184-0

Med. Sci. Res., 1987; **15**, 235

Biological activity of *Aloe vera*

Robert H. Davis, Mark G. Leitner, Joseph M. Russo and Nicholas P. Maro

Pennsylvania College of Podiatric Medicine, Department of Physiological Sciences, 8th at Race Street, Philadelphia, Pennsylvania 19107, USA

With the advent of cortisone as a therapeutic agent, a great deal of attention focused on the making of powerful synthetic drugs and steroids to combat inflammation and arthritis. While the benefit of these agents was observed, unwanted responses were also recorded. As a result, a search for more natural substances was begun [1, 2]. Our laboratory has shown that *Aloe vera* (*Aloe barbadenses*) was effective against adjuvant induced arthritis [3]. The genus *Aloe* belongs to the lily family. It contains a colorless, waterthin gel covered by a hard green brine. The common name *Aloe vera* means "the true Aloe". The chemical makeup of the gel consists of lignins, saponins, anthraquinones, minerals, vitamins and polysaccharides from which the biological activity comes. There are no known side effects of the plant gel. The active amino acids, vasoconstrictors and acetylsalicyclic acid found in the gel account for its anti-inflammatory activity but the experimental work needed to verify its beneficial responses appears to be lacking. In the present study, we attempted to show the comparative biological activity of *Aloe vera* as measured by standard anti-inflammatory tests: polymorphonuclear leukocyte (PMN) infiltration, mustard induced edema, antifibrosis and wound healing.

Materials and methods: Adult male ICR mice (20–30 g b.wt; 12 animals per group) were placed under ether anesthesia and were shaven on one side. A marking pencil was used to outline a 6 mm diameter area. Within this area, each animal was injected subcutaneously with 2% gelatin to form a skin bled or blister followed by a second subcutaneous injection of 2 mg/kg *Aloe vera* (Terry Corporation, Melbourne, Florida) outside the designated circle. PMN infiltration into this inflamed area was determined by staining the subdermal tissue with Wright stain and counting the PMNs per high power field in each animal in 3 h [4]. Adult male Sprague-Dawley rats (250–300 g; 12 animals per group) were given a 2% mustard (Colman, UK) injection (0.1 ml) into the planter surface of the paw. *Aloe vera* subcutaneous injections of 10 mg/kg were given at the same time as well as the day before. The injection was given subcutaneously away from the paw area. Plethysmographic volumetric readings were recorded immediately after the mustard injection and 6 h later. The difference in volumes was recorded as an index of edema. The reduction in paw volume by *Aloe vera* as compared to mustard controls was noted as percentage inhibition [5].

In adult Sprague-Dawley rats (150–300 g; 12 animals per group) two cotton pellets were implanted under the skin. *Aloe vera* was subcutaneously injected daily for 12 days in various dose groups up to 400 mg/kg. The next day the pellets with granuloma tissue were removed and dried. The constant dry weight of the tissue was obtained in a drying oven. A reduction in tissue weight by *Aloe vera* was taken as an index of antifibrosis activity [6].

A 6 mm Baker punch was used to remove a circular piece of skin from both shaven sides of ICR mice (35–40 g; 12 animals per group) and Sprague Dawley rats (180–270 g; 12 animals per group) under ether anesthesia. A Vernier caliper was used to measure wound diameters as an expression of wound healing. The mice and rats were then daily subcutaneously injected with 100 and 10 mg/kg, respectively, of *Aloe vera* over 7 days. The percentage reduction of wound diameter compared to non-treated controls was recorded [7].

Table 1: Inhibition of inflammation by Aloe vera *(means ± SEM; n = 12)*

Test	Animal	Dose (s.c.) (mg/kg)	Inhibition (%)
Wound healing	Mouse	100	24 ± 1.3
	Rat	10	31 ± 0.8
Mustard edema	Rat	10	44 ± 4.1
PMN infiltration	Mouse	2	58 ± 3.4
Antifibrosis	Rat	400	0 ± 0.0

Results and discussion: *Aloe vera* improved wound healing in rats and mice (Table 1). In mice at a dosage of 100 mg/kg, a significant 24 ± 1.3% improvement of wound healing was observed as compared to 31 ± 0.8% for rats at 10 mg/kg. The response on wound size reduction is dose related in a straight line fashion. A slightly greater response on inhibiting mustard induced edema was demonstrated by 10 mg/kg *Aloe* at 44 ± 4.1%. Increasing the dose of *Aloe vera* increased the response up to a maximum of 70 ± 9.1% inhibition. The most marked effect of *Aloe vera* was noted in its reduction of the PMN infiltration into an area irritated by gelatin. A 58 ± 3.4% inhibition was observed by an *Aloe vera* dose of 2 mg/kg. No inhibition of the growth of granuloma tissue around a cotton pellet was demonstrated at doses in various dose groups up to 400 mg/kg. These data indicate that *Aloe vera* works effectively against inflammation and that it improves wound healing. Since *Aloe vera* was most active on the PMN infiltration, we feel that it does not act like a steroid and that it is most effective on the acute inflammation phase. Possibly, *Aloe vera* inhibits inflammation by blocking mediators such as prostaglandin, thromboxane, kinin, enzymes or immune mechanisms. However, much more information is needed to understand how *Aloe vera* works. The army of substances present in *Aloe vera* may prevent the antigen-antibody reaction and block the action or release of inflammatory mediators while, at the same time, improving wound healing.

1. Silber, R.H. (1959) *Ann. NY Acad. Sci.*, **82**, 821-827
2. Hanley, D.C., Solomon, W.A., Davis, R.H. *et al.* (1982) *J. Am. Pod. Assoc.*, **72**, 275-284
3. Davis, R.H., Shapiro, E. and Agnew, P.S. (1985) *J. Am. Pod. Assoc.*, **75**, 229-237
4. Davis, R.H., Fisher, J.S. and McGowan, L. (1968) *J. Endocrinol.*, **41**, 603-604
5. Levy, A.C., Davis, R.H., Holtkamp, D.E. *et al.* (1961) *Fed. Proc.*, **20**, 158
6. Boland, E.W. (1959) *Ann. NY Acad. Sci.*, **82**, 887-901
7. Davis, R.H. (1986) *J. Am. Pod. Assoc.*, (in press).

Dr R.H. Davies, Pennsylvania College of Podiatric Medicine, 8th at Race Street, Philadelphia, Pennsylvania 19107, USA.

Paper received: 30th December, 1986; amended 3rd February, 1987