The Therapeutic Efficacy and Properties of Topical *Aloe Vera* in Thermal Burns

Juraiporn Somboonwong MD, M.Sc*,
Naphatsanan Duansak MSc**

** Department of Physiology, Faculty of Medicine, Chulalongkorn University.*
*** Inter-Department of Physiology, Graduate School, Chulalongkorn University*

Aloe vera has been used as a popular herbal medicine since ancient times for many conditions including burns. Much evidence has reported the efficacy of topical Aloe vera gel in the treatment of thermal burns through its different pharmacological actions. This review article consists of pathophysiology of the thermal burns, the botany and chemical constituents of Aloe vera, and therapeutic properties of Aloe vera on thermal burns. The mechanisms that may underlie its action include: anti-inflammation, antimicrobials, wound healing promotion, and biological/immunological modulation.

Keywords: *Herb, Thermal burns, Aloe vera, Wound healing*

Aloe vera has been used medicinally for centuries, certainly since Roman times and perhaps long before[1,2], yet most physicians seem to know little about its benefits. The therapeutic claims made for *Aloe vera* range over a broad list of conditions e.g., arthritis, asthma, candida, chronic fatigue syndrome, digestive and bowel disorders, ulcers and skin problems including eczema, psoriasis, acne, frostbite and burns.[3] In this article, the botany and chemical constituents of *Aloe vera* are reviewed with emphasis on its therapeutic properties for burn wound. The pathophysiology of the burn wound is also described briefly for better understanding of how *Aloe vera* can take part in the treatment.

Pathophysiology of the thermal burn

1. Local and systemic responses[4]

The pathophysiological changes in the burn wound are characterized by effects caused by heat *per se* and superimposed on these is a pronounced acute inflammatory process. A sudden increase in body surface temperature results in prompt local responses by the blood vessels in the area in an attempt to dissipate heat by vasodilatation. A further increase in tissue temperature starts an inflammatory reaction caused by local release of inflammatory mediators and cascades of reactions then take place. The inflammatory mediators which control blood supply and microvascular permeability in the wound have been extensively studied and are largely understood (Table 1). Prostaglandins, thromboxanes and leukotrienes are produced through the arachidonic cascade (Fig. 1).

The inflammatory responses to injury, infection and antigen challenge with overproduction of chemical mediators, activation of leukocytes and endothelial cells and an alteration in circulating cytokines may all contribute to systemic effects. Thus in patients with major burns these effects are: increased susceptibility to infection, the systemic inflammatory response syndrome (SIRS), adult respiratory distress syndrome (ARDS) and multiple organ dysfunction syndrome (MODS), which may develop further into progressive organ failure and death.

2. Postburn changes[4]

Usually the burn wound initially has different depths in different regions. There are three degrees of burns, the first in which the epidermis only is damaged, the second where some dermal changes also occur but where epithelial regeneration is possible and the third where both epidermis and dermis are irreversibly damaged.[5]

Table 1. Major inflammatory mediators, which control blood supply and vascular permeability or modulate cell movement. The main sources are given (modified from Arturson G, 1996)

Mediator	Origin	Actions
Bradykinin	Kinin system (Kininogen)	Vasodilation Increased microvascular permeabilty Smooth muscle contraction Pain
Fibrinopeptides Fibrin split products	Coagulation system	Increased microvascular permeability PMNL and macrophage chemotaxis
C3a	Complement C3	Mast cell degranulation Smooth muscle contraction
C5a	Complement C5	Mast cell degranulation PMNL activation PMNL and macrophage chemotaxis Smooth muscle contraction Increased microvascular permeability
Substance P	Sensory nerve ending	Vasodilation Increased microvascular permeability
Histamine	Mast cells Basophils	Increased microvascular permeability Smooth muscle contraction Chemokinesis
5-Hydroxytryptamine (5HT=serotonin)	Platelets Mast cells	Increased microvascular permeability Smooth muscle contraction
Platelet activating factor (PAF)	PMNL Macrophages Basophils	Increased microvascular permeability Smooth muscle contraction PMNL activation
Prostaglandin E_2 (PGE_2)	Cyclooxygenase pathway	Vasodilation
Prostaglandin $F_{2\alpha}$ ($PGF_{2\alpha}$)	Cyclooxygenase pathway	Vasoconstriction
Prostacyclin (PGI_2)	Cyclooxygenase pathway	Vasodilation Antiaggregation
Thromboxane A_2 (TXA_2)	Cyclooxygenase pathway	Vasoconstriction Proaggregation
Leukotriene B_4 (LTB_4)	Lipoxygenase pathway	PMNL chemotaxis
Leukotriene D_4 (LTD_4)	Lipoxygenase pathway	Increased microvascular permeability Smooth muscle contraction

Often the wound is characteristically made up of several zones of tissue damage due to different heat transfer.[4] In the middle, usually the site of greatest heat transfer, irreversible skin death occurs, resulting in the ***zone of coagulation***. This zone is surrounded by the ***zone of stasis***, characterized by a pronounced inflammatory reaction. This potentially salvageable area could be converted to full destruction by infection or drying of the wound. Outermost is the ***zone of hyperemia***, which is the site of minimal cell involvement and early spontaneous recovery. Burn wound changes over time mainly in the zone of stasis can be discerned as follows:

1. A period of rapid ***local edema formation*** with a maximum at about 1-3 hr postburn due to vasodilataton, increased extravascular osmotic activity and increased microvascular permeability.

2. These changes are followed by heterogenous reductions in perfusion, the so called ***no reflow phenomenon*** leading to ***local tissue ischemia*** and further necrosis. The microcirculation is compromised to the worst extent at around 12-24 hr postburn.

3. A period of ***adhesion of endothelial cells, platelets and leukocytes***. This leads to leukocyte margination followed by extravasation and migration to the injured parenchymal cells and microorganisms. Platelets removed from the circulation contribute at different levels to hemostasis and local thrombosis.

4. A later phase of ***wound repair*** with high rates of wound perfusion to support wound metabolic requirements and maintain adequate defence against invasive burn wound infections.

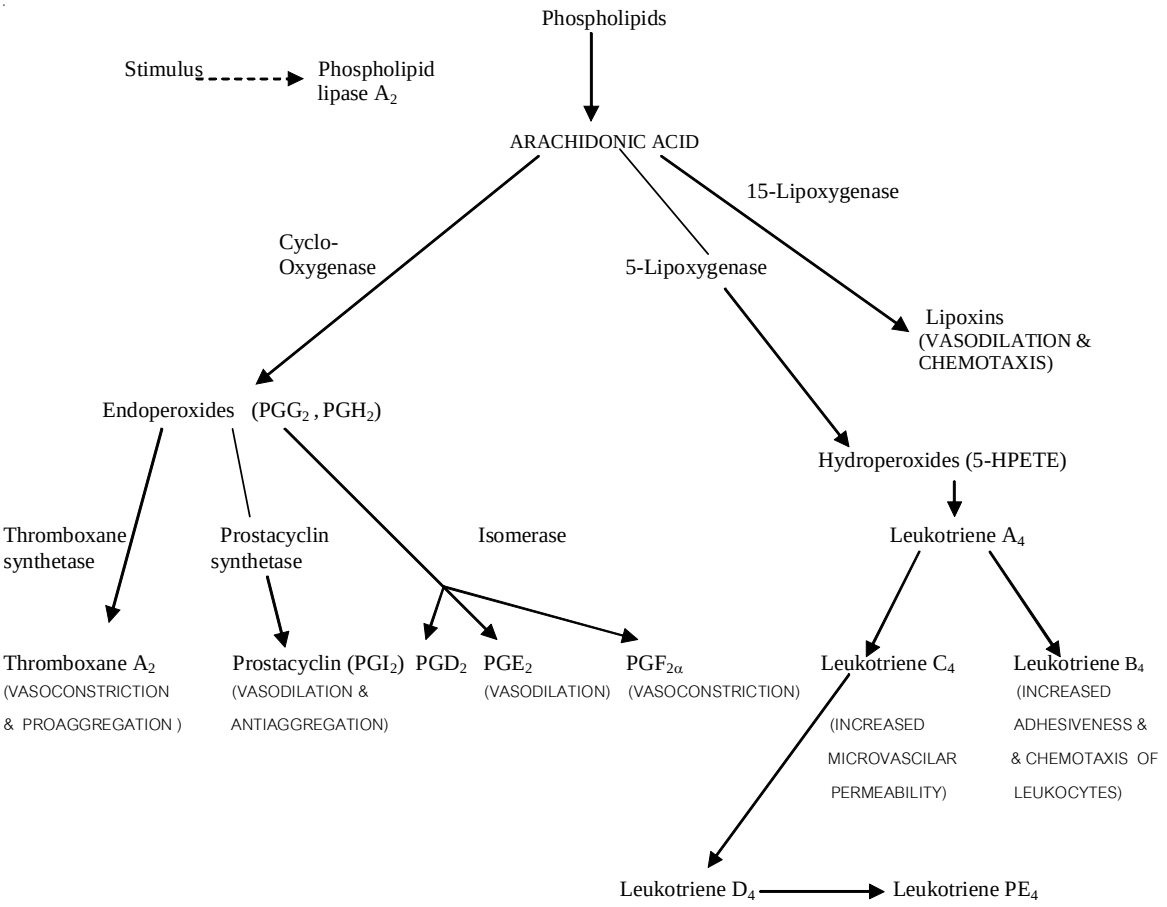

Fig. 1 The arachidonic acid cascade. The most frequently observed microcirculatory effects are shown in parentheses below the active metabolites (Arturson G, 1996)

5. ***Burn wound microbial colonization and infection***. Gram-positive bacteria in the depths of hair follicles and sweat glands may heavily colonize the wound within the first 48 hr postburn, especially if topical chemotherapy is not applied. The microorganisms present in the wounds of hospitalized patients change with time after injury. Usually gram-positive organisms (*Staphylococcus aureus, Streptococcus pyogenes*) during the first week postburn are superseded by gram- negative organisms (*Pseudomonas aeruginosa, Escherichia coli*) during the second week. Sometimes candida species (*Candida albicans*, aspergillus, phycomycetes) are detected later.

3. Immunological responses to burn injury[4,6]

Several T-cell functions seems to fail following a burn. It is established that burn injury induces more depression in the T-cell compartment than in the B-cell system, with particularly a reduction in the number of helper T cells. Most cytokine activities are chaotic and abnormal. The major cytokines shown to involved are interleukin-1 (IL-1), interleukin-2 (IL-2), interleukin-6 (IL-6), interleukin-8 (IL-8), tumor necrosis factor-a (TNF-a) and interferon-g (IFN-g). Other products of immune cells, like prostaglandins, oxygen free radicals and consequently lipid peroxides, are also found to be involved. Neutrophil function is ultimately abnormal.

The *Aloe Vera* Plant

Aloe vera belongs to the Liliaceal family, of which there are about 360 species. The scientific name is *Aloe vera* (Linn.) Burm. f. (synonym: *Aloe barbadensis* Miller). *Aloe vera* is a cactus-like plant that grows

readily in hot and dry climate. It is a short-stemmed succulent herb.The succulent leaves are crowded on the top of their stems, spreading, grayish green and glaucous; spotted when young, 20-50 cm long, 3-5 cm wide at the base, tapering gradually to the point tip, 1-2.5 cm thick, having edeges spiny and bitter latex inside. Flowers borne on the upper part of a slender stalk, 50-100 cm high. Forms of the species vary in sizes of leaves and color of flowers.[7]

The epidermis of the leaf has a thick cuticle, and beneath is zone of parenchyma which obtains pericyclic cells. The latex or yellow juice contains within the pericyclic cells. The central bulk of the leaf contains the colorless mucilaginous pulp, made up of large thin-walled mucilaginous cell containing the aloe gel itself.[8] The different properties of *Aloe vera* are ascribed to the inner, colorless leaf gel and to the yellow juice or exudate from the outer layer.[9]

Chemical Constituents

Several studies of chemical constituent analysis in the latex portion have found anthraquinone glycosides derivatives such as aloin, barbaloin, isobarbaloin, anthranol, aloe emodin, chrysophanic acid, 1,8- dihydroxy-anthraquinone.[10,11,12]

The fresh gel had been found to consist of 99.5% water and 0.5 % solid component.[13,14] Analysis of the solid components revealed that the largest number of active substances (97%) were in the mixed polysaccharides.[15] Besides, the solid components had been found to comprise glycoprotein, aloctin A, aloctin B; amino acids, vitamins, inorganic compounds, enzymes, uric acid, salicylic acid, cholesterol, triglycerides, steroids, etc. Table 2 summarizes its most important compositions.[3]

It was believed that a strong synergistic relationship existed between polysaccharides and other active substances in aloe such as amino acid and vitamins.[11,16,17,18] Certain amino acids and vitamins showed strong anti-inflammatory activity[19], suggesting that these substances might have a triggering effects on enzymes and polysaccharides activity needed for antiinflammation.[20] The healing properties of *Aloe vera* as well as the anti-inflammatory effects of *Aloe vera* had polysaccharides base as active ingredient, and also needed synergistic effects of their active substances.[11,14,17,18]

Therapeutic Properties of *Aloe Vera* on Thermal Burn

Since ancient times use of *Aloe vera* as a remedy had repeatedly come up in folklore, along with testimonials related to the therapeutic properties of the mucilage when applied to burned skin. Besides, it has also been used for the treatment of frostbite, dermatitis and ulcers. Virtually the use of *Aloe vera* as a medicinal plant for the treatment of thermal burns has been relied largely on historical/anecdotal evidence. For the last several decades, the scientists have begun seriously validating efficacy and exploring mechanisms of action of *Aloe vera*.

Table 2. Chemical Compositions of *Aloe vera* (modified from Vogler BK and Ernst, 1999)

Constituent	Identification
Anthraquinones	Aloin, barbaloin, isobarbaloin, anthranol, aloetic acid ester of cinnamic acid, aloe-emodin, emodin, chrysophanic acid, ethereal oil, resistannol
Inorganic compounds	Calcium, sodium, chlorine, manganese, zinc, chromium, potassium, sorbate, copper, magnesium,iron
Saccharides	Cellulose, glucose, mannose, L-rhamnose, aldopentose
Enzymes	Cyclooxygenase, oxidase, amylase, catalase, lipase, alkaline phosphatase, carboxypeptidase
Vitamins	B_1, B_2, B_6, choline, folic acid, C, α-tocopherol, β-carotene
Essential amino acids	Lysine, threonine, valine, leucine, isoleucine, methionine, phenylalanine
Nonessential amino acids	Histidine, arginine, hydroxyproline, aspartic acid, proline, glycine, alanine, tyrosine
Miscellaneous	Cholesterol, triglycerides, steroids, β-sitosterol, uric acid, gibberellin, lectin-like substance, lignins, arachidonic acid, salicylic acid

In 1943, aloe was shown to be successful in the treatment of thermal second-degree burns and radium burns.[21] Later in 1957, use of aloe gel against controlled thermal and radiation burns in laboratory animals and humans failed to demonstrate any healing properties of the gel.[22] In contrast, another study in 1959 gave a positive result.[23] Then in the early 1980s, precise experimental scald burns were compared by a variety of criteria and therapeutic benefits were recorded[12,24]. Similar positive results were also obtained from this type of test system in 1993.[25]

In 1988, the full-thickness thermal burns covering 3 percent of body surface area of guinea pigs by using hot metal plate were demonstrated to heal better with *Aloe vera* gel (30 days) than with silver sulfadiazine (47 days) and plain gauze occlusive dressing (50 days), but no vehicle control was used in this study.[26] The same technique was further elaborated in 1996 to produce first, second or third degree burns by precisely timed exposure to hot metal plate with positive results.[5] Recently,in 2000, *Aloe vera* was demonstrated to exhibit the actions of anti-inflammation and wound healing promotion when applied on a second degree burn in rats.[27] During the 1980s to 1990s, some clinical studies also showed that *Aloe vera* could control bacterial growth[28] and accelerate wound healing in burn patients.[29] However, in 1989, a negative result is offered from another study in experimental second-degree burns that aloe hindered the healing process.[30] The conflicting evidence may be explained by three possible factors: the purities of the gel (contamination of the latex or others into the gel portion), the fragility of the active ingredients after harvesting, and the varying location of wound.

Aloe vera is known to contain several pharmacologically active ingredients. Based on the available information from the peer-reviewed scientific literature, a list of mechanisms of action underlying therapeutic properties of *Aloe vera* on thermal burn can be compiled as follows:

1. Anti-inflammation

Inflammation is a tissue reaction by the body to injury and typically follows burn or other skin insults, which is characterized by swelling, pain, redness and heat as well as loss of function. It is thus a complex process and investigations into the therapeutic properties of *Aloe vera* gel should take account of its effects on these various symptoms and signs. In vivo demonstration using intravital fluorescent microscopic technique in second-degree burn model in rats indicated that the anti-inflammatory effects of *Aloe vera* have been characterized by the inhibition of the abnormalities of vascular diameter changes, vascular permeability and leukocyte adhesion. It is suggested that a combination of active ingredients in *Aloe vera* can restore the normal endothelial functions of cutaneous microcirculature after inflammatory responses to thermal injury.[27] Based on the existing data, the anti-inflammatory activities of *Aloe vera* can be explained by the following mechanisms.

1.1 Antithromboxane/antiprostaglandin activities

Many studies have revealed that *Aloe vera* has both antithromboxane and antiprostaglandin activities. The production of thromboxane A_2 (TXA_2) and thromboxane B_2 (TXB_2), along with PGF2 alpha in the burn wounds has been shown to decrease as a result of aloe therapy.[31,32] It has been suggested that unspecified substances in aloe gel inhibited arachidonic acid oxidation and thereby reduced inflammation.[33] In a much later study, it was found to reduce vasoconstriction and increase tissue survival or preserve tissue necrosis by actively inhibiting the localized production of thromboxanes.[25]

There are more than one active components in the aloe gel which may display antithromboxane and antiprostaglandin activities. ***Steroids*** are included as a component of aloe gel.[34] This explains the decreased amount of prostaglandins by decreasing release of arachidonic acid from phospholipid as a result of steroid action. In another test of surgical cuts in mice, it was shown that ***aloe vera sterols, including lupeol, campesterol***, ***beta-sitosterol*** had significant antii-nflammatory effects.[35] Of the three sterols, lupeol caused the greatest reduction in inflammation by 37.0%.

In addition, a glycoprotein component of the gel, ***aloctin A,*** was demonstrated to inhibit prostaglandin E2 production but over a relatively long incubation time.[36,37] ***Salicylic acid*** present in *Aloe vera* gel is known to inhibit the production of prostaglandin and thromboxane from arachidonic acid by inhibiting cyclooxygenase.[38] However, *Aloe vera* also provides cyclooxygenase enzyme which can counteract these antithromboxane and antiprostaglandin activities of *Aloe vera* itself by conversion of arachidonic acid into different prostanoids.[39]

1.2 Antibradykinin activity

Bradykinin was both a vasodilator and potent pain producing agent at the site of acute inflammation.

An in vivo study found that lyophilized powder aloe contained ***bradykininase***. This result was confirmed that the bradykininase activity of aloe vera could hydrolyze bradykinin and angiotensin I to convert into angiotensin II, resulting in suppressing vasodilation and pain.[40]

Besides, the ***carboxypeptidase*** was reported to be enzyme in *Aloe vera* gel that could hydrolyze bradykinin and angiotensin I in vitro.[41] The carboxypeptidase from aloe could inhibit bradykinin in vivo, yet decreasing pain at the site of acute inflammation.[42] Another antibradykinin active material in *Aloe vera* which was tested on isolated guinea pig ileum in vitro, was estimated to be a ***glycoprotein***.[15] It was suggested that aloe glycoprotein had the presence of carboxypeptidase N-and P-like enzymes with proteolytic activity. These results might provide a pharmaceutical basis for the anti-inflammatory action of *Aloe vera*.[43]

1.3 Antihistamine activity

Magnesium lactate in *Aloe vera* was known to inhibit the conversion of histidine to histamine while ***barbaloin and aloctin*** could inhibit histamine release from mast cells.[44] The antihistamine activity then results in decreasing vasodilatation, inflammation and pruritus.

1.4 Anti-inflammatory cytokine activity

Very recently, a study using second degree burn wound model in rats has revealed that treating burn wound with *Aloe vera* could prevent the elevation of serum TNF-a and IL-6, the active inflammatory cytokines released following thermal injury, at 3, 7 and 14 days postburn.[45]

1.5 Other activities

The anti-inflammatory action of *Aloe vera* was also shown to be attributable to some active components through other activities in addition to those described above. Both fresh and commercial aloe preperations were found to contain high levels of ***lectin-like substances***.[46] Lectins are hemagglutinating proteins that bind to glycoproteins and decrease inflammation. ***Certain amino acids, vitamins, and RNA*** in 50% ethanol supernatant aloe were shown to have anti-inflammatory activity and it could normalize the acute vascular response.[47,48] ***Gibberellin*** acts as a growth hormone and also decreases inflammation.[49] A new agent as ***cinnamoyl-C-glucosylchromone*** containing in *Aloe vera* was demonstrated to exhibit topical anti-inflammatory activity using croton oil-induced ear inflammmation model.[50]

2. Antimicrobials

Antibacterial activity of aloe can be confirmed both in vitro and in vivo. Aloe gel is bacteriostatic or bactericidal against a variety of common wound-infecting bacteria in vitro: *Staphylococcus aureus, Streptococcus pyogenes, Serratia marcescens, Klebsiella pneumoniae, Pseudomonas aeruginosa, Escherichia coli, Salmonella typhosa* and *Mycobacterium tuberculosis.*[24,51]

A report of clinical cases suggested that the gel was bactericidal towards *Pseudomonas aeruginosa*.[23] In a clinical trial, aloe gel was used to treat burns and it could control bacterial growth which was otherwise present in the untreated controls.[28] Similar results were achieved in experimental trials.[26,52] However, some studies failed to demonstrate antibacterial activity, especially in deep wounds which became so heavily infected that death eventually ensued.[5]

There are at least two explanations for the antimicrobial activity of *Aloe vera*. First, some ingredients in *Aloe vera* were demonstrated to act as antimicrobials. ***Acemannan*** prevented adhesion of *Pseudomonas aeruginosa* to human lung epithelial cells in monolayer culture[53] and also blocked the reproduction of Herpes[54] and the AIDS virus.[55] Second, it may be that antibacterial factors are released by the healing tissues in responses to aloe treatment.[56]

3. Wound healing promotion

A variety of studies have shown wound healing properties of *Aloe vera*.[26,27,29,57] Using full thickness burn wound model to compare the effect of *Aloe vera* extract, silver sulfadiazine, salicylic acid cream and untreatment, the average time to complete healing in the *Aloe vera*-treated group (30 days) was significantly less than the untreated group (50 days). In addition, wound bacterial counts were effectively decreased by silver sulfadiazine and by *Aloe vera* extract.[26] Partial thickness burns in twenty-seven patients were observed to heal more rapidly when treated with aloe gel, compared with vaseline. The average time of healing in the aloe gel area was 11.89 days and 18.19 days for the vaseline gauze treated wound. In histologic studies, it showed

early epithelialization in the treated aloe gel area, both growth of epithelial cells and organization of fibrovascular and collagen tissue being stimulated.[29] Wound healing effect of *Aloe vera* on induced second degree burn wounds in rats was also demonstrated during seventh and fourteenth days after burn.[27] Wound healing properties of *Aloe vera* can be explained by the following mechanisms:

3.1 Moisturizing effect

One of the first explanations of its efficacy was its high water content which kept the wound moist and increased epithelial cell migration.[1,58] In addition, *Aloe vera* may act in an occlusive manner to keep the wound moist and to prevent water loss from the wound to increase mitosis, proliferation, and migration of epidermal cells.[16]

3.2 Increased oxygen access

It was also suggested that one of the factors enhanced by aloe gel was increased oxygen access as a result of increased blood supply to injured areas.[59]

3.3 Stimulation of fibroblast and collagen synthesis

It was found that *Aloe vera* stimulated fibroblasts to increase in number in a dose-response fashion.[35] In a trial using topical application of Aloe vera-derived allantoin gel for respiratory tract disorders, stimulation of fibroblast activity and collagen proliferation was demonstrated.[60] ***Amino acid, ascorbic acid, zinc, lignins, and saponins***, which were discovered in *Aloe vera*, increased the synthesis of collagen and counterbalanced collagen breakdown with subsequent increase on wound tensile strength.[20,61] The fresh *Aloe vera* leaves had ***lectin-like compounds*** which enhanced the growth of normal human cell in tissue culture.[62] The growth factors including ***giberellin, auxins and mannose-6-phosphate***, which were identified in *Aloe vera*, were effective in promoting wound healing.[35,63] A ***glycoprotein (Pg 21-2b)*** with cell-proliferation promoting activity has been reported from *Aloe vera* gel.[64]

3.4 Promotion of angiogenesis

Angiogenesis, the growth of new blood capillaries, is a necessary part of tissue regeneration and vascularity of burn tissue of a guinea pig was shown to be reestablished by topical application of aloe gel.[65] This may be the effects of ***a low molecular weight component and other ingredients*** resided in methanol-soluble fraction of the gel.[66]

3.5 Activation of macrophage

Macrophage play a considerable part in controlling microorganisms and it was shown that young active macrophages accelerated the rate of wound healing. Activation of macrophages by ***acemannan,*** an aloe gel polysaccharide, was claimed.[67]

4. Biological / immunological modulation

Characterization studies of *Aloe vera* have identified two distinct components of the plant extract. ***Glycoproteins*** inhibits the production of free oxygen radicals by polymorphonuclear leukocytes (inhibitory system). A polysaccharide called ***acemannan*** stimulates antibody production and the synthesis of a variety of immunologically active interleukins (stimulatory system).[16,68] The interaction between the stimulatory and inhibitory systems is referred to as biological modulation. It is a means by which the cells, under the influence of *Aloe vera*, adapts to mechanically or immunologically induced trauma.

All living systems have the ability to receive stimuli and select an appropriate response. This selection process is referred to as modulation. *Aloe vera* has an inhibitory system (anti-inflammatory activity) and a stimulatory system (wound healing activity) that may act as a modulator of wound healing and inflammation. The modulatory systems in *Aloe vera* represent the interactions between many components, which involves ***enzymes, chemical reactions, and growth factors***.[16]

Conclusion

There is much scientific evidence in animal models to suggest that topical application of *Aloe vera* seems to be useful as a treatment for thermal burns, especially superficial and partial thickness burns. The efficacy in humans also exists in some trials. The mechanisms that may take into account of its actions include anti-inflammation, antimicrobials, wound healing promotion, and, interestingly, biological/immunological modulation. However, there is still a little conflicting evidence which may be explained by three possible factors: the purities of the gel (contamination of the latex or others into the gel portion), the fragility of the active ingredi-

ents after harvesting, and the varying locations of the wound. Thus, continuing research is needed to provide more scientific knowledge for its therapeutic benefit in humans. The conclusion is drawn that well-controlled clinical trials should be implemented with the use of a standardized preparation of *Aloe vera* to define its efficacy in humans. In the future, *Aloe vera* might be a valuable therapeutic agent for burn-wound patients.

References

1. Morton JF. Folk uses and commercial exploitation of Aloe leaf pulp. Econ Bot 961; 15: 311-9.
2. Crosswhite FS, Crosswhite CD. *Aloe vera*, plant symbolism and the threshing floor. Desert plants 1984; 6:43-59.
3. Vogler BK, Ernst E. Aloe vera : a systemic review of its clinical effectiveness. Br J Gen Prac 1999; 49: 823-8.
4. Arturson G. Pathophysiology of the burn wound and pharmacological treatment. The Rudi Hermans Lecture, 1995. Burns 1996; 22; 255-74.
5. Bunyapraphatsara N, Jirakulcaiwong S, Thrirawarapan S, Manonukul J. The efficacy of *Aloe vera* cream in the treatment of first, second and third degree burns in mice. Phytomed 1996; 2: 247-51.
6. Sparkes BG. Immunological responses to thermal injury. Burns 1997; 23: 106-13.
7. Grindlay D, Reynolds T. The *Aloe vera* phenomenon: A review of the properties and modern uses of the leaf parenchyma gel. J Ethnopharmacol 1986; 16: 117-51.
8. Klein AD, Penneys NS. *Aloe vera*. J Am Acad Dermatol 1988; 18: 714-20.
9. Reynolds T, Dweck AC. *Aloe vera* leaf gel : a review update. J Ethno- pharmacol 1999 ; 68 : 3-37.
10. Hirata T, Suga T. Biologically active constituents of leaves and roots of *Aloe arborescens* var. *natalensis*. Zeitschrift Fur Naturforshung 1977; 32: 731-4.
11. Henry R. An updated review of *Aloe vera*. Cosmetics and Toiletries 1979; 94: 42-50.
12. Robson MC, Heggers JP, Hagstrom WJ. Myth, magic, withchcraft or fact? *Aloe vera* revisited. J Burn Care Rehabil 1982; 3: 157-63.
13. Gjerstad G. Chemical studies of *Aloe vera* juice-I: Amino acid analysis. Advanc Front Plant Sci 1971; 28: 311-5.
14. Mckeown E. *Aloe vera*: The quest for the curative missing link. D&CL 1983; 30-4.
15. Yagi A, Harada N, Yamada H, Iwadare S, Nishioka I. Antibradykinin active material in *Aloe saponaria*. J Pharma Sci 1982; 71: 1172-4.
16. Davis RH, Parker WL, Samson RT, Murdoch DP. Isolation of a stimulatory system in aloe extract. JAPMA 1991; 81: 473-8.
17. Leung AY. *Aloe vera* in cosmetics. Excelsa 1978; 8: 65-68.
18. Waller GR, Mangiafic S, Ritchey CR. A chemical investigation of *Aloe barbadensis* Miller. Proceedings of the Oklahoma Academy of Science 1978; 58: 69-76.
19. Hanley D, Solomon W, Saffran. The evaluation of natural substances in the treatment of adjuvant arthritis. JAPMA 1982; 72: 275.
20. Coats BC, A modern study of *Aloe vera*. The silent healer, Garlan, TX, 1979.
21. Tchou MT. *Aloe vera* (jelly leeks). Arch Dermatol Syphilol. 1943; 47: 249.
22. Ashley FL, O ' Loughlin BJ, Peterson R, et al. The use of *Aloe vera* in the treatment of thermal and radiation burns in laboratory animals and humans. Plast Reconstr Surg . 1957; 20: 383-96.
23. Rovatti B, Brennan RJ. Experimental thermal burns. Industrial Med Surg 1959; 28: 364-8.
24. Cera LM, Heggers JP, Robson MC. The therapeutic efficacy of *Aloe vera* cream (Dermaide Aloe) in thermal injuries: Two case reports. J Am Animal Hosp Assoc1980; 16: 768-72.
25. Heggers JP, Pelley RP, Robson MC. Beneficial effects of aloe in wound healing. J Ethnopharmacol 1993; 7: 48-52.
26. Rodriguez- Bigas M, Cruz NI Suarez A. Comparative evaluation of *Aloe vera* in the management of burn wounds in guinea pigs. Plast Reconstr Surg 1988; 81: 386-9.
27. Somboonwong J, Jariyapongskul A, Thanamittramanee S, Patumraj S. Therapeutic effects of *Aloe vera* on cutaneous microcirculation and wound healing in second degree burn model in rats. J Med Assoc Thai 2000; 83: 417-24.
28. Heck E, Head M, Nowak D, Helm P, Baxter C. *Aloe vera* (gel) cream as a topical treatment for outpatient burns. Burns 1981: 7: 291-4.
29. Visuthikosol V, Sukwanarat Y, Chowchen B, Sriuratana S, Boonpuknavig V. Effect of *Aloe vera* gel to healing of burn wound: Clinical and histologic study. J Med Assoc Thai 1995; 75: 403-8.
30. Kaufman T, Newman AR, Wexler MR. *Aloe vera* and burn wound healing. Plast Reconstr Surg 1989; 83: 1075-6.
31. Heggers JP, Loy G . Robson MC. Histological demonstration of prostaglandins and thromboxanes in burned tissue . J Surg Res 1979; 28: 110-7.
32. Robson MC, DelBeccaro EJ, Heggers JP. Increasing dermal perfusion after burning by decreasing thromboxane production. Plast Reconstr Surg 1980; 20: 722-5.

33. Penneys NS. Inhibition of arachidonic acid oxidation in vitro by vehicle components. Biochem Pharmacol 1982; 62: 59-61.
34. El – Zawahry ME, Hegazy MR, Helal M. Use of aloe in treating leg ulcers and dermatoses. Inter J Dermatol 1973; 12: 68-73.
35. Davis RH, DiDonato JI, Johnson RWS, Stewart CB. *Aloe vera*, hydrocortisone, and sterol influence on wound tensile strength and antiinflammation. JAPMA 1994; 84: 614-21.
36. Saito H, Ishiguro T, Imanishi K, Suzuki I. Pharmacological studies on a plant lectin aloctin A II. Inhibitory effect of aloctin A on experimental models of inflammation in rats. Jap J Pharmacol 1982; 32: 139-42.
37. Ohuchi K, Watanabe M, Takahashi E, et al. Lectins modulate prostaglandin E_2 production by rat peritoneal macrophages. Agents and Actions 1984; 15: 419-23.
38. Davis RH, Kabbani J, Maro J. *Aloe vera* and inflammation. Proceeding of the Pennsylvania Academy of Science 1986: 60-7.
39. Afzal M, Ali M, Hassan RAH, Sweedan N, Dhami MSI. Identification of some prostanoids in *Aloe vera* extract. Planta Med 1991; 57: 38-40.
40. Rubel BL. Possible mechanism of the healing actions of aloe gel. Cosmetics and Toiletries 1983; 98: 109-14.
41. Fujita K, Ito S, Teradaira R, Beppu H. Properties of a carboxypeptidase from aloe. Biochem Pharmacol 1979; 28: 1261-2.
42. Klein AD, Penneys NS. *Aloe vera*. J Am Acad Dermatol 1988; 18: 714-20.
43. Yagi A, Nishimura H, Shida I, Nishioka I. Structure determination of polysaccharides in *Aloe arbrescens var natalensis*. Planta Medica 1986; 52: 213-7.
44. Nakagomi K, Oka S, Tomizuka N, Yamamoto M, Masui T, Nakazawa H. A novel biological activity in aloe components. Effects on mast cell dlegranulation and platelet aggregation. abst. 1984; 20: 29.
45. Duansak D, Somboonwong J, Patumraj S. Effects of *Aloe vera* on leukocyte adhesion and TNF-a and IL-6 levels in burn wounded rats. Clin Hemorheol Microcirculation 2003; 29: 239-46.
46. Danof IE, McAnalley W. Stabilised *Aloe vera* : effect on human skin cells. Drug and Cosmetic Industry 1983; 33; 52,24: 105-106.
47. Forst MB, Davis RH. Effects of tryptophane, anthranilic acid and ribonucleic acid on adjuvant arthritis JAPMA 1979; 69: 643.
48. Davis RH, Rosenthal KY, Cesario LR, et al, Vitamin C influence on localized adjuvant arthritis. JAPMA 1990; 80: 14.
49. Davis RH, Maro NP. *Aloe vera* and gibberellin anti-inflammatory activity in diabetics. JAPMA 1989; 79: 24.
50. Hutter JA, Salman M, Stavinoha WB, Satsangi N, Williams RF, Streeper RT, et al. Antiinflammatory C-glucosyl chromone from *Aloe barbadensis*. J Nat Prod 1996; 59: 541-3.
51. Lorenzetti L, Salisbury R, Beal J, Baldwin J. Bacteriostatic property of *Aloe vera*. J Pharmacol Sci 1964; 53: 1287.
52. Kivett WF. *Aloe vera* for burns. Plast Reconstr Surg 1989; 83: 195.
53. Azghani AO, Williams I, Holiday DB, Johnson AR. A beta – linked mannan inhibits adherence of *Pseudomonas aeruginosa* to human lung epithelial cells. Glycobiol 1995; 5: 39-44.
54. Kahlon J, Kemp MCX, Yawei N, Carpenter RH, Shannon WM, Mc Analley BH. In vitro evaluation of the synergistic antiviral effects of acemannan in combination with azidothymidine and acyclovir. Mol Biother 1991 b; 3: 214-23.
55. Kemp MC, Kahlon JB, Chinnah AD, Carpenter RH, McAnalley BH, McDaniel HR, Shannon WM. In vitro evaluation of the antiviral effects of acemannan on the replication and pathogenesis of HIV-1 and other enveloped viruses: modification of the processing of glycoprotein precursors. Antiviral Res 1990; 13: 83.
56. Heggers JP, Kucukcelibi A, Stabenau CJ, KOF, Broemeling LD, Robson MC, Winters WD. Wound healing effects of Aloe gel and other topical antibacterial agents on rat skin . Phytother Res 1995; 9: 455-7.
57. Davis RH, Leitner MG, Russo JM. *Aloe vera* : A natural approach for treating wounds, edema and pain in diabetes. JAPMA 1988; 78: 60-8.
58. Erazo S, Lemus I, Garcia R. Evaluation of the humectant properties of *Aloe perryi* Baker. Plantes Med Phytother 1985; 19: 240-7.
59. Davis RH, Rosenthal KY, Cesario LR, Rouw GA, Processed *Aloe vera* administered topically inhibits inflammation. JAPMA 1989b; 79: 395-7.
60. Thompson JE. Topical use of *Aloe vera* derived allantoin gel in otolaryngology. Ear Nose Throat J 1991; 70,56,119.
61. Engel E, Erlick N, Davis R. Diabetes mellitus: impaired wound healing from zinc deficiency. JAPMA 1981; 71: 536.
62. Winters W, Benavides R, Clouse WJ. Effect of aloe extracts on human normal and tumor cells *in vitro*. Econ Bot 1981; 35: 89.
63. Morgan DO, Edman JC, Standring DN, et al. Insulin-like growth factor II receptor as a multifunctional binding protein. Nature 1987; 329: 301.
64. Yagi A, Egusa T, Arase M, Tanabe M,Tsuji H. Isolation and characterization of the glycoprotein fraction with a proliferation-promoting activity on human and hamster cells *in vitro* from *Aloe vera* gel. Planta Medica 1997; 63: 18-21.
65. Heggers JP, Pelley RP, Hill DP, Stabenau CJ, Winters WD. Wound healing with aloe substances. Academic/Industry

Joint Conference 1992; 41.
66. Lee MJ, Lee OH, Yoon SH, Lee SK, Chung MH, Park YI et al., *In vitro* angiogenic activity of *Aloe vera* on calf pulmonary artery endothelial (CPAE) cells. Arch Pharmacol Res 1998; 21: 260-5.
67. Maxwell B, Chinnah H, Tizard I. Activated macrophages accelerate wound healing in aged rats. Wound Repair and Regeneration 1996; 4: 165.
68. Merrian EA, Camplsell BD, Flood LP, Welsh CJR, McDaniel HR, Busbee DL. Enhancement of immune function in rodents using a complex plant carbohydrate which stimulates macrophage secretion of immunoreactive cytokines. Advances in Anti-Aging Medicine 1996; 1: 181-203.

ประสิทธิภาพและคุณสมบัติของว่านหางจระเข้ชนิดทาในการรักษาแผลไหม้จากความร้อน

จุไรพร สมบุญวงค์, นภัสนันท์ เดือนศักดิ์

ว่านหางจระเข้ เป็นพืชสมุนไพรที่รู้จักกันอย่างแพร่หลายมาตั้งแต่โบราณ สำหรับรักษาโรคต่าง ๆ รวมทั้งแผลไหม้ มีหลักฐานมากมายที่รายงานถึงประสิทธิภาพของว่านหางจระเข้เมื่อใช้ทารักษาแผลไหม้ โดยออกฤทธิ์ทางเภสัชวิทยาหลายประการ บทความนี้เป็นการทบทวนวารสารที่มีเนื้อหาครอบคลุมเกี่ยวกับพยาธิสรีรวิทยาของแผลไหม้ ลักษณะทางพฤกษศาสตร์ และ ส่วนประกอบทางเคมีของว่านหางจระเข้ รวมทั้งคุณสมบัติของว่านหางจระเข้ในการรักษาแผลไหม้ซึ่งประกอบด้วย ฤทธิ์ต้านการอักเสบ ฤทธิ์ต้านจุลชีพ การส่งเสริมการสมานแผล และการปรับสมดุลทางชีววิทยาและระบบภูมิคุ้มกัน