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EFFECT OF TOTAL AQUEOUS EXTRACT OF *SACOGLOTTIS GABONENSIS* (HUMIRIACEAE) STEM BARK ON HEMATOLOGICAL AND BIOCHEMICAL PARAMETERS DURING WOUND HEALING IN WISTAR RATS

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ABSTRACT

Wound healing is a complex, dynamic process involving a number of molecular and cellular events involving thrombocytes, macrophages, neutrophils, lymphocytes, endothelial cells and fibroblasts, endothelial cells and fibroblasts. The aim of this study was to evaluate the effect of the total aqueous extract of *Sacoglottis gabonensis* stem bark (TAESg) on hematological and biochemical parameters during wound healing after oral and dermal administration in wistar rats. 42 rats were divided into seven groups of six rats. Group 1 received no treatment; groups 2, 3 and 4 were treated orally with distilled water, *Flukocin*® and TAESg respectively; groups 5, 6 and 7 were treated dermally with distilled water, *Baneocin*® and TAESg respectively. Three blood samples were taken from the retro-orbital sinus before induction, after induction and after wound treatment. These blood samples were used to determine the levels of hematological and biochemical parameters. This study showed that thrombocyte count and sedimentation rate in TAESg-treated rats were reduced compared with those of negative control rats treated with distilled water after treatment. TAESg also restored leukocyte, albumin and CRP levels. The same observations in TAESg-treated rats were made in positive control rats treated with *Flukocin*® and *Baneocin*®. Ultimately, TAESg possesses healing activity by acting on key molecules and cells in the wound healing process.

KEYWORDS

Sacoglottis gabonensis, Wounds, healing, Hematological parameters, Biochemical parameters and Rat.

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INTRODUCTION

A wound is a traumatic or surgical condition characterized by a spreading of the lips of the skin or a mucous membrane. This spreading may be passive, if it is linked to tissue elasticity, or mechanical, through contraction of an injured

muscle, for example[1,2]. There are two types of wound, acute and chronic[3]. Acute wounds are the result of trauma affecting healthy individuals. Burns, frostbite, bites, abrasions and surgery fall into this category. The healing process associated with this type of wound is predictable, and the duration can be estimated with some reliability. Chronic wounds are those characterized by abnormal prolongation of the healing process, from six months to several years. There may be many underlying reasons, but in many cases, chronic wounds reveal a more serious problem such as diabetes, infection, cancer or vascular disorders. Unlike acute wounds, this type of wound shows no signs of tissue repair or regeneration. Instead, there is a high rate of cell death and strong inflammation leading progressively to tissue necrosis[3]. The process of wound healing is achieved through a complex and dynamic process including several molecular and cellular events involving PDGF, FGF, TNF, IGF, and TGF α and β, thrombocytes, macrophages, neutrophils, lymphocytes, endothelial cells and fibroblasts[4]. In Côte d'Ivoire, *Sacoglottis gabonensis* is a plant used orally and dermally in the treatment of Buruli ulcer[5]. Buruli ulcer is a chronic necrotizing infection of the skin and soft tissues caused by *Mycobacterium ulcerans*[6]. Studies have shown that the total aqueous extract of *Sacoglottis gabonensis* stem bark (TAESg) possesses healing activity and haemostatic potential[7,8]. The aim of the present work is to evaluate the effect of TAESg on hematological and biochemical parameters during wound healing after oral and cutaneous administration in wistar rats.

MATERIAL AND METHODS

Material

Plant material

This consisted of *Sacoglottis gabonensis* (Humiriaceae) stem bark harvested at Ingrakon in the Alépé region, a town about 45km from the Abidjan district. A sample was identified in accordance with that kept at the Centre National de Floristique (CNF) under number 1154 dated June 16, 1965.

Animal material

The experiments were carried out on male and female albino rats of the wistar strain *Rattus norvegicus*. The experimental protocol and animal handling procedures were carried out in accordance with Good Laboratory Practice[9].

Methods

Preparation of TAESg

Freshly harvested bark is crushed into small pieces and then dried in the laboratory on the bench at 25°C for four weeks. The dried bark is ground to a fine powder using a Retsch SM 100 mill. The total aqueous extract is prepared according to the preparation method described by Kouassi[10]. Four hundred grams (400g) of *Sacoglottis gabonensis* stem bark powder is dissolved in two liters (2L) of distilled water and the whole, is boiled for 30 minutes. After cooling, the decoctate is filtered, first on absorbent cotton, then on Wattman N°1 paper. The filtrates are oven-dried at 50°C for 48 hours. A dry, brown powder of TAESg is obtained and stored in the freezer at -5°C.

Assessment of wound-healing activity

Wound-healing activity has been assessed in an experimental burn model in rats[11,12]. The adaptation lies in the temperature and wound induction time to create extensive wounds[13]. To this end, forty-two (42) rats were homogeneously divided into seven groups of six rats, which were separated in individual cages.

Burn induction

Rats were anesthetized by inhalation in a bell jar containing ethyl ether-soaked cotton for 30 seconds to 1 minute. The dorsal flanks of anesthetized rats were shaved and cleaned with alcohol 24 h before burn induction. Experimental burns were induced using a 3cm diameter metal cylinder connected to a rod with a handle in all rats of the different groups except group 1. The cylinder, heated to a temperature of 200°C, was applied for 20 seconds with gentle pressure to the surface of the rats' shaved skin to induce extensive deep second-degree burns[13]. This burn is characterized by damage to the epidermis and dermis, with the presence of

phlyctenes (liquid of vascular origin forming a bubble that develops at the epidermis-dermis interface) with a red background and whitish areas[14].

Treatment of animals

All the animals in these different groups received the various treatments once a day from 9 a.m. over a period of 35 days as follows:

Group No.1, not induced, received no treatment;

Group No.2, the induced A negative control, received distilled water orally;

Group No.3, positive control A induced, receives *Flukocin*® 500mg orally at a dose of 14.28mg/kg bw;

Group No.4, Test A induced, receives TAESg at a dose of 3.5mg/kg bw orally;

Group No.5, negative control B induced, receives distilled water via the cutaneous route

Group No.6, positive control B induced, receives *Baneocin*® at a dose of 81.6mg/kg bw via the cutaneous route;

Group No.7, Test B induced, receives TAESg at a dose of 5000mg/kg bw.

Blood bioassays

Blood samples were taken according to the method described by Waynforth[15]. In this study, three blood samples were taken, before wound induction, four days after wound induction and at the end of the experiment. The blood taken immediately was collected in tubes containing the anticoagulant ethylene diamine tetra-acetic acid (EDTA) for blood count[16,17] using an automatic analyzer (Sysmex XT-2000 i, Japan), in black tube containing sodium citrate for sedimentation rate and in dry tubes for biochemical parameters using the automated biochemistry system (Cobas C 111, Switzerland).

Statistical analysis of results

Data are analyzed using Graph Pad Prism 8.0.1 (San Diego, CA, USA). Results are expressed as the mean followed by the standard error of the mean (M ± SEM). Statistical significance is determined by ANOVA 1 followed by the Turkey test. These tests will give us the degree of significance for $p< 0.05$. In the presentation of results, symbols (*, **, ***, ****/ $^{\#}$, $^{\#\#}$, $^{\#\#\#}$, $^{\#\#\#\#}$) will indicate significant decreases and increases compared to controls.

RESULTS AND DISCUSSION

Evolution of hematological parameters during wound healing

Before wound induction

The results showed that the erythrocyte count and erythrocyte indices namely hemoglobin, hematocrit, GMV, MCHT and MCHC of the different groups 2; 3; 4; 5; 6 and 7 were statistically identical ($p> 0.05$) to those of healthy control group 1 rats (Table No.1). The erythrocyte count for groups 2; 3; 4; 5; 6 and 7 was 7.75 ± 0.31; 7.79 ± 0.29; 7.67 ± 0.24; 8.01 ± 0.19; 7.80 ± 0.41 and 7.83 ± 0.38.10^6/mm^3 respectively, compared with 7.63 ± 0.22.10^6/mm^3 for group 1. The same observation was made for leukocyte parameters and indices, as well as for thrombocytes (Table No.2).

After wound induction

After skin wound induction, the results showed that there was no significant difference ($p> 0.05$) in the erythrocyte count and erythrocyte indices of the different groups 2; 3; 4; 5; 6 and 7 compared with that of healthy control group 1 rats (Table No.3).

With regard to leukocyte parameters and leukocyte indices (Table No.4), after skin wound induction, analysis of the results showed a very highly significant ($p< 0.0001$) increase in leukocyte count and leukocyte indices as well as thrombocytes in rats from groups 2; 3; 4; 5; 6 and 7 compared with that of rats from the healthy control group 1. The high leukocyte count for groups 2; 3; 4; 5; 6 and 7 was 15.16 ± 0.18; 15.80 ± 0.14; 16.03 ± 0.17; 15.67 ± 0.12; 17.50 ± 0.11; 16.28 ± 0.18.10^6/mm^3 respectively, compared with 10.04±0.54.10^6/mm^3 for the healthy control group 1. Thrombocyte levels were as follows in rats from group 2 (1012 ± 110.2.10^6/mm^3); group 3 (1008.8 ± 120.8.10^6/mm^3); group 4 (1002 ± 123.5.10^6/mm^3); group 5 (1080 ± 125.0.10^6/mm^3); group 6 (1101 ± 112.4.10^6/mm^3) and group 7 (996 ± 208.2.10^6/mm^3) compared with the healthy control group 1 (375.6 ± 18.37.10^6/mm^3).

After wound treatment

After 35 days of skin wound treatment, the erythrocyte and erythrocyte indices of the various treated groups did not vary ($p > 0.05$) compared with those of untreated healthy control group 1 rats, irrespective of the route of administration (Table No.5).

Treatment of groups 4 and 7 with TAESg according to route of administration brought leukocyte counts and leukocyte indices back to normal values (Table No.6). These levels were similar to those of rats in positive control groups 3 and 6 (A and B) treated with the reference substances by route of administration.

These different levels in rats from these different groups by route of administration were statistically identical ($p > 0.05$) to those in untreated healthy control group 1 rats. However, rats from negative control groups 2 and 5 (A and B) treated with distilled water according to the routes of administration presented very highly superior levels ($p < 0.0001$) of leukocytes and leukocyte indices compared with untreated healthy control group 1. These rates were 15.30 ± 0.62 and 14.20 ± $0.32.10^6/mm^3$ respectively for groups 2 and 5 compared with group 1, which was 10.78 ± $0.38.10^6/mm^3$.

Similarly, thrombocyte count (Table No6), showed a statistically significant decrease ($p < 0.0001$) in rats from groups 4 and 7 with TAESg depending on the route of administration compared with negative control groups (A and B) treated with distilled water. However, thrombocyte levels in TAESg-treated rats, irrespective of the route of administration, showed a statistically significant increase ($p < 0.0001$) compared with untreated healthy control group 1 rats. The same observation was made for the oral and cutaneous routes of TAESg-treated rats, compared with the reference substances for these same routes.

Evolution of sedimentation rate

Before induction, no significant difference ($p > 0.05$) in sedimentation rate was observed in rats from the different groups 2; 3; 4; 5; 6 and 7 compared with the untreated healthy control group 1 (Table No.7).

On day 4 after wound induction, sedimentation velocity showed a statistically significant increase ($p < 0.0001$) in rats from different groups: Negative controls (A and B) treated with distilled water, positive controls (A and B) treated with reference substances and trials treated with TAESg (Trial A and B), depending on the route of administration, compared with group 1, the untreated healthy control.

After 35 days of treatment, the sedimentation rate decreased very significantly ($p < 0.0001$) in rats from groups 4 and 7 treated with TAESg compared with rats from groups 2 and 5, positive controls (A and B) treated with distilled water by the oral and cutaneous routes respectively at both 1st and 2nd hour. This reduction in sedimentation rate in rats from groups treated with TAESg was statically identical ($p > 0.05$) to that exerted by the reference substances in rats from positive control groups 3 and 6 (A and B) and was close to that of the healthy control group 1 untreated by all routes of administration at 1 hr. However, at 2 h, sedimentation rate showed a statistically significant increase ($p < 0.01$) in rats from groups treated with TAESg and reference substances at all routes of administration compared with the untreated healthy control group 1. However, between rats from groups treated with TAESg and reference substances, no statistically significant difference ($p > 0.05$) in sedimentation rate was noted at 2 h at all routes of administration.

Evolution of biochemical parameters

Before induction

Before skin wound induction, no statistically significant variations ($p > 0.05$) in total protein, albumin, alkaline phosphatase, calcium and c-reactive protein levels were observed (Table No.8). These total protein levels were 6.1 ± 0.16; 6.52 ± 0.23; 6.74 ± 0.18; 6.13 ± 0.20; 6.34 ± 0.25 and 6.53 ± 0.31 mg/dL respectively for groups 2; 3; 4; 5; 6 and 7, compared with the normal control group 1 of 6.55 ± 0.29 mg/dL. For albumin, these levels were 3.39 ± 0.19; 3.38 ± 0.11; 3.53 ± 0.14; 3.41 ± 0.19;

3.62 ± 0.19; 3.60 ± 0.16 and 3.33 ± 0.13g/dL compared with group 1 normal control which is 3.39 ± 0.19g/dL. Alkaline phosphatase levels were 393.3 ± 8.77; 402.1 ± 5.39; 405.7 ± 8.55; 404.5 ± 11.66; 389.3 ± 10.16 and 409 ± 2.66 IU/L for groups 2; 3; 4; 5; 6 and 7 respectively, compared with 398.5 ± 12.51 IU/L for group 1, the normal control. Calcium levels varied from 12.57 ± 0.44; 13.27 ± 0.17; 12.59 ± 0.46; 12.75 ± 0.33; 12.32 ± 0.59; 12.32 ± 0.35mmol/L for groups 2; 3; 4; 5; 6 and 7 respectively, versus 12.27 ± 0.47mmol/L for normal controls. CRP levels were 0.018 ± 0.003; 0.018 ± 0.005; 0.018 ± 0.003; 0.018 ± 0.006; 0.015 ± 0.006; 0.021 ± 0.003mg/L for groups 2; 3; 4; 5; 6 and 7 respectively, compared with 0.020 ± 0.006mg/L for group 1, the normal control.

After induction

After induction of skin wounds, total protein and albumin showed a very, very statistically significant ($p< 0.0001$) drop in their mean levels in all rats in groups 2; 3; 4; 5; 6 and 7 compared with the untreated healthy control group 1 (Table No.9).

This decrease in total protein levels was 3.99 ± 0.44; 3.69 ± 0.29; 3.57 ± 0.17; 3.63 ± 0.42; 3.67 ± 0.44 and 3.78 ± 0.27mg/dL respectively for groups 2; 3; 4; 5; 6 and 7 versus 6.38 ± 0.28mg/dL for the untreated healthy control group 1. In terms of albumin, the mean levels for groups 2; 3; 4; 5; 6 and 7 were 2.55 ± 0.12; 2.70 ± 0.11; 2.31 ± 0.14; 2.46 ± 0.12; 2.60 ± 0.14 and 2.33 ± 0.13g/dL respectively, compared with 3.35 ± 0.16g/dL for group 1, the untreated healthy control. However, PAL, calcium and CRP, showed a highly statistically significant ($p< 0.0001$) increase in their mean levels in all rats in groups 2; 3; 4; 5; 6 and 7 compared to those in group 1 untreated healthy control. This increase in PAL was 598.3 ± 26.62; 597.4 ± 30.33; 598.8 ± 29.86; 617.8 ± 18.74; 614 ± 14.21 and 573 ± 20.31 IU/L respectively for groups 2; 3; 4; 5; 6 and 7, compared with the untreated healthy control group 1 of 406.3 ± 6.79IU/L. Calcium levels in groups 2, 3, 4, 5, 6 and 7 were 18.92 ± 0.79, 19.45 ± 0.76, 19.92 ± 0.72, 19.25 ± 0.67, 20.16 ± 0.87 and 19.32 ± 0.72mmol/L respectively, compared with an untreated healthy control of group 1, whose mean level was 12.77 ± 0.20 mmol/L. CRP levels were 0.165 ± 0.019; 0.150 ± 0.021; 0.150 ± 0.016; 0.168 ± 0.009; 0.195 ± 0.002 and 0.175 ± 0.019g/L, compared with 0.016 ± 0.004g/L for group 1 untreated healthy control.

After treatment

After 35 days of treatment, total protein and albumin still showed a decrease that was statistically significant ($p< 0.0001$) and an increase that was statistically significant ($p< 0.0001$) in rats from groups 2 and 5 treated orally and with distilled water respectively, compared with the untreated healthy control group 1 (Table No.10). These decreases were 3.65 ± 0.30 and 3.63 ± 0.26mg/dL respectively for groups 2 and 5 compared with the untreated healthy control of 6.05 ± 0.28mg/dL. For total protein, the decrease was 1.97 ± 0.10 and 1.97 ± 0.06g/dL respectively for groups 2 and 5 compared with the untreated healthy control, which was 3.25 ± 0.23g/dL for albumin. On the other hand, groups 3; 4; 6 and 7 showed a non-statistically significant variation ($p> 0.05$) in mean total protein and albumin levels compared with those of the untreated healthy control group 1.

A statistically significant ($p< 0.0001$) increase in PAL levels and a statistically significant ($p< 0.0001$) increase in calcium and CRP levels were noted in rats from groups 2 and 5 treated orally and dermally with distilled water respectively, compared with the untreated healthy control group 1. In contrast, rats from groups 3; 4; 6 and 7 showed a non-significant variation ($p> 0.05$) in mean PAL, calcium and CRP levels compared with those from the untreated healthy control group 1. On the other hand, groups 3 and 4 treated respectively with *Flukocin*® at a dose of 14.28mg/kg bw and TAESg at a dose of 3.5mg/kg bw. showed statistically significant ($p< 0.0001$) decreases in PAL levels (447.4 ± 8.89 and 435.5 ± 12.97 IU/L) and statistically significant ($p< 0, 0001$) in calcium (13.5 ± 0.40 and 13.93 ± 0.27mmol/L) and CRP (0.035 ± 0.004 and 0.033 ± 0.004g/L) levels compared with group 2 negative control A (548.3 ± 30.36 IU/L; 17.75 ± 0.23mmol/L and 0.170 ± 0.009g/L respectively for PAL, calcium and CRP)

treated orally with distilled water. The same observation was made for the different groups treated by the cutaneous route. For the cutaneous route, PAL levels were 431 ± 5.98 and 422.3 ± 9.28 IU/L for groups 6 and 7 treated respectively with *Baneocin*® at 81.6mg/kg bw and TAESg at 5000mg/kg bw, compared with 567.8 ± 44.73 IU/L for group 5 negative control B treated with distilled water. Calcium levels were 14.00 ± 0.22 and 13.87 ± 0.28mmol/L for groups 6 and 7 treated respectively with *Baneocin*® at 81.6mg/kg bw and TAESg at 5000mg/kg bw, compared with group 5 negative control B treated with distilled water at 17.91 ± 0.40mmol/L. CRP levels were 0.030 ± 0.003 and 0.028 ± 0.005g/L for groups 6 and 7 treated with *Baneocin*® 81.6mg/kg bw and TAESg 5000 mg/kg bw respectively, compared with 0.142 ± 0.007g/L for group 5 negative control B treated with distilled water.

Discussion

Wound healing is a natural biological process that takes place after the soft tissues of the body have been opened up by a hot mechanical, chemical, electrical or infectious agent, with the aim of healing the lesion by filling in lost substances and reuniting the wound edges[18,19].

Hematologically, before induction, quantitative results showed similar levels of erythrocyte, leukocyte and thrombocyte parameters in all rats from the different groups. However, after induction, only leukocyte parameters, including leukocyte indices, showed a highly significant increase in their mean levels. Indeed, induction of burn wounds provoked inflammation, triggering the active release of leukocytes responsible for the body's immune defense[20]. After treatment, the leukocyte count and leukocyte indices of rats from groups treated with TAESg and the reference substances showed a statistically significant drop compared with rats from negative control groups A and B treated with distilled water from the different treatment routes. *Flukocin*® and *Baneocin*® are antibiotics that contain molecules capable of acting on wounds by reducing the inflammatory process responsible for the reduction in leukocytes. TAESg effect on leukocytes is similar to that of *Flukocin*® and *Baneocin*®. TAESg also accelerates wound healing, which is thought to reduce or halt the inflammatory process, resulting in lower leukocyte counts and leukocyte indices. The mean thrombocyte count and sedimentation rate were statistically significantly higher than in healthy control rats. Thrombocytes are involved in hemostasis and play a role in local vasoconstriction[21]. Furthermore, according to Pence and Woods[22], wound induction causes inflammation through the activation of factor XII, triggering cascades of proteolysis that lead to the production of fibrin from fibrinogen, thus initiating coagulation. Therefore, the increase in fibrinogen could be one cause of the increase in both the erythrocyte sedimentation rate (ESR) and the platelet count. However, after treatment with TAESg and reference substances, both the ESR and platelet count decreased significantly compared to the different negative control groups A and B treated orally and transdermally. This decrease in ESR and platelet count is thought to be due to the presence of tannins in TAESg. Indeed, tannins have a healing power by strengthening blood vessels and thus contributing to the accumulation of vitamin K in the body[23,24]. These results are similar to those obtained by Singh *et al*[25], who showed that aqueous extract of Pleurotus ostreatus at doses of 100 and 300mg/kg bw restores platelet count and erythrocyte sedimentation rate. The persistence of elevated leukocyte counts, including leukocyte indices, erythrocyte sedimentation rate, and platelet counts in negative control groups A and B rats treated orally and topically with distilled water, could be explained by an infectious wound condition.

At the biochemical level, prior to induction, there were no statistically significant variations in total protein, albumin, alkaline phosphatase, calcium, or CRP levels in any of the rats across the different groups. After wound induction, the results showed a statistically significant decrease in total protein and albumin levels and a statistically significant increase in alkaline phosphatase, calcium, and CRP

levels in all treated rats across the different groups compared to the untreated healthy control. The decrease in total protein and albumin levels could be attributed to the heat generated during wound induction. Indeed, heat leads to protein denaturation, resulting in a loss of skin elasticity and thus contributing to inflammatory activity[26]. This protein denaturation is primarily characterized by alterations in hydrophobicity, electrostatics, hydrogen bonds, and disulfide bridges that stabilize the molecules[27,26]. The increase in ALP levels could be due to heightened liver activity in the synthesis of hemostatic substances and inflammatory mediators. These enzymes, present at very high concentrations in the liver, can promote hepatocellular damage[28]. The increased calcium level would be explained by its enhanced synthesis by the liver. According to Verlander[29], calcium is necessary for the functioning of numerous intracellular and extracellular processes, including cell contraction and coagulation. The platelet count and erythrocyte sedimentation rate showed a very significant increase, which would explain its elevation after induction.

As for CRP, its level increased very significantly because the heat induced by the causative agent during induction, leading to loss of skin continuity, would trigger inflammatory stimulation responsible for the secretion of IL-6 by macrophages and T cells, which in turn release CRP in the liver[30,31]. After treatment, rats in the groups treated with TAESg and the reference substances orally and topically showed a restoration of total protein, albumin, ALP, calcium, and CRP levels. This restoration in the TAESg-treated groups was similar to that in the group treated with the reference substances. According to Nabil *et al*[23], tannins possess hemostatic activity that precipitates and protects proteins from denaturation. Thus, the catecholic tannins in TAESg would be responsible for restoring these proteins. The persistence of low total protein and albumin levels and high ALP, calcium and CRP levels in the groups of rats treated with distilled water orally and topically could be explained by a chronic inflammatory state due to the lack of adequate treatment, leading to prolonged protein denaturation and high liver activity.

Table No.1: Erythrocyte parameter values before wound induction

S.No		Group 1	Group 2	Group 3	Group 4	Group 5	Group 6	Group 7
1	Erythrocytes (10^6/mm^3)	7.63±0.22	7.75±0.31	7.79±0.29	7.67±0.24	8.01±0.19	7.80±0.41	7.83±0.38
2	Hemoglobin (g/dL)	14.64±0.19	14.60±0.16	15.01±0.09	14.30±0.11	14.44±0.10	13.98±0.15	14.76±0.13
3	Hematocrit (%)	42.84±0.88	40.82±0.72	40.72±0.87	41.55±0.67	41.72±0.72	41.81±0.76	40.96±0.96
4	VGM (fL)	56.40±1.52	54.30±2.60	54.63±2.14	53.50±2.60	52.90±3.16	54.25±2.08	55.06±1.80
5	TCMH (Pg)	22.14±0.85	21.34±1.60	20.72±2.10	20.81±2.40	21.16±1.88	22.08±0.90	20.72±3.12
6	CCMH (g/dL)	35.00±0.51	36.03±0.42	35.80±0.61	34.71±0.58	33.80±0.67	34.60±0.80	35.65±0.90

Values are presented as the mean followed by the standard error of the mean (ME ± SEM). The comparison is made between group 1 and groups 2, 3, 4, 5, 6 and 7 treated according to the routes of administration. n = 6. Group No.1: Untreated rats; Group No.2: Rats treated with distilled water orally; Group No.3: Rats treated with Flukocin® at a dose of 14.28mg/kg bw orally; Group 4: rats treated with TAESg at a dose of 3.5mg/kg bw orally; Group No.5: Rats treated with distilled water topically; Group No.6: Rats treated with Baneocin® at a dose of 81.6mg/kg bw topically; Group No.7: Rats treated with TAESg at a dose of 5000mg/kg bw topically.

Table No.2: Leukocyte parameter values before wound induction

S.No		Group 1	Group 2	Group 3	Group 4	Group 5	Group 6	Group 7
1	Leukocytes ($10^3/mm^3$)	10.38±0.63	10.21±0.72	10.30±0.67	11.03±0.58	10.28±0.56	9.87±0.74	10.16±0.71
2	Lymphocytes ($10^3/mm^3$)	9.30±0.51	10.12±0.48	9.87±0.60	10.03±0.70	11.02±0.42	11.06±0.38	10.37±0.80
3	Monocytes ($10^3/mm^3$)	0.85±0.11	0.86±0.10	0.91±0.13	0.90±0.11	0.88±0.30	0.92±0.16	0.97±0.12
4	Granulocytes ($10^3/mm^3$)	1.09±0.17	1.14±0.16	1.14±0.18	1.13±0.16	1.08±0.20	1.12±0.18	1.09±0.17
5	Thrombocytes ($10^3/mm^3$)	358.9±40.68	401.1±10.12	390.8±23.7	385.0±22.76	403.0±5.72	402.0±8.05	396.0±27.8

Values are presented as the mean followed by the standard error of the mean (ME ± SEM). The comparison is made between group 1 and groups 2, 3, 4, 5, 6 and 7 treated according to the routes of administration. n = 6. Group No.1: Untreated rats; Group No.2: Rats treated with distilled water orally; Group No.3: Rats treated with Flukocin® at a dose of 14.28mg/kg bw orally; Group No.4: Rats treated with TAESg at a dose of 3.5mg/kg bw orally; Group No.5: Rats treated with distilled water topically; Group No.6: Rats treated with Baneocin® at a dose of 81.6mg/kg bw topically; Group No.7: Rats treated with TAESg at a dose of 5000mg/kg bw topically.

Table No.3: Variation in erythrocyte parameter levels after wound induction

S.No		Group 1	Group 2	Group 3	Group 4	Group 5	Group 6	Group 7
1	Erythrocytes ($10^6/mm^3$)	7.80±0.19	8.14±0.16	7.96±0.22	8.21±0.17	7.82±0.28	7.47±0.31	8.05±0.11
2	Hemoglobin (g/dL)	14.56±0.19	14.50±0.20	15.11±0.13	14.44±0.18	13.89±0.23	14.08±0.18	15.06±0.11
3	Hematocrit (%)	42.76±0.83	42.60±0.72	41.36±0.98	43.07±0.60	42.33±0.71	42.48±0.70	42.72±0.80
4	VGM (fL)	56.90±1.54	55.80±2.14	56.00±0.60	56.14±0.92	55.90±1.80	55.48±1.92	56.10±1.42
5	TCMH (Pg)	22.31±0.79	21.40±1.10	21.61±1.12	20.72±2.15	20.88±2.30	21.80±1.72	22.13±0.80
6	CCMH (g/dL)	34.50±0.44	34.00±0.30	35.03±0.28	33.72±1.79	35.00±0.36	34.09±0.79	33.90±1.37

Values are presented as the mean followed by the standard error of the mean (ME ± SEM). The comparison is made between group 1 and groups 2, 3, 4, 5, 6 and 7 treated according to the routes of administration. n = 6. Group No.1: Untreated rats; Group No.2: Rats treated with distilled water orally; Group No.3: Rats treated with Flukocin® at a dose of 14.28mg/kg bw orally; Group No.4: Rats treated with TAESg at a dose of 3.5mg/kg bw orally; Group No.5: Rats treated with distilled water topically; Group No.6: Rats treated with Baneocin® at a dose of 81.6mg/kg bw topically; Group No.7: Rats treated with TAESg at a dose of 5000mg/kg bw topically.

Table No.4: Variation in leukocyte parameter levels and platelet count after wound induction

S.No		Group 1	Group 2	Group 3	Group 4	Group 5	Group 6	Group 7
1	Leukocytes ($10^3/mm^3$)	10.04 ±0.54	15.16 ±0.18####	15.80 ±0.14####	16.03 ±0.17####	15.67 ±0.12####	17.50 ±0.11####	16.28 ±0.1####
2	Lymphocytes ($10^3/mm^3$)	9.13 ±0.51	13.08 ±0.76####	13.21 ±0.69####	13.80 ±0.81####	13.74 ±0.78####	13.06 ±0.81####	13.57 ±0.87####
3	Monocytes ($10^3/mm^3$)	0.68 ±0.11	1.92 ±0.03####	1.80 ±0.05####	1.76 ±0.08####	1.84 ±0.02####	2.06 ±0.02####	1.91 ±0.04####
4	Granulocytes ($10^3/mm^3$)	1.71 ±0.16	2.69 ±0.14####	2.64 ±0.13####	2.68 ±0.12####	2.77 ±0.14####	2.81 ±0.15####	2.86 ±0.10####
5	Thrombocytes ($10^3/mm^3$)	375.6 ±18.37	1012 ±110.2####	1008.8 ±120.8####	1002 ±123.5####	1080 ±125.0####	1101 ±112.4####	996 ±208.2####

Values are presented as Mean followed by Standard Error of Mean (ME ± SEM). The comparison is made between group 1 and groups 2, 3, 4, 5, 6 and 7 treated according to the routes of administration. $p < 0.05$, $n = 6$; Group No.1: Untreated rats; Group No.2: Rats treated with distilled water orally; Group No.3: Rats treated with Flukocin® at a dose of 14.28mg/kg bw orally; Group No.4: Rats treated with TAESg at a dose of 3.5mg/kg bw orally; Group No.5: rats treated with distilled water topically; Group No.6: Rats treated with Baneocin® at a dose of 81.6mg/kg bw topically. Group No.7: Rats treated with TAESg at a dose of 5000mg/kg bw via the cutaneous route.

Table No.5: Variation in erythrocyte parameter levels after wound treatment

S.No		Group 1	Group 2	Group 3	Group 4	Group 5	Group 6	Group 7
1	Erythrocytes ($10^6/mm^3$)	8.20±0.15	8.69±0.14	7.80±0.15	8.08±0.17	8.79±0.18	8.32±0.13	7.81±0.14
2	Hemoglobin (g/dL)	13.31±0.14	14.12±0.15	13.68±0.16	14.08±0.16	13.89±0.23	13.82±0.18	13.90±0.15
3	Hematocrit (%)	41.60±0.66	41.36±0.80	40.80±0.81	41.39±0.69	42.33±0.71	42.24±0.72	40.16±0.67
4	VGM (fL)	54.81±1.50	56.80±1.58	55.00±1.66	54.31±1.62	55.90±1.80	55.36±1.70	55.15±1.56
5	TCMH (Pg)	21.06±0.81	22.15±0.68	21.60±0.90	21.40±0.96	20.88±2.30	20.90±0.84	21.32±0.58
6	CCMH (g/dL)	34.11±0.48	33.66±0.52	34.08±0.67	34.02±0.44	35.00±0.36	33.48±0.37	34.10±0.73

Values are presented as the mean followed by the standard error of the mean (ME ± SEM). The comparison is made between group 1 and groups 2, 3, 4, 5, 6 and 7 treated according to the routes of administration. $n = 6$. Group No.1: Untreated rats; Group No.2: Rats treated with distilled water orally; Group No.3: Rats treated with Flukocin® at a dose of 14.28mg/kg bw orally; Group No.4: Rats treated with TAESg at a dose of 3.5mg/kg bw orally; Group No.5: Rats treated with distilled water topically; Group No.6: Rats treated with Baneocin® at a dose of 81.6mg/kg bw topically; Group No.7: Rats treated with TAESg at a dose of 5000mg/kg bw topically.

Table No.6: Variation in leukocyte parameter levels and platelet count after wound treatment

S.No		Group 1	Group 2	Group 3	Group 4	Group 5	Group 6	Group 7
1	Leukocytes ($10^3/mm^3$)	10.78±0.38	15.30±0.62 ####	11.06±0.44	10.90±0.79	14.20±0.32 ####	10.94±0.28	11.08±0.30
2	Lymphocytes ($10^3/mm^3$)	9.20±1.08	14.06±2.16 ####	10.07±1.08	10.78±1.14	13.62±2.63 ####	11.02±1.30	10.76±1.27
3	Monocytes ($10^3/mm^3$)	0.91±0.86	1.81±0.77 ####	0.98±0.60	1.03±0.58	1.86±0.62 ####	1.00±0.38	1.12±0.31
4	Granulocytes ($10^3/mm^3$)	1.07±0.10	3.11±0.18 ####	1.56±0.16	1.38±0.18	2.82±0.06 ####	1.32±0.09	0.94±0.07
5	Thrombocytes ($10^3/mm^3$)	389.1±30.32	1120±10.8 ####	638.8±22.2 ####	710±20.72 ####	1030±21.6 ####	596.8±33.4 ####	668±27.80 ####

Values are presented as Mean followed by Standard Error of Mean (ME ± SEM). The comparison is made between group 1 and groups 2, 3, 4, 5, 6 and 7 treated according to the routes of administration. $p < 0.05$, $n = 6$; Group No.1: Untreated rats; Group No.2: Rats treated with distilled water orally; Group No.3: Rats treated with Flukocin® at a dose of 14.28mg/kg bw orally; Group No.4: Rats treated with TAESg at a dose of 3.5mg/kg bw orally; Group No.5: Rats treated with distilled water topically; Group No.6: rats treated with Baneocin® at a dose of 81.6mg/kg bw topically. Group No.7: Rats treated with TAESg at a dose of 5000mg/kg bw via the cutaneous route.

Table No.7: Evolution of sedimentation velocity before and after induction and after treatment

S.No			Group 1	Group 2	Group 3	Group 4	Group 5	Group 6	Group 7
1	Before induction	1h	6.2±0.15	5.2±0.24	6.3±0.17	6.1±0.12	5.7±0.28	7.1±0.13	6.3±0.14
		2h	12.3±0.31	12.4±0.28	13.3±0.19	12.6±0.36	13.7±0.23	14.4±0.18	15.3±0.15
2	4 days after induction	1h	7.2±2.66	18.3±3.80 ####	19.2±2.90 ####	20.1±0.76 ####	18.4±2.70 ####	20.3±0.72 ####	19.6±1.98 ####
		2h	15.4±1.50	33.6±3.58 ####	34.5±2.66 ####	33.3±2.62 ####	36.2±1.80 ####	35.3±1.70 ####	33.6±2.56 ####
3	35 days after treatment	1h	7.6±2.80	17.4±1.68 ####	9.9±1.20 ****	8.7±2.30 ****	16.3±2.30 ####	10.2±1.84 ****	11.4±0.88 ****
		2h	14.3±1.48	28.6±3.52 ####	20.5±0.80 **	19.4±0.68 **	30.7±0.36 ####	21.8±0.37 **	19.8±1.73 **

Values are presented as the mean followed by the standard error of the mean (ME ± SEM). The comparison is made between group 1 and groups 2, 3, 4, 5, 6 and 7 on the one hand and between groups 2 and 5 and groups 4 and 7 treated according to the routes of administration on the other hand. $p < 0.05$, $n = 6$; Group No.1: Untreated rats; Group No.2: Rats treated with distilled water orally; Group No.3: Rats treated with Flukocin® at a dose of 14.28mg/kg bw orally; Group No.4: Rats treated with TAESg at a dose of 3.5mg/kg bw orally; Group No.5: Rats treated with distilled water topically; Group No.6: Rats treated with Baneocin® at a dose of 81.6mg/kg bw topically. Group No.7: Rats treated with TAESg at a dose of 5000mg/kg bw via the cutaneous route.

Table No.8: Variation in the levels of some biochemical parameters before wound induction

Groups	Total protein (PT)	Albumin (Alb)	Alkaline phosphatase (PAL)	Calcium (Ca^{2+})	C-Reactive Protein (CRP)
Group 1	6.55±0.29	3.39±0.19	398.5±12.51	12.27±0.47	0.020±0.006
Group 2	6.1±0.16	3.38±0.11	393.3±8.77	12.57±0.44	0.018±0.003
Group 3	6.52±0.23	3.53±0.14	402.1±5.39	13.27±0.17	0.018±0.005
Group 4	6.74±0.18	3.41±0.19	405.7±8.55	12.59±0.46	0.018±0.003
Group 5	6.13±0.20	3.62±0.19	404.5±11.66	12.75±0.33	0.018±0.006
Group 6	6.34±0.25	3.60±0.16	389.3±10.16	12.32±0.59	0.015±0.006
Group 7	6.53±0.31	3.33±0.13	409±2.66	12.32±0.35	0.021±0.003

Values are presented as Mean followed by Standard Error of Mean (ME ± SEM). Vo: Oral route; Vc: Cutaneous route; Voc: Oral route combined with the cutaneous route. The comparison is made between groups 2 and 5 and groups 4 and 7 treated according to the routes of administration. $p < 0.05$, $n = 6$; Group No.1: Normal control; Group No.2: Positive control treated orally with distilled water; Group No.3: Negative control treated orally with Flukocin® at a dose of 14.28mg/kg bw; Group No.4: Trial treated orally with TAESg at a dose of 3.5 mg/kg bw; Group No.5: Positive control treated cutaneously with distilled water. Group No.6: Negative control treated cutaneously with Baneocin® at a dose of 81.6mg/kg bw and Group No.7: Trial treated with TAESg cutaneously at a dose of 5000mg/kg bw.

Table No.9: Variation in the levels of some biochemical parameters 4 days after wound induction

Groups	Total protein (PT)	Albumin (Alb)	Alkaline phosphatase (PAL)	Calcium (Ca^{2+})	C-Reactive Protein (CRP)
Group 1	6.38±0.28	3.35±0.16	406.3±6.79	12.77±0.20	0.016±0.004
Group 2	3.99±0.44****	2.55±0.12***	598.3±26.62####	18.92±0.79####	0.165±0.019####
Group 3	3.69±0.29****	2.70±0.11***	597.4±30.33####	19.45±0.76####	0.150±0.021####
Group 4	3.57±0.17****	2.31±0.14***	598.8±29.86####	19.92±0.72####	0.150±0.016####
Group 5	3.63±0.42****	2.46±0.12***	617.8±18.74####	19.25±0.67####	0.168±0.009####
Group 6	3.67±0.44****	2.60±0.14***	614±14.21####	20.16±0.87####	0.195±0.002####
Group 7	3.78±0.27****	2.33±0.13***	573±20.31####	19.32±0.72####	0.175±0.019####

Values are presented as Mean followed by Standard Error of Mean (ME ± SEM). Vo: Oral route; Vc: Cutaneous route; Voc: Oral route combined with the cutaneous route. The comparison is made between groups 2 and 5 and groups 4 and 7 treated according to the routes of administration. $p < 0.05$, $n = 6$; Group No.1: Normal control; Group No.2: Positive control treated orally with distilled water; Group No.3: Negative control treated orally with Flukocin® at a dose of 14.28mg/kg bw; Group No.4: Trial treated orally with TAESg at a dose of 3.5mg/kg bw; Group No.5: Positive control treated cutaneously with distilled water. Group No.6: Negative control treated cutaneously with Baneocin® at a dose of 81.6mg/kg bw and Group No.7: Trial treated with TAESg cutaneously at a dose of 5000mg/kg bw.

Table No.10: Change in the levels of some biochemical parameters after 35 days of treatment

Groups	Total protein (PT)	Albumin (Alb)	Alkaline phosphatase (PAL)	Calcium (Ca^{2+})	C-Reactive Protein (CRP)
Group 1	6.05±0.28	3.25±0.23	428.0±15.94	13.02±0.17	0.013±0.003
Group 2	3.65±0.30****	1.97±0.10**	548.3±30.36##	17.75±0.23####	0.170±0.009####
Group 3	6.19±0.44####	3.20±0.20#	447.4±8.89**	13.5±0.40****	0.035±0.004****
Group 4	6.52±0.26####	3.15±0.29#	435.5±12.97**	13.93±0.27****	0.033±0.004****
Group 5	3.63±0.26****	1.97±0.06**	567.8±44.73###	17.91±0.40####	0.142±0.007####
Group 6	5.67±0.19####	3.23±0.20##	431±5.98***	14.00±0.22****	0.030±0.003****
Group 7	5.78±0.20####	3.17±0.33#	422.3±9.28***	13.87±0.28****	0.028±0.005****

Values are presented as Mean followed by Standard Error of Mean (ME ± SEM). Vo: Oral route; Vc: Cutaneous route; Voc: Oral route combined with the cutaneous route. The comparison is made between groups 2 and 5 and groups 4 and 7 treated according to the routes of administration. $p < 0.05$, $n = 6$, Group No.1: Normal control; Group No.2: Positive control treated orally with distilled water; Group No.3: Negative control treated orally with Flukocin® at a dose of 14.28mg/kg bw; Group No.4: Trial treated orally with TAESg at a dose of 3.5mg/kg bw; Group No.5: Positive control treated cutaneously with distilled water. Group No.6: Negative control treated cutaneously with Baneocin® at a dose of 81.6mg/kg bw and Group No.7: Trial treated with TAESg cutaneously at a dose of 5000mg/kg bw.

CONCLUSION

This study concludes that daily oral and topical administration of TAESg for 35 days has a healing effect by acting on key molecules and biological cells involved in the wound healing process, promoting wound closure. This study has scientifically verified and confirmed the traditional use of this plant in the treatment of Buruli ulcer in Côte d'Ivoire.

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CONFLICT OF INTEREST

We declare that we have no conflict of interest.

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