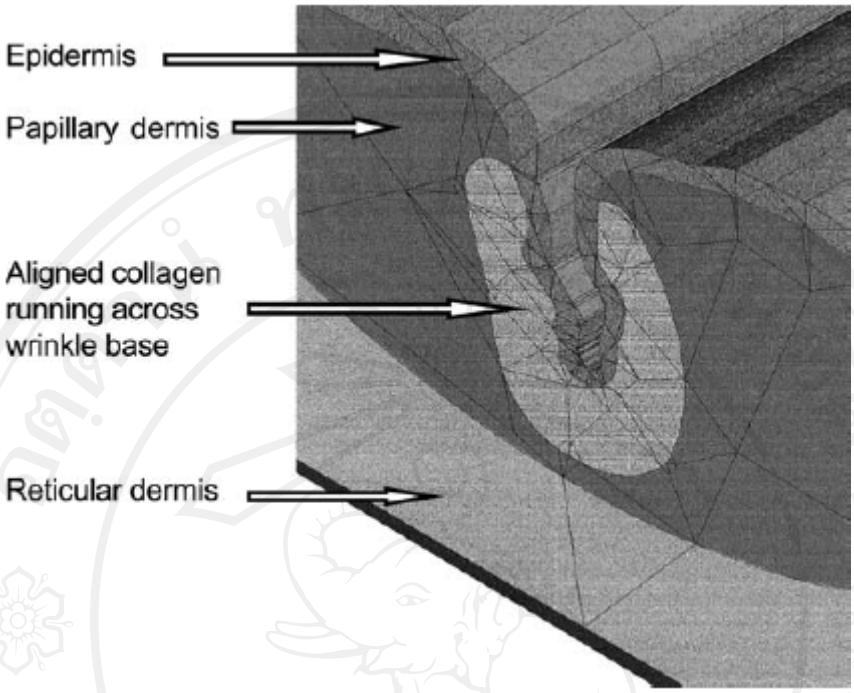

Figure 2.4 Computer model of the wrinkle [51].

Part III Free radical and antioxidation

Free radical

In chemistry, radicals (often referred to as free radicals) are atoms, molecules, or ions with unpaired electrons on an open shell configuration [25]. The unpaired electrons cause them to be highly chemically reactive [52]. Once formed these highly reactive radicals can start a chain reaction, like dominoes. Their chief danger comes from the damage they can do when they react with important cellular components such as DNA, or the cell membrane [53]. Cells may function poorly or die if this occurs.

Type of free radical

- **Reactive oxygen species, ROS**

  - Superoxide anion (O_2^-),

  - Hydroxyl radical ($OH^{\bullet}$),

- Singlet oxygen ($^1O_2$),
- Hydrogen peroxide (H_2O_2)

- **Reactive nitrogen species, RNS**
  - Nitric oxide, $NO^{\bullet}$
- **Reactive chlorine species, RCS**
  - Atomic chlorine, Cl

Reactive oxygen species (ROS) are free radicals that contain the oxygen atom [54]. ROS thus have "extra" electrons. ROS include the familiar oxygen compound hydrogen peroxide, H_2O_2, produced when O_2^- is reduced with two electrons, and reactive forms of oxygen including superoxide, O_2^- , and hydroxyl radical, $OH^{\bullet-}$ [55]. Complete reduction of O_2 by addition of four electrons yields molecules of water, H_2O, a stable compound that is not an ROS [56].

All of the ROS (superoxide, hydrogen peroxide, and hydroxyl radical) are produced as a consequence of normal metabolism, and have roles as cell signaling molecules as well as in defense from invading micro-organisms. However, when any ROS is produced in uncontrolled amounts it can damage proteins, DNA, and lipids [57]. Mechanism of cell injury from free radical was shown in figure 2.5 [58].

In chemistry, free radicals take part in radical addition and radical substitution as reactive intermediates. Chain reactions involving free radicals can usually be divided into three distinct processes: *initiation*, *propagation*, and *termination* [53].

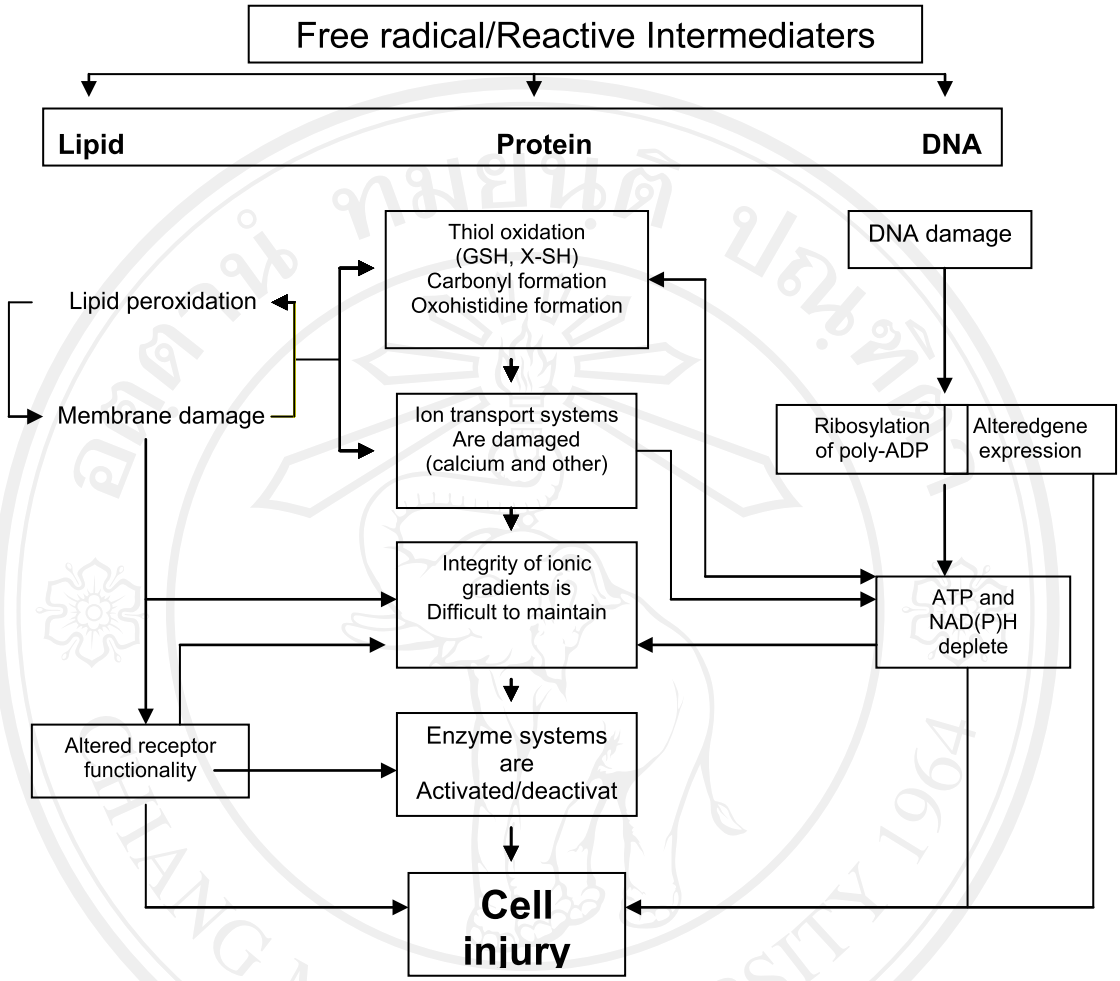

Figure 2.5 Mechanism of cell injury from free radical [58]

- **Initiation** reactions are those that result in a net increase in the number of free radicals. They may involve the formation of free radicals from stable species as in Reaction 1 above or they may involve reactions of free radicals with stable species to form more free radicals [24, 58].

Four mechanisms produce free radical.

- Homolysis
- Photolysis
- Radiolysis
- Redox reactions

- **Propagation reactions** are those reactions involving free radicals in which the total number of free radicals remains the same. It occurs when the number of radicals is conserved [24].

Four major mechanistic types of propagations have been identified that may be relevant to biology.

a) Atom(or group) transfer

b) electron transfer

c) addition

d) ß scission

- **Termination reactions** are those reactions resulting in a net decrease in the number of free radicals. Typically two free radicals combine to form a more stable species. There are three molecular processes which can terminate a chain of radical reaction [59].

1. Homolinking and Cross-Linking of radical
2. Radical scavenging
3. Electron transfer

Lipid peroxidation

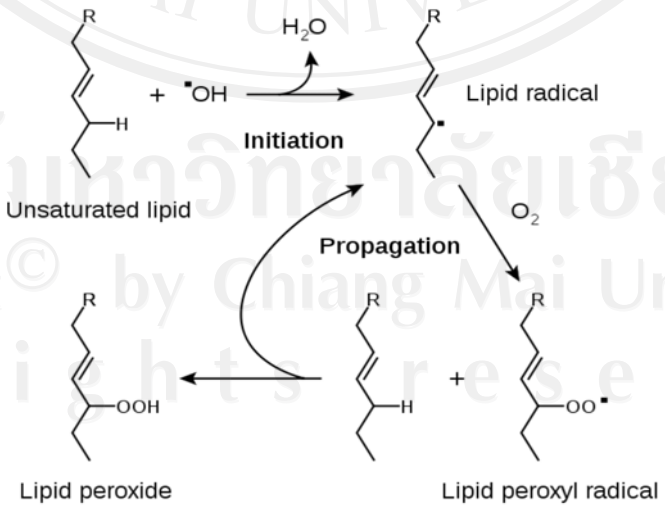

Figure 2.6 Mechanism of lipid proxidation (copy from http://en.wikipedia.org)

Lipid peroxidation refers to the oxidative degradation of lipids. It is the process whereby free radicals "steal" electrons from the lipids in cell membranes, resulting in cell damage [60]. This process proceeds by a free radical chain reaction mechanism. It most often affects polyunsaturated fatty acids, because they contain multiple double bonds in between which lies methylene -CH_2- groups that possess especially reactive hydrogens. As with any radical reaction the reaction consists of three major steps: initiation, propagation and termination (figure 6). Lipid peroxidation is the important reaction that can cause skin aging [61].

Active oxygen species, including $O_2^{\bullet-}$, $^{\bullet}OH$, $HO_2^{\bullet}$, and $LOO^{\bullet}$ can initiate and propagate lipid peroxidation reactions (figure 2.6). Free radical mediated lipid peroxidation generally requires radicals and weakly bonded polyunsaturated fatty acids for the initial steps. Oxygen and a lipid radical are necessary for the intermediate or propagation steps which ultimately produce lipid peroxidation byproducts [62].

Antioxidant

Free radicals have the capacity to react in an indiscriminate manner leading to damage to almost any cellular component, an extensive range of antioxidant defenses, both endogenous and exogenous, are present to protect cellular components from free radical induced damage [63].

An antioxidant is a molecule capable of slowing or preventing the oxidation of other molecules [64]. Oxidation is a chemical reaction that transfers electrons from a substance to an oxidizing agent. Oxidation reactions can produce free radicals, which start chain reactions that damage cells [27]. Antioxidants terminate these chain reactions by removing free radical intermediates, and inhibit other oxidation reactions by being oxidized themselves. As a result, antioxidants are often reducing agents such as thiols, ascorbic acid or polyphenols [65]. Antioxidants can be either endogenous compounds, produced by the organism as part of its ROS defense, or can be exogenous compounds acquired from the diet. The endogenous system includes both enzymes and nonenzymatic antioxidants, and dietary antioxidants are small molecules (figure 2.7).

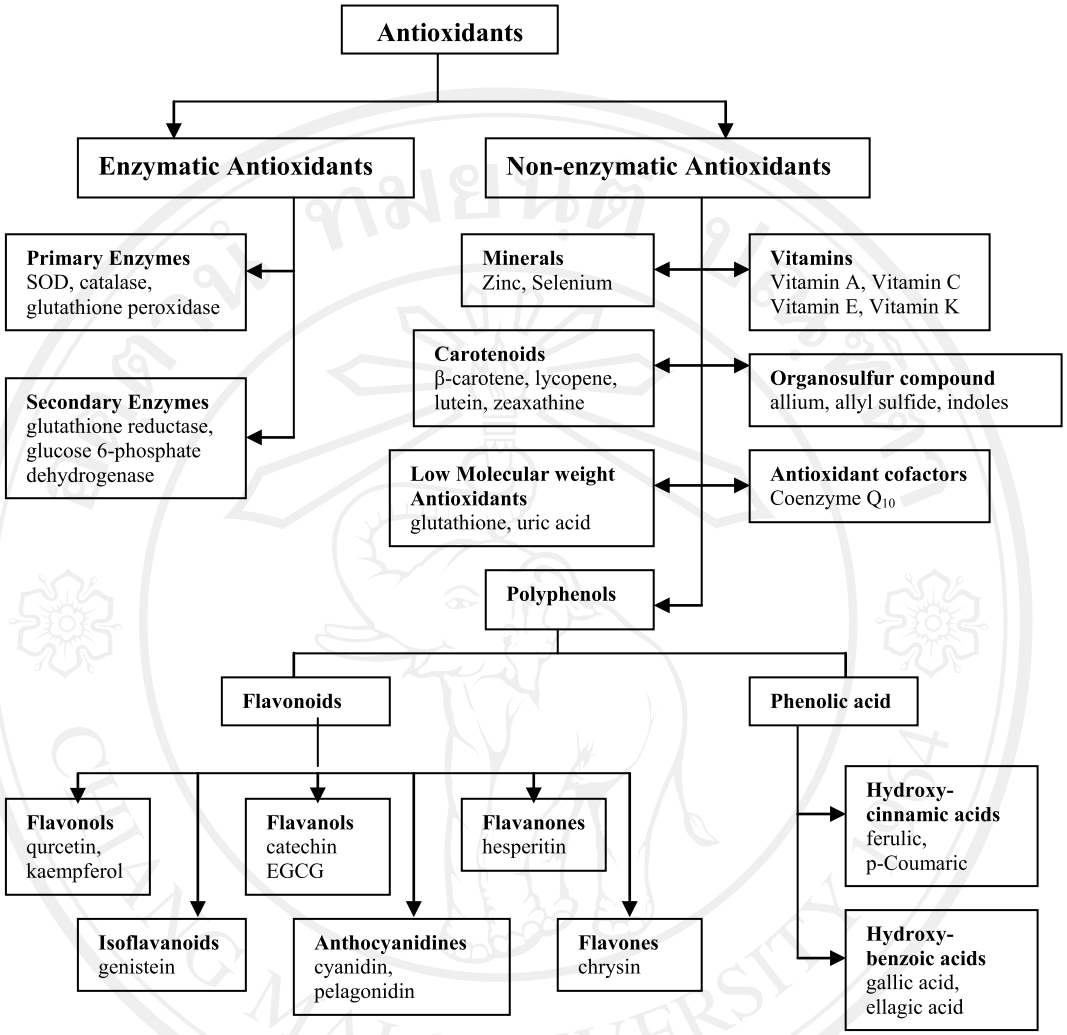

Figure 2.7 Type of antioxidants [66-68]

Table 2.2 Major enzymatic physiological antioxidants [68]

Enzymatic antioxidants	**Properties**
Superoxide dismutase	•located in both mitochondria and cytosol •dismutates superoxide radicals
Glutathione peroxidase	•located in mitochondria, cytosol and cell membrane •removes hydrogen peroxide and organic hydroperoxides
Catalase	•located primarily in peroxisomes •removes hydrogen peroxide

Table 2.3 Major nonenzymatic physiological antioxidants [68]

Non-enzymatic antioxidants	**Properties**
Vitamin E	•lipid soluble phenolic compound; major chain breaking antioxidant found in cell membranes,
Vitamin C	•major classes: tocopherols and tocotrienols
Vitamin A	•located in aqueous phase of cell; acts of radical scavenger and recycles vitamin E •derived from cleavage of carotene in intestine •has biological activity of retinol •unsaturated lipid •inhibits lipid peroxidation
Uric acid	•by-product of purine metabolism in humans and higher apes; may be an important physiological antioxidant; scavenges hydroxyl radicals
Glutathione	•nonprotein thiol in cells; serves multiple roles in the cellular antioxidant defense
a-Lipoic acid	•effective as an antioxidant and in recycling vitamin C •potent pro-glutathione agent
Carotenoids	•lipid soluble antioxidants located primarily in membranes of tissues •major component of "phytochemicals"
Flavonoids and polyphenols	•lipid soluble radical savenger and metal chelators
Bilirubin	•by-product of heme metabolism; may serve as an extracellular antioxidant
Ubiquinones	•lipid soluble quinone derivatives; reduced forms are efficient antioxidants
Melatonin	•pineal hormone •thought to be localized in nucleus of cell •lipid soluble radical scavenger

Phenolic as antioxidant

Phenolics are an ecologically significant class of secondary metabolites, accounting for about 40% of the organic carbon circulating in the biosphere [69]. Much of this photoassimilated carbon is in the form of lignin, suberin and related structural polymers, which have clearly defined roles in mechanical support, vascular transport and structural reinforcement of long-lived plant tissues [70].

Phenolic compounds are excellent antioxidants by virtue of the electron donating activity of the 'acidic' phenolic hydroxyl group. Indeed, many natural and synthetic antioxidants, including α-tocopherol (Vitamin E) and butylated hydroxytoluene (BHT), are phenolic compounds. Two properties of phenolic compounds account for their radical scavenging properties. First, the one-electron reduction potentials of phenolic (phenoxyl) radicals are typically lower than those of oxygen radicals such as superoxide ($O2^{-\bullet}$), peroxyl ($ROO^{\bullet}$), alkoxyl ($RO^{\bullet}$) and hydroxyl ($HO^{\bullet}$) radicals, meaning that these species will readily oxidize phenolics to their respective phenoxyl radicals [71, 72]. Second, phenoxyl radicals are generally less reactive than oxygen radicals [73]. Consequently, phenolic compounds can directly scavenge harmful reactive oxygen intermediates and inactivate them without promoting further oxidative reactions.

Many studies have demonstrated the radical scavenging properties of plant phenolic compounds. Rice-Evans *et al*. (1996) measured the radical scavenging activity of flavonoids and phenolic acids based on their ability to scavenge a preformed radical cation chromophore of 2, 2'-azinobis-(3-ethylbenzothiazoline-6-sulfonic acid) ($ABTS^{+\bullet}$) at pH 7.4. Among flavonoids, the highest scavenging activities were found for the flavonol quercetin, the anthocyanidins cyanidin and delphinidin, and the green tea flavan-3-ols epicatechin gallate and epigallocatechin gallate [64].

Among flavonoids, antioxidant activity is further enhanced by the presence of a 2, 3 double bond on the C ring, a free hydroxyl group at the 3 position on the C ring and the presence of hydroxyl groups in the 3 and 5 positions on the A ring (figure 2.8) [74].

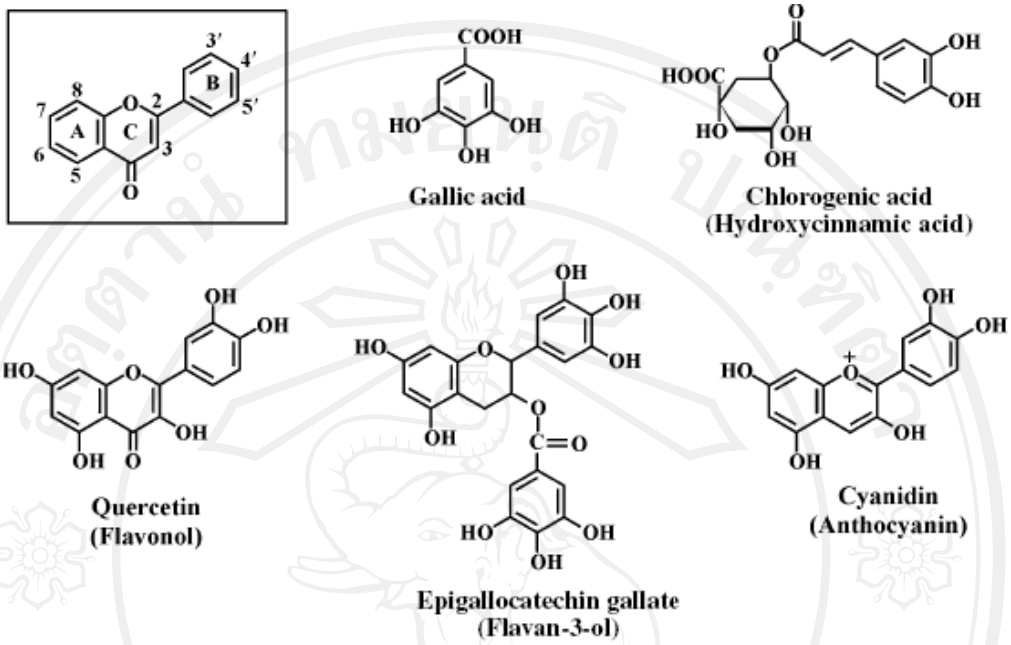

Figure 2.8 Numbering system for the flavonoid ring and examples of plant phenolic compounds with demonstrated antioxidant activity [70].

Part IV Plant material

Figure 2.9 *Litchi chinensis* Sonn.

Scientific name	*Litchi chinensis* Sonn .
Family name	Sapindaceae
Common name	Litchi, Laichi, Lychee, Lici, Lichia

Lychee is the most renowned of a group of edible fruits of the soapberry family, Sapindaceae. It is botanically designated as *Litchi chinensis* Sonn. (*Nephelium litchi* Cambes) and widely known as Litchi and regionally as Litchi, Laichi, Lychee, Lici, Lichia [10].

Lychee in Thailand

There are two groups of cultivars grown in Thailand. The cultivars of the first group are grown in lowland areas of the Central, Eastern and Western regions and they require moderately low temperatures for flowering and fruiting. The most important cultivar of this group is “Kom”, a compact canopy. Other cultivars of less importance are “Kra-lok Bai-Yaw”, Sampao Kaew”, Sa-rack Tong”, “Jean”, “Jean Yai”, “Tai”,”Tai Yai” and Pantip. The cultivars of the second group reuire lower temperatures for their flowering and fruiting and are grown in the north. “Hong Huay” is the most important cultivar of this group, followed by “Chakrapad”, “Kim Cheng”,

“O-Hia”, and a few other. “Chakrapad” usually fetches higher prices due to its larger fruit size.

Thailand exports Lychee to Singapore, Malaysia, Hong Kong, Europe and USA. In 1999, Hong Kong was the largest importer of fresh lychee, while Malaysia and the USA were the major importers of canned lychee.

Botanical Description [10]

The botany of Lychee has been reviewed by Groff (1921), Singh and Singh (1954), Joubert (1985, 1986), Menzel and Simpson (1986, 1990), Menzel *et al*. (1993) and Stern and Gazit (2003). The species is a medium to large evergreen tree, which can grow up to 10–12 m or even 20 m in very old specimens. Its crown is generally round, dense, compact and symmetrical. In some cultivars, the branches are tightly curved or twisted and hang down to the ground. Some cultivars are more erect than others. Trees normally have a thick, straight, short trunk and dark brown-grey bark; however, branches often have V-shaped crotches and are easily broken off by strong winds.

The leaves are pinnately compound with 4–7 leaflets about 7 cm long. They are glossy dark green on the upper surface and grey-green on the under surface. The leaflets are arranged in opposite or slightly oblique order along the rachis on short petioles of their own. Mature leaflets are usually 5–15 cm long and 2.5–4.0 cm wide, and elliptical to lance-shaped. The bases of the blades are wedge-shaped or rounded.

The inflorescence is determinate and composed of several panicles produced on current-season shoots. The panicles are normally produced terminally in clusters of ten or more, but in some trees a high proportion of subterminals or axillaries may be produced. Panicles are generally in mixed form with the lowest buds producing leaves only, the middle buds producing floral buds in the axils of the leaves, and the topmost buds producing only floral branches and sometimes very small leaves that do not persist. Panicles are 10–40 cm long and produce hundreds of small, white, green or yellow flowers, which produce a distinctive scent when the tree is in full bloom.

The flowers are 3–6 mm wide when fully open and rest on 1.5 mm pedicles. They possess a cup-shaped calyx with 4–5 short, serrated sepals, but have no petals. Each flower has 6–10 stamens. Flowers are usually produced from late autumn to

early spring, with three types opening in succession on the same panicle. The flowers vary in sex, length and function of the stamens, and development and function of the.

Fruit take 80–112 days to mature depending on the cultivar and weather. The fruit are drupes and may be round, ovoid or heart-shaped, varying up to 5 cm long and 4 cm wide. The skin or pericarp is thin, tough, hard and frangible. Fruit are green when immature and various shades of red when mature. The skin has sharp protuberances in some cultivars, but is smooth in others.

The edible part of the fruit is an aril formed from cells in the outer seed coat. The aril grows continuously from about 4 weeks after flowering and separates easily from the seed and pericarp at harvest. The aril contains little material from the cell membranes and is white to off-white, translucent, slightly acid, juicy and sweet, and similar in texture to a grape.

Fruit with aborted seed

Fruit with normal seed

Figure 2.10 Longitudinal sections of mature Lychee showing normal and aborted seed [10]

The fruits normally contain one chestnutbrown to dark-brown, ovoid to oblong seed, 1.0–3.3 cm long and 0.6–1.2 cm wide. In some cultivars, a high proportion of

seeds may be abortive. The abortive seeds are small and shrivelled and are known as ‘chicken tongues’ (Figure 2.10.). Fruit with abortive seeds are preferred and often attract a high price, for although they are somewhat smaller than fruit with normal seeds, they usually contain a higher proportion of flesh. The proportion of small or shriveled seeds is an important characteristic of a cultivar and varies from season to season and orchard to orchard. Fruit with chicken-tongue seeds are more susceptible to drought and other stresses and are usually shed before fruit containing normal seeds. Average fruit weight is 16–35 g and flesh recovery 50–70%.

Chemical constituents and biological activity of Lychee

Fruit pericarp of Lychee are found to contain flavonol and anthocyanins as the major components. List of compounds and their biological activity found in fruit pericarp of Lychee is shown in table 2.4.

Table 2.4 Chemical constituents and biological activity of Lychee fruit pericarp [75-77]

Chemical substance	Category
- cyanidin -3-rutinoside	Anthocyanin
- cyanidin -glucoside	Anthocyanin
- quercetin -3-rutinoside (rutin)	Anthocyanin
- quercetin glucoside	Anthocyanin
- procyanidin B4	Flavonol
- procyanidin B2	Flavonol
- epicatechin	Flavonol

Litchi flavanols and anthocyanins exhibit good potential antioxidant activity [77]. Anthocyanins from litchi fruit pericarp strongly inhibited linoleic acid oxidation and exhibited a dose-dependent free radical scavenging activity against DPPH radical, superoxide anions and hydroxyl radical [78]. Moreover lychee fruit pericarp presented the anticancer activity against human breast cancer [79, 80]. A large number of polysaccharides are present in the pericarp tissues of harvested litchi fruits. The

polysaccharide fractions extracted and purified from litchi fruit pericarp tissues exhibited strong antioxidant activities [81].

Lychee seed was the next interesting part in antioxidant activity. Lychee seed extract showed the strong antioxidant capacity, scavenging the 1,1-diphenyl-2-picryl hydrazyl (DPPH) radical and inhibitory activity against lipid peroxidation. The five phenolic compounds, namely, gallic acid, procyanidin B2, (-)-gallocatechin, (-)-epicatechin and (-)-epicatechin-3-gallate were identified [82].