

Evaluation of In Vitro and In Vivo Wound Healing Activity of Polyherbal Formulation in Wistar Rats

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ABSTRACT

In this study, the effect of a prepared polyherbal formulation on the effective healing of wounds was evaluated by applying excision wound models in healthy Wistar rats. In the excision wound model, rats were categorized into three groups of six rats each. Group 1 served as controls, Group 2 received standard treatment with silver sulfadiazine, while Group 3 was treated with a prepared polyherbal formulation (study group). The wound-healing activity was assessed by estimating the reduction in wound surface area, antioxidant activity, skin-breaking strength, and biochemical parameters, such as hydroxyproline, collagen, and hexosamine. Biochemical parameters such as hydroxyproline, hexosamine, and collagen turnover were increased in test drug-treated animals compared to untreated animals. A histological study of granulation tissue was carried out to determine the extent of collagen formation in the wound tissue. In the Excision wound model, Polyherbal-treated animals showed a significant ($p < 0.001$) reduction in the wound surface area and a faster rate of wound contraction than controls (276.85 ± 03.19 and 422.033 ± 5.82 , respectively). Furthermore, histological studies showed significantly higher activity in the formulation-treated group. Our present study revealed that the prepared polyherbal formulation possesses potent wound healing activity, and although less potent than standard silver sulfadiazine, it could be a good choice of remedy for wound healing

Keywords: Polyherbal formulation; Wound healing; Excision wound; Silver sulfadiazine

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INTRODUCTION

Traditional medicine is based on the study of pharmacognosy and the application of medicinal plants, which are known as herbal medicines (also known as herbalism). There is little scientific proof of the safety and effectiveness of plants used in 21st-century herbalism, which typically lacks guidelines for dosage or purity. Minerals, shells, some animal parts, and products from fungi and bees are frequently included in the scope of herbal therapy. Another name for herbal medicine is phytotherapy or phytomedicine. The term "paraherbalism" refers to alternative and pseudoscientific methods of employing raw plant or animal extracts as untested medications or health-promoting substances. There is no proof to support the paraherbalism theory, which holds that keeping different chemicals from a particular source with less processing is safer or more efficient than manufactured goods. Skin: The human body has amazing defenses against infections. The microorganisms that have assaulted the body are eliminated by the internal defense mechanism. Microbes are kept out of the body by the external defense system. Although it plays a significant

role in the body's exterior protection system, the skin serves other purposes as well. It serves as a mechanical wall separating the body's interior from the outside. Anatomy of Skin: The skin is the body's biggest organ. There are three layers to it. The outermost epidermis is followed by the dermis, which is followed by the hypodermis.¹⁻⁸

Epidermis: The epidermis, the skin's outermost layer, acts as a barrier to protect the body's surface. It is in charge of keeping the body hydrated and guarding against infections. The stratified squamous epithelium that makes up this structure is made up of differentiating suprabasal keratinocytes and proliferating basal keratinocytes. In addition, the epidermis aids in controlling body temperature.^{8,9}

Stratum Basale (Basal Cell Layer): Basal cells comprise the deepest single-layered sublayer of the epidermis. It functions similarly to a dermal border. Keratinocytes are produced by it. Melanocytes are also seen in this stratum. Approximately 8% of the water in the epidermis is stored

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there. Its capacity to hold water diminishes with age and **thinning**.^{7,8,10}

Stratum Spinosum (Prickle Cell Layer): In contrast to the stratum basale, it has 10–20 layers. These layers are formed by basal cells becoming relatively flat (multisided) through the process of turnover. These cells, which go by the name "prickle cells," feature tiny spines on the exterior of their membrane. This sublayer's thickness usually ranges from 50 to 150 μm .

Dermis: The dermis, which is made up of connective tissue and shields the body from stress and strain, is located beneath the epidermis. Through an extracellular matrix made up of collagen fibrils, microfibrils, and elastic fibres embedded in proteoglycans, it provides the skin with tensile strength and elasticity. The dermis also contains blood arteries, lymphatic vessels, sweat glands, sebaceous glands, apocrine glands, and hair follicles. The dermis's primary function is temperature regulation. The epidermis receives nourishment from the encapsulated blood vessels of this layer as well. The dermis is 3–5 mm thick.³⁷

Wound: Any break or damage to the skin's surface is considered a wound. One definition is the loss or disruption of the cellular, anatomical, and functional continuity of live tissue¹². Damage can range from a minor break in the skin epithelium to more extensive damage that penetrates deeper into subcutaneous tissue and affects internal organs such as tendons, muscles, arteries, nerves, and even bone. The tissue may be attacked physically, chemically, thermally, microbiologically, or immunologically.^{18,32,35}

Open wounds are created when the skin is torn, sliced, or pierced, while closed wounds are created when blunt force trauma results in a contusion. On the other hand, burn wounds are caused by fire, heat, radiation, chemicals, electricity, or sunlight. A wound is an opening in the skin (the epidermis is the skin's outermost layer). Cuts or scrapes typically result in wounds; two types of tissue injury include swelling, stiffness, soreness, discolouration, skin tightness, scabbing, itching, and scar development.

Classification of Wounds: In wound healing and damage care, time is of the essence. Depending on how long it takes for a wound to heal, it can be classified as either acute or chronic.

Acute Wounds: A wound is considered acute if it heals in a rapid and ordered manner through predictable stages. Usually, the healing process takes 5-10 days or less than 30 days. Acute wounds are caused by either surgery or traumatic tissue loss.

Chronic Wounds: When the normal healing process is interfered with at one or more stages of hemostasis, inflammation, proliferation, or remodelling, the wound is considered chronic and takes longer to heal than expected. The healing process could take a few weeks or, in certain situations, up to 6 weeks.²²

Diabetic Wounds: People who have diabetes or peripheral vascular disease, for example, are more likely to develop foot ulcers. Foot ulcers in diabetic people can be divided into

Neuropathic: It leads to loss of protective sensation. However, foot pulses may still be noticed

Neuro-Ischaemic: This disorder results in ischaemia, a lack of blood supply, and loss of protective feelings. Wounds from foot ulcers may be deep, including tendons, bones, and other tissues, or superficial, affecting only the outermost layer of skin.

Basic Principles of Wound Healing: Wound healing is the process by which tissue repair occurs. It consists of an ongoing series of inflammation and repair pathways, wherein fibroblasts, platelets, inflammatory cells, endothelial cells, and epithelial cells momentarily assemble outside of their normal domains and interact to restore resemblances of their used discipline and to resume their normal function. The process of wound healing is dynamic and intricate. As a person's health status varies, so does the wound environment. A foundation for comprehending the fundamentals of wound healing is provided by knowledge of the physiology of the typical wound healing trajectory through the stages of hemostasis, inflammation, granulation, and maturation.²⁰

MATERIALS AND METHODS

In Vivo Pharmacological Activity

Experimental Animals

Healthy albino rats weighing 150–200 g were utilized in the investigation. They were obtained from RV Northland Institute, Dadri, Greater Noida, Gautam Buddha Nagar, India. The study's procedures were authorized by the Animal House as well as by each experimental protocol. The study protocol (Register Number:1149/PO/RE/S/07 CPCSEA) was approved by the Institutional Animal Ethics Committee and met the requirements of the Committee for the Purpose of Control and Supervision of Experiments with Animals (CPCSEA) in India. Following their acclimation to the standard laboratory conditions, which included a temperature of $25 \pm 2^\circ\text{C}$, a relative humidity of 60%–70%, and a light and dark cycle of 12:12 hours, the animals were fed a standard diet and water at will throughout the trial.^{8,9}

Wound Healing Activity

Using an excision wound healing model, the wound healing activity of the polyherbal formulation was evaluated.

Excision Wound Model

Using toothed forceps, a circular, 500 mm² diameter, full-thickness surgically excised incision was made in 18 rats along the marking. Ketamine hydrochloride (20 mg/kg, intramuscular) was used to anaesthetize the surgical blade and pointed scissors over the dorsum part using depilatory cream (Veet) following thorough cleansing and shaving.

Three groups of six creatures each were created from the animals.

Group I: Control group treated with normal saline solution.

Group II: Standard group treated with silver sulfadiazine (1% w/w).

Group III: Test group treated with polyherbal formulation applied.

The animal was given 0.1 g of its body weight in an equal amount of a polyherbal mixture twice a day for 21 days. Every day, at least two dressings were applied. Both the research and control groups' scar characteristics and the amount of time required to complete epithelization were noted. Using a digital Vernier calliper (least count = 0.001 mm), the wound area in the excision wound model was measured for each group on days 0, 3, 6, 9, 12, 15, and 21. By monitoring the wound's contraction every day until it was completely healed, the percentage of the wound area that had healed was determined. The percentage of wound contraction was calculated using an initial wound size of 100%. The percentage of wound contraction was computed using the formula.¹¹

Formulae = (Initial – final)

Initial x 100.

The skin was separated into three sections at the conclusion of the investigation. Histopathology was utilized in one section, biochemical measures in the second, and antioxidant enzyme evaluation in the third.

Biochemical Parameters

Estimation of Hydroxyproline

The healed tissue was removed and allowed to dry in glass vials for 48 hours at 110 °C. Five milligrams of lyophilized material was hydrolysed with 5 mL of 6 N HCl at 110°C for 18–20 hours in a sealed tube to estimate the amount of hydroxyproline.⁸

Estimation of Collagen

The hydroxyproline content may be converted to its equivalent collagen through multiplication by the factor 7.46

Estimation of Hexosamine

To evaluate hexosamine, weighed granulation tissues were hydrolysed for eight hours at 98°C in 6 N HCl, neutralized with 4 N NaOH to lower the pH to 7, and then diluted with Milli-Q water. The hexosamine content of granulation tissues was measured using a modified version of the technique[20]. The diluted solution was heated to 96°C for 40 minutes alongside the acetyl acetone solution. To cool the liquid, 96% ethanol and an Ehrlich's agent solution

were utilized. The absorbance was measured with a Shimadzu UV/Visible spectrophotometer set to 530 nm after sufficient mixing and an hour at room temperature. The amount of hexosamine in the tissue was calculated as milligrams per gram of dry weight.

Antioxidant enzymes

Catalase

The 10% tissue homogenate in pH 7 was diluted with phosphate buffer pH-7 (1:100) after a detergent, such as 1% Triton X-100, was added. Catalase was estimated using the method outlined.

Reduced Glutathione assay

Using 2.5 mL of 5% TCA and 0.5 g of skin tissue, a tissue homogenate was created. Centrifugation of the precipitated protein for 10 minutes at 1000 rpm was performed. GSH was estimated using 0.1 mL of the supernatant.^{8,38}

Lipid Peroxidation Assay

All tubes were vortexed for a few seconds in a boiling water bath, cooled to room temperature, and then centrifuged for 15 minutes after adding 1 mL of tissue homogenate to TCA-TBA. HCl. OD was measured at 535 nm against a blank after supernatants were pipetted out in cuvettes.

Histopathological Study

The histological analysis was carried out in compliance with a standard protocol. The tissue samples (the wound area of the skin) from each group were dried using different alcohol concentrations and then preserved in paraffin wax. The paraffin wax-fixed tissue (5 µm) sections were made on a microtome and dried overnight in an oven before being stained with hematoxylin and eosin. Using a light microscope, sequential biopsies conducted on days 03, 06, 09, 12, 15, and 21 in all three groups showed histological changes in both animal models, i.e., excised wounds.

RESULTS

Excision Wound Model

When compared to animals treated with the usual medication silver sulfadiazine and animals treated with the test formulation, control rats exhibited a delayed contraction rate of the circular excised wound ($p < 0.001$) (**Table 1**). After 21 days, the Polyherbal formulation demonstrated 99.1% wound contraction, which was higher than the usual medication's 98.2% wound contraction. When compared to the animals in the control group, which had an 88.3% reduction in wound size, both the test and conventional drug-treated animals demonstrated a substantial reduction.^{38,8}

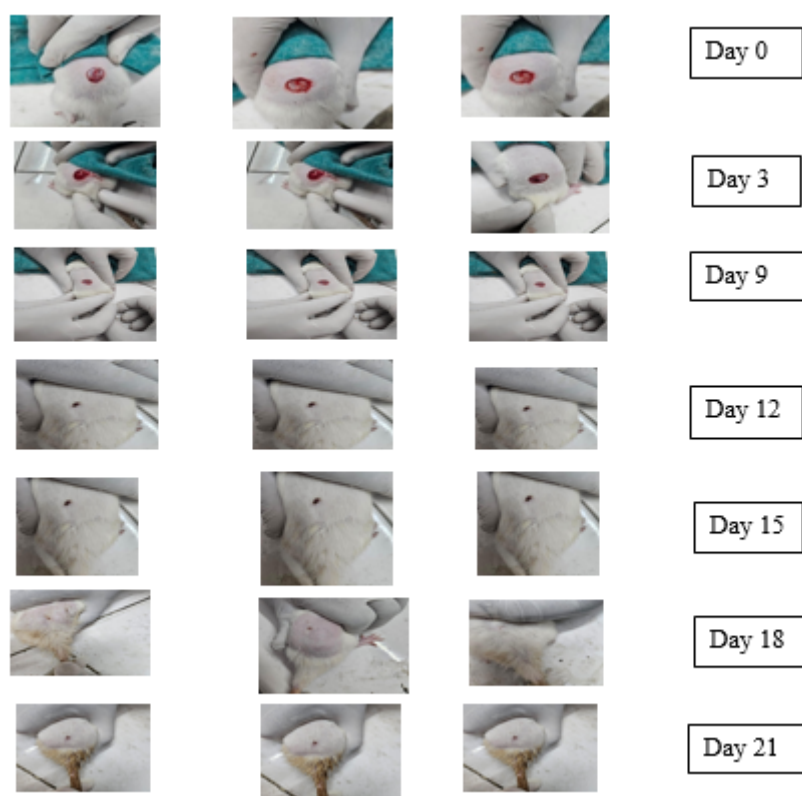


Figure 1. Healing pattern at different time intervals.

Figure 1 shows the healing pattern at different time interval^{13,4,19}

Table 1. Effect of Polyherbal formulation on excision wound contraction in mm².

Groups	0 day	3rd day	6th day	9th day	12th day	15th day
Control	387.40±7.06	326.00±5.73	290.67±5.54	140.23±8.07	62.50±6.15	45.22±6.93
Std.	403.33±7.82	225.67±6.24	147.83±9.78	61.78±6.42	23.33±3.67	6.52±2.43
Polyherbal formulation	388.50±7.39	297.50±4.97	202.61±4.46	111.00±6.22	44.17±5.32	3.00±1.79

Biochemistry of Wound Healing

The tissue content of hydroxyproline, collagen, and hexosamine on various postoperative days is displayed in **Table 2**. Hexosamine and hydroxyproline levels in the polyherbal formulation-treated groups were significantly higher than those in the control groups. The hydroxyproline level in the standard and polyherbal

formulation groups climbed gradually over a few days, but the hydroxyproline level in the control group increased slowly until the 9th day. The hydroxyproline, collagen, and hexosamine content of the extracellular matrix was found to be more significant in all treated groups than in the control groups during the healing process. These chemicals mark the wound healing process well.^{13,37}

Table 2. Effect of polyherbal formulation biochemistry of wound healing¹³.

Groups	Hydroxyproline	Collagen	Hexosamine
Control	31.2 ± 3.4	241.6 ± 25.2	6.4 ± 2.4
Std. drug	48.9 ± 3.6	362.0 ± 29.2	22.4 ± 1.2
Polyherbal formulation	62.5 ± 5.4	470.2 ± 42.2	27.3 ± 2.2

Antioxidant Activity in Wound Healing

Catalase and GSH levels were significantly higher in the polyherbal formulation than in the control group. Catalase levels in dry tissue were 1.82 ± 0.6 U/gm in the control group, 3.88 ± 0.6 U/gm in animals treated with standard drugs, and 5.44 ± 0.8 U/gm in animals treated with aqueous formulation. GSH levels were determined to be 173.3 ± 37.3 mol/gm in the control group, 331.7 ± 42.5

mol/gm in the standard drug group, and 564.6 ± 46.4 mol/gm in the test drug group. While the control group's tissue MDA values were higher (256.6 ± 24.5 U/gm), the standard drug-treated group's values were 109.9 ± 9.6 U/gm, and the formulation-treated group's values were 83.8 ± 5.6 U/gm. This indicates that the polyherbal formulation significantly reduced MDA levels ($p < 0.001$) (Table 3).³⁸

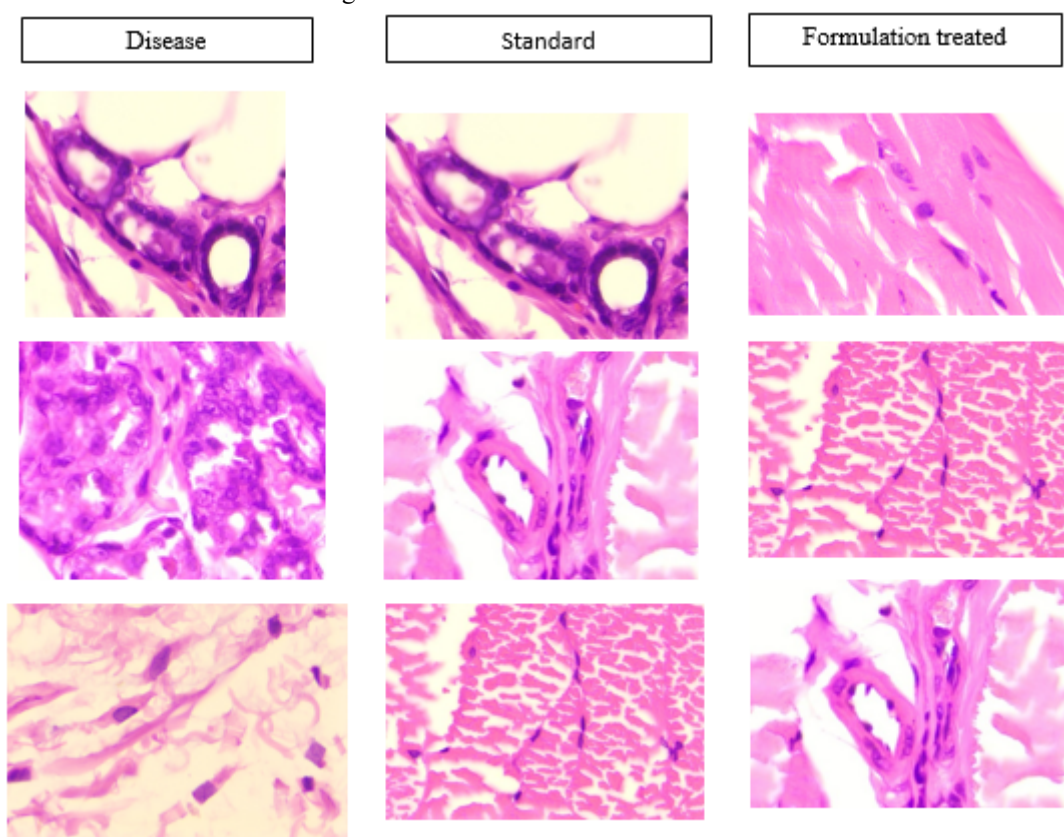
Table 3. Effect of polyherbal formulation on tissue antioxidant parameters.

Groups	Catalase (U/gm)	MDA(U/gm)	GSH (Moles/gm)
Control	1.72 ± 0.5	257.6 ± 25.2	174.2 ± 35.2
Std. drug	2.89 ± 0.5	110.0 ± 9.2	332.6 ± 41.4
Polyherbal formulation	6.22 ± 0.7	84.7 ± 5.6	563.3 ± 45.2

Histopathology Using Regenerated Skin Tissue

The regenerated tissue of the animal treated with the polyherbal formulation showed neovascularization, re-epithelialization of the epidermis, and subepidermal cells. Additionally, fibroblastic growth was observed where the wound was healing. Animals treated with standard and test drugs had normal epidermis with more collagen in the adjacent skin. Both test and standard drug-treated animals

showed fibroblastic proliferation and vascular proliferation covered with granulation tissue when the wound had fully healed. Collagen fibres were visible in fibrous growth beneath the normal epidermis. Beneath a large area of scab formation, polymorphs are trapped. In disease control (untreated) mice, fibroblastic and vascular growth with minimal mononuclear cell infiltration was noted. This is shown in Figure 2.^{32,35,26,28,31}

**Figure 2.** Histopathology using regenerated skin tissue.**CONCLUSION**

Wound contraction is the process of the wound's area shrinking, while wound healing is the process of returning injured tissue as nearly as possible to its original state. It mostly depends on the tissue's capacity to heal itself, the kind and degree of damage, and the overall health of the tissue. Fibroblasts, collagen, edema, and tiny new blood vessels make up the majority of the granulation tissue of the wound. Along with the fibrin strands, the undifferentiated mesenchymal cells of the wound margin transform into fibroblasts, which begin to migrate into the wound gap. The primary component of extracellular tissue, which offers strength and support, is collagen, which is made up of amino acids (hydroxyproline). Free hydroxyproline and its peptides are released as collagen is broken down; measuring hydroxyproline may serve as a

gauge for collagen turnover. It is possible to draw the conclusion that the prepared polyherbal formulation encourages wound-healing activity based on the data provided. As a result, it might be recommended for treating a variety of human wounds. To fully comprehend the process of wound healing action, more research including purified ingredients is required

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The authors have nothing to report.

Conflict Of Interests

The authors declare no conflict of interest.

REFERENCES

1. Deshmukh TP, Jennifer F, Akarte A, Emmanuel T. Wound healing activity of Calotropis gigantea root

- bark in rats. *J Ethnopharmacol.* 2009;125(1):178–181.
2. Natarajan V, Venugopal PV, Menon T. Effect of azadirachta (Neem) on the growth pattern of dermatophytes. *Indian J Med Microbiol* 2003;21:98–101.
4. Singh A, Singh DK. Molluscicidal activity of *Lawsonia inermis* and its binary and tertiary combinations with other plant derived molluscicides. *Indian J Exp Biol* 2001;39:263–268.
5. Kumar R, Katoch SS, Sharma S. Adrenoceptor agonist treatment reverses denervation atrophy with augmentation of collagen proliferation in denervated mice gastrocnemius muscle. *Indian J Exp. Biol* 2006;44:371–376.
6. Douglas M, Alan L, Miller ND. Nutritional support for wound healing. *Alternat Med Rev.* 2003;8(4):359–377.
7. 6 Dubey S, Dixit AK. Preclinical evidence of polyherbal formulations on wound healing: A systematic review on research trends and perspectives. *J Ayurveda Integr Med.* 2023;14(2):100688. doi: 10.1016/j.jaim.2023.100688. Epub 2023 Feb 24. PMID: 36841194; PMCID: PMC9988554.
8. Georgescu M, Chifiriuc CM, Marutescu L. Bioactive wound dressing's for the management of chronic wounds. *Curr Org Chem.* 2017;21:53–63.
9. Talekar YP, Apte KG, Paygude SV, Tondare PR, Parab PB. Studies on wound healing potential of Polyherbal formulation using in vitro and in vivo assays. *J Ayurveda Integr Med.* 2017;8(2):73–81.
10. Patel AM, Kurbetti SM, Savadi RV, Raje VN, Takale VV. Formulation and evaluation of new polyherbal formulations for their wound healing activity in rat. *Int J Res Allied Sci.* 2013;2(2):66–9.
11. Dorsett-Martin WA. Rat models of skin wound healing: a review. *Wound Repair Regen.* 2004;12(6):591–599.
12. Nasir MA, Mohammed NL, Roshan S, Ahmed MW. Wound healing activity of polyherbal formulation in albino rats using excision wound model, incision wound model, dead space wound model and burn wound model. *Int J Res Develop Pharm Life Sci.* 2016;5(2):2080–2087.
13. Lacman Leon, Herbert A Leberman. Theory and practice of industrial pharmacy. 3rd ed. Nirali Publication; 1987.p. 557.
14. Aly Usama F. Preparation and evaluation of novel topical gel preparation for wound healing in diabetics. *Int J Pharm Pharm Sci.* 2012;4:76–77.
15. Moon RS, Shrikhande R, Purohit N. Formulation and study of burn wound effect of herbal creams advance in pharmacology and toxicology. *Indian J Pharm Sci.* 2007;8:1–25.
16. **Indian Pharmacopoeia.** (1996) vol. 2, Delhi, The Delhi Controller of Publications.
17. 16 Raina R, Pravez S, Verma PK, and Pankaj NK. Medicinal plants and their role in wound healing. *Vet Scan.* 2008;3(1):1–7.
18. 17 Robson MC, Steed DL, Franz MG. Wound healing: biologic features and approaches to maximize healing trajectories. *Curr Probl Surg.* 2001;38:72–140.
19. Kumar B, Vijayakumar M, Govindarajan R, Pushpangadan P. Ethnopharmacological approaches to wound healing-exploring medicinal plants of India. *J Ethnopharmacol.* 2007;114:103–113.
20. Sachin J, Jain N, Tiwari A, Balekar N, Jain DK. Simple evaluation of wound healing activity of a Polyherbal formulation of roots of *Ageratum conyzoides* Linn. *Asian J. Research Chem.* 2009;2(2):135–138.
21. Strodtbeck F. Physiology of wound healing. *New Inf Nurs Rev.* 2001;1:43–52.
22. Gurtner GC, Werner S, Barrandon Y, Longaker MT (2008) Wound repair and regeneration. *Nature.* 2008;453:314–321.
23. 22 Schultz GS (2007) Molecular Regulation of Wound Healing. In: *Acute and Chronic Wounds: Current Management Concepts.* 3rd ed. Mosby Inc., St. Louis: 2007, p. 82–99.
24. 23 Balandrin MF, Kinghorn AD, Farnsworth NR. Plant-Derived Natural Products in Drug Discovery and Development. In: *Human Medicinal Agents from Plants*, Kinghorn AD, Balandrin MF ed, 534, ACS, Washington DC: 1993, p. 2–12.
25. Jagetia GC, Rajanikant GK. Role of curcumin, a naturally occurring phenolic compound of turmeric in accelerating the repair of excision wound, in mice whole-body exposed to various doses of gamma-radiation, *J Surg Res.* 2004;120:127–138.
26. K Mohan AV. Effect of topical phenytoin on burn wound healing in rats. *Indian J Exp Biol* 2011;49:56–59.
27. Ahmad NS, Mohd SA, Ahmad A. Effect of *Carica papaya* Linn, latex on the healing of burns wounds in rats. *J Sains Kesihatan Malaysia* 2005;3:39–47.
28. Moon RS, Shrikhande R, Purohit N. Formulation and study of burn wound effect of herbal creams advance in pharmacology and toxicology. *Indian J Pharm Sci.* 2007;8:1–25.

29. Gopalkrishnan V, Rao L., Sharma VK, Bhavna V. Antimicrobial activity of extract of *acalypha indica* linn. *Indian J Pharm Sci.* 2000;62:347–349.
30. K Ramanjaneyulu, A Bhargavi, P Rajvarma. Evaluation of phytochemical and antibacterial activity of *Butea monosperma* leaf extract. *Int J Pharm Res.* 2011;2:1563–1565.
31. Rajvaidhya S, Signh GK. A review on *Acacia arabica* in Indian medicinal plants. *Int J Pharm Sci Res.* 2012;3:1995–2005.
32. Subalakshmi M, Saranya A, Maheswari MU, Jarina A, Kavimani S. An overview of the current methodologies used for the evaluation of drugs having wound healing activity. *Int J Exp Pharmacol.* 2014;4:127–131.
33. Kumar A. Wound healing and *Ficus arnottiana* Miq. *Indian Health J.* 2011;5:68–58.
34. Das MK, Mazumder P. Evaluation of antibacterial activity in *Butea monosperma*: a comprehensive review. *Int Res J Plant Sci.* 2011;2;7:215-219.
35. Malviya S. Medicinal attributes of *Acacia arabica* Linn—A comprehensive review on ethopharmacological claims. *Int J Pharm Life Sci.* 2011;4:830–837.
36. Aly Usama F. Preparation and evaluation of novel topical gel preparation for wound healing in diabetics. *Int J Pharm Pharm Sci.* 2012;4:76–78.
37. Li B, Wang James HC. Fibroblasts and myofibroblasts in wound healing: force generation and measurement. *J Tissue Viab.* 2011;20(4):108e20.
38. Natarajan V, Krithica N, Madhan B, Sehgal PK. Preparation and properties of Tannic acid cross-linked collagen scaffold and its application in wound healing. *J Biomed Mater Res B Appl Biomater.* 2013;101(4):560e7.
39. Suntar I, Kupeli AE, Nahar L, Satyajit D, Sarker SD. Wound healing and antioxidant properties: do they coexist in plants. *Fr Rad Antioxid.* 2012;2(2):1–7.