

Aloe Ferox

The healing properties of aloe have been known for millennia. The use of aloe was discovered on a Mesopotamian clay tablet (ca. 2100 BC). Aloes were listed in the Ebers papyrus (ca. 1500 BC) as an established cathartic.

Legend has it that aloe was an important part of the beauty regimen of the Egyptian queens, Nefertiti and Cleopatra. The Greek physician Dioscorides, while accompanying Nero's army, mentioned aloe in his writing (ca 100 AD). Alexander the Great (356–323 BC) was persuaded by Aristotle to capture the island of Socrota in the Indian Ocean to secure its aloe supplies to treat his wounded soldiers (Bruce 1975).

Numerous Aloe species have been used medicinally but only Aloe ferox, Aloe perryi and Aloe vera have demonstrated any commercial importance (Grindlay 1986). Scientific literature now documents various medical applications.

Aloe gel has demonstrated anti-inflammatory (Vázquez 1996, Bautista 2004), wound healing (Davis 1989, 1994, Heggors 1996), anti-tumour (Kim 1999, Pecere 2000), antiviral (McDaniels 1990a,b), anti-microbial (Wang 1998) and anti-diabetic (Reynolds 1999) activity.

It has also shown immune stimulating (Zhang 1996, Strickland 2001) and cholesterol lowering activity (Tizard 1989).

The active constituent in the aloe exudate (bitter) is the anthrones. They are degraded in the colon by bacteria to aloe-emodin, which function as a stimulant laxative (Blumenthal, 1998).

Studies have also demonstrated aloe-emodin to be antiviral (Sydiskis 1991), an antioxidant (Yen, 2000), effective for liver cancer prevention (Kuo 2002) and inhibits neuroectodermal tumor cell growth (Pecere 2000).

Anti-inflammatory

Inflammation is a non-specific immune response by the body to any type of injury. It is characterized by redness, heat, swelling and pain. According to Clayton (1993) the steps in inflammation are:

1. vasodilation that reduces blood pressure and increases blood flow (causing redness and heat)
2. followed by swelling due to an excessive amount of tissue fluid and
3. pain.

Vázquez (1996) demonstrated the anti-inflammatory effect of **aloe ferox** gel. It inhibited prostaglandin E2 production from arachidonic acid. While Yagi (1982) showed that the glycoprotein of aloe gel cleaved the bonds of the bradykinin molecule reducing pain and inflammation. In a later study (Bautista 2004) the antibradykinin effect was associated with the inhibition of prostaglandin synthesis.

Inflammation is also involved in conditions such as arthritis. Rheumatoid arthritis closely resembles adjuvant arthritis in rats and was studied by Davis (1992). According to this experiment aloe was injected and decreased inflammation (50%) and stimulated fibroblast growth repair.

Hanley (1982) showed when rat paws were injected with **Aloe ferox** it decreased inflammation (48%) and inhibited the immune response (72%). A subsequent study (Davis 1985) showed that when **Aloe ferox** was applied topically in a hydrophilic cream it reduced inflammation (39%) and subsequent arthritis (45%).

It has also been found that aloe has analgesic properties that can be ascribed to the presence of salicylates, which has an aspirin like effect (Shelton 1991).

Wound healing

A wound to the skin may pierce two layers, the dermis and epidermis. Healing follows the following steps (Reynolds 1999) by:

1. temporary repair is effected by fibrin clot to fill the gap which is invaded by cells that produce the inflammatory response and carry out the permanent repair.
2. The epidermis is repaired in 3 phases: (Davis 1994)
 - o fibroblasts migrate to the wound site causing granulation tissue to fill the gap,
 - o they proliferate and mature to produce collagen, elastin and proteoglycans.
 - o Proteoglycans form the basis in which collagen and other connective fibres are embedded.
3. It is essential to avoid microbial infection, as this will retard wound healing.

Wounds treated with **aloe ferox** showed rapid granulation and increased oxygen supply as a result of the increased blood flow (Davis 1989). The skin punch wounds healed more rapidly. The aloe gel reduced wound diameter, seemed to reduce scarring and inhibited acute inflammation.

In another study, (Heggors 1996) stimulation of fibroblast activity and collagen proliferation was demonstrated. **Aloe ferox** also expedites wound contraction and enhanced wound breaking strength.

Choi (2001) isolated a glycoprotein from Aloe that stimulated the formation of epidermal tissue. It also enhanced wound healing with significant cell proliferation and migration.

In the treatment of burn wound Heggors (1993) showed that the **Aloe Ferox** gel penetrated the tissue, relieve pain, reduce inflammation and increase blood supply by inhibiting the synthesis of thromboxane A₂, a potent vasoconstrictor.

A recent study (Barrantes 2003) demonstrated aloe gel enriched with aloins (bitter) to inhibit collagenase and metalloproteases activity, which can degrade collagen connective tissue when unchecked. This activity supports the use of **aloe ferox** in the treatment of chronic ulcers, burns and wounds.

Immune modulation

Research on immune stimulation has indicated that acemannan, a polysaccharide within **aloe ferox**, stimulated macrophage cytokine production and killer T cells (Zhang 1996).

Chronic exposure to UV radiation causes sunburn, premature aging of the skin and genetic mutations leading to skin cancer. UV radiation causes systemic suppression of immune responses. Strickland (2001) showed that the gel prevented systemic suppression of T cell mediated immune response and the production of IL-10. The aloe polysaccharides are immunostimulants by interfering with the activation of suppressor mechanisms.

Acemannan used for HIV-1+ patients showed a significant increase in the number of circulating monocytes and macrophages (McDaniels 1990a). In a pilot study treating HIV infected people acemannan increased the number of white blood cell and improved symptoms (McDaniels 1990b).

Gastrointestinal functions

The **aloe ferox** juice has been used as a tonic in a series of trials (Bland 1985) on human patients. It indicated a tonic effect on the intestinal tract with:

1. a reduction in pH;
2. a reduction in bowel transit time;
3. intestinal bacterial flora benefited with a reduction in yeast;
4. bowel putrefaction was reduced and
5. protein digestion and absorption was improved.

Yamamoto (1973) showed that a component of **Aloe ferox** suppresses ulcer growth and L-histidine decarboxylase in rats. Recently the gastropreventative of aloe was demonstrated by inhibiting gastric acid

secretion, which makes it suitable for peptic ulcer treatment (Yusuf 2004).

When **aloe ferox** juice is given orally to animals, mannans have been shown to lower cholesterol by inhibiting cholesterol absorption (Tizard 1986). In a small trial with monkeys it was found that **aloe ferox** juice lowered total cholesterol by 61% with a proportionate rise in HDL (Dixit, 1983).

Aloe ferox juice has been used with success to lower blood sugar and triglyceride levels. Diabetic patients that failed to respond to other medication responded to aloe treatment (Reynolds, 1999). It has been demonstrated that both the **aloe ferox** exudate (bitter) and gel decreased blood glucose levels in mice. Similarly it has been found that both compounds have a protective effect against hepatotoxic liver injury (Can 2004).

The cathartic and laxative action of **aloe ferox** bitter is well established. Its primary effect is caused by its influence on the motility of the colon. This results in an accelerated intestinal passage and a reduction in liquid absorption increasing water content in the faeces (Blumenthal 1998).

In addition to the purgative effect the anthraquinone (bitter) substances stimulate the flow of gastric juices thus improving digestion. Soeda (1964) found that fractions from **Aloe ferox** gave a prophylactic effect. While in a subsequent study, Soeda (1966) found the **aloe ferox** juice to have inhibitory action against some bacteria and fungi, particularly *Pseudomonas aeruginosa* and *Proteus vulgaris*.

Anti-cancer activity

An early report by Soeda (1969) reported anti-tumour activity of **Aloe ferox**. Both plant fractions have been shown to inhibit tumour growth. Aloe-emodin has shown mutagenesis inhibition as well as the glycoproteins (lectins) and polysaccharides from the gel.

Kuo (2002) has demonstrated that aloe-emodin induced apoptosis (cell disintegration) and acted as an effective anticancer effect in human liver cancer. Similarly, Pecere (2000) found that aloe-emodin did not inhibit fibroblast proliferation while selectively inhibiting human neuroectodermal tumour cells.

A purified polysaccharide indicated anticarcinogenic effects by inhibiting the uptake of B[a]P and subsequently binding to cellular DNA. It also had no cytotoxic effect (Kim 1999). Strickland (2001) demonstrated the polysaccharides efficacy to prevent non-melanoma skin cancers by preventing T cell immune suppression.

Anti-microbial

Reynolds (1999) has reviewed the antimicrobial activity of **aloe ferox**.

1. Antibacterial:

- The **aloe ferox** gel and bitter is bactericidal against: a variety of common wound infecting bacteria: *Streptococcus pyogenes*, *Serratia marcescens*, *Klebsiella pneumonia*, *Staphylococcus aureus*, *E. coli*, *Mycobacterium tuberculosis*, *Pseudomonas aeruginosa* and *Corynebacterium xerose*.
- The gel is effective against: *Streptococcus faecalis* responsible for urinary infection. Ferro (2003) showed effective growth inhibition of *Shigella flexneri* and *Streptococcus pyogenes* responsible for gastroenteritis.
- Aloe-emodin in bitter has been shown to inhibit: growth of *Helicobacter pylori*, which is responsible for peptic ulcers (Wang 1998). *Citrobacter*, *Enterobacter aerogenes*, *Serratia* and *Klebsiella* that cause gastroenteritis. *Proteus vulgaris* an opportunistic pathogen of the urinary tract and *Salmonella paratyphi* causing fever.

2. Antiviral:

- **Aloe ferox** gel has been proven to be virucidal to: HIV-1+ patients showing increased numbers of white blood cells and improvement in symptoms (McDaniels 1990b). Herpes simplex infection with significant faster healing time and higher number of healed lesions compared to the control (Syed 1997).

- **Aloe ferox** bitter was virucidal:
by disrupting the coating of the herpes and influenza virus (Sydiskis 1991).

3. Antifungal:

- **Aloe ferox** gel is shown to be fungicidal to:
Candida albicans responsible for yeast infections of the mucous membranes.
Trichophyton spp. by **Aloe ferox** juice (Soeda 1966) responsible for infectionssuch as athlete's foot and candidiasis (thrush).

Skin

The skin is composed of polypeptide chains that form aggregates of collagen fibrils, which influences the swelling and water uptake by the skin. The diffusion of water through the skin is limited and controlled by the stratum corneum (skin surface) that is in equilibrium with the atmosphere and underlying tissue.

Since aloe is approximately 99% water it penetrates through the surface of the skin (stratum corneum) to the vascular dermal area thus hydrating the skin. Concurrently, the gel forms a cover to prevent the escape of moisture in the skin.

Aloe ferox gel increases the penetration of the skin by water hydration, occlusiveness and by increasing compound solubility. Subsequently, Davis (1991) has demonstrated that **aloe ferox** gel enhanced the penetration of hydrocortisone and adds to its biological activity.

Concomitantly, **aloe ferox** gel increased oxygen supply as a result of increased blood flow (Davis 1989) and stimulates fibroblast activity and collagen proliferation (Thompson 1991) essential for skin tissue regeneration. Subsequently aloe gel it is used extensively in cosmetics.

Aloe ferox gel reduces photo aging by restoring the activity of epidermal cells reduced by UV exposure. The gel increase soluble collagen levels and biosynthesis possibly through macrophage stimulation (Lindblad 1994)

In a large clinical trial Syed (1996) studied the effect of an **Aloe ferox** cream on psoriasis vulgaris. They found that the aloe cream cured 83.3% of the patients compared to 6.6% of the placebo group with a concomitant clearing of the psoriatic plaques. There were no adverse drug reactions or side effects.

A study by Vardy (1999) has demonstrated the effectiveness of an aloe lotion for treating seborrheic dermatitis (excessive excretion of sebaceous glands, dandruff) when applied on the skin twice a day.
